INVITED PAPER

JOURNAL OF APPLIED BIOMECHANICS, 1997, **13**, 135-163 © 1997 by Human Kinetics Publishers, Inc.

The Use of Surface Electromyography in Biomechanics

Carlo J. De Luca

This lecture explores the various uses of surface electromyography in the field of biomechanics. Three groups of applications are considered: those involving the activation timing of muscles, the force/EMG signal relationship, and the use of the EMG signal as a fatigue index. Technical considerations for recording the EMG signal with maximal fidelity are reviewed, and a compendium of all known factors that affect the information contained in the EMG signal is presented. Questions are posed to guide the practitioner in the proper use of surface electromyography. Sixteen recommendations are made regarding the proper detection, analysis, and interpretation of the EMG signal and measured force. Sixteen outstanding problems that present the greatest challenges to the advancement of surface electromyography are put forward for consideration. Finally, a plea is made for arriving at an international agreement on procedures commonly used in electromyography and biomechanics.

Electromyography is a seductive muse because it provides easy access to physiological processes that cause the muscle to generate force, produce movement, and accomplish the countless functions that allow us to interact with the world around us. The current state of surface electromyography is enigmatic. It provides many important and useful applications, but it has many limitations that must be understood, considered, and eventually removed so that the discipline is more scientifically based and less reliant on the art of use. To its detriment, electromyography is too easy to use and consequently too easy to abuse.

The scope of this paper is not to review past contributions to the advancement of electromyography in biomechanics. Many researchers have contributed to that chronicle.¹ Instead this discussion will focus on the complex and interrelated factors underlying the relationship between the electromyographic (EMG) signal and the force produced by a muscle. Many intricate issues will be raised to identify the intrinsic complexity of the relationship. I hope that this discussion will raise awareness of the usefulness and limita-

135

Carlo J. De Luca is with the NeuroMuscular Research Center and Biomedical Engineering Department and Neurology Department, Boston University, 44 Cummington St., Boston, MA 02215.

This paper was presented to the International Society for Biomechanics in Paris on July 5, 1993, as the Wartenweiler Memorial Lecture.

^{&#}x27;The list of contributors is long. The inclusion of a comprehensive bibliography would distract from the simplicity and clarity of the messages, which are the dominant features of the lecture. This lecture is not intended to be a critical review. When necessary, I have referred only to my work and to that of my scientific collaborators, knowing that by doing so, I run the risk of self-aggrandizement. Instead, I hope that the reader will accept the notion that this construct was intended to maintain neutrality among the many candidates who qualified for inclusion.

tions of the EMG signal/force relationship so that practitioners will clear the pitfalls and direct applications to those areas where the use can be beneficial.

When EMG signal and force measurements are made, there are five cardinal questions (and related subquestions) that the investigator should consider to be assured of proper usage:

- 1. Is the EMG signal detected and recorded with maximum fidelity?
 - What are the configuration, dimension, and electrical characteristics of the electrode unit?
- 2. How should the EMG signal be analyzed?
 - How are the initiation and cessation times of the EMG signal measured?
 - What are the preferred parameters for measuring the amplitude of the EMG signal?
 - What are the preferred parameters for measuring the frequency spectrum?
- 3. Where does the detected EMG signal originate?
 - Is there any crosstalk? That is, does any of the detected signal originate from nearby muscles?
 - Where is the electrode placed on the surface of the muscle in relation to its anatomical structures?
 - How much fatty tissue is there between the electrode and the muscle surface?
- 4. Is the EMG signal sufficiently stationary for the intended analysis and interpretation?
 - Does the muscle change length during the contraction?
 - Is the activation pattern of the motor units stable? That is, do some motor units alternate between the state of recruitment and derecruitment?
- 5. Where does the measured force originate?
 - What is the state of the synergistic and antagonistic muscles associated with the task?
 - Are the motor control characteristics of the contraction stable for the intended interpretation? Is there any change in the relative force contribution among muscles during the contraction?
 - Is the force generated homogeneously throughout the muscle?

All these factors are important, albeit to different extents, depending on the situation. Of the above factors, the issues dealing with the electrode and the type of muscle contraction can be addressed most directly and with the greatest success, although even the concerns over these issues cannot be completely dissipated by known technology and knowledge of anatomy. Nonetheless, judicious applications of known facts can ensure the fidelity of the EMG signal, reduce crosstalk, and provide sufficient stationarity in the signal; normalization of the signal amplitude may remove the influence of many other variables.

In biomechanics, three applications dominate the use of the surface EMG signal: its use as an indicator of the initiation of muscle activation, its relationship to the force produced by a muscle, and its use as an index of fatigue processes occurring within a muscle. As an indicator of the initiation of muscle activity, the signal can provide the timing sequence of one or more muscles performing a task, such as during gait or in the mainte-

Surface Electromyography

nance of erect posture. Another important application of the EMG signal is to provide information about the force contribution of individual muscles as well as groups of muscles. Use in the individual muscle provides the greater attraction. The resultant muscular moment acting on a joint during a specific task is only in exceptionally rare cases due to one muscle. (The interosseous muscles of the hand, the flexor pollicis longus, and the extensor pollicis are among the few that come to mind.) Thus, in the vast majority of cases of interest, the ability to determine noninvasively the force contribution of individual muscles provides an enormous advantage, particularly when biomechanical models are developed to describe the workings of a segment of the musculoskeletal system. The use of the EMG signal to provide a fatigue index has considerable appeal because it has been shown that the signal displays time-dependent changes prior to any force modification, thus having the potential to predict the onset of contractile fatigue.

I will discuss these applications and review technical considerations in individual sections. Each section begins with the cardinal questions relevant to the topic. At the end of each section, I have listed recommendations that may assist the practitioner in detecting, analyzing, and interpreting the EMG signal and measured force. These recommendations should be viewed as guidelines that will evolve and be modified as our knowledge increases. They are numbered consecutively because they should be considered as cumulative; that is, a recommendation appearing in an earlier section also applies to subsequent sections. Also, I have posed problems which, in my view, present the greatest challenges to the advancement of the field of surface electromyography.

In order to facilitate discussion on these application topics, I have found it useful to identify numerous factors that influence the detected EMG signal and the measured force.

Factors That Affect the EMG Signal and Force Produced by a Muscle

One of the most frustrating, or appealing (depending on your perspective), aspects of the surface EMG signal is that when rectified and sufficiently smoothed, its amplitude is qualitatively related to the amount of torque (or force) measured about a joint, but more often than not, an accurate quantitative relationship is elusive. The reason for this quandary is that the EMG signal is the result of many physiological, anatomical, and technical factors. The effects of some of these factors may be managed by proper detection methods, but others are not easily regulated with current technology, and their potential effect on the signal may only be surmised and considered. To use the signal effectively, it is first necessary to understand as much as possible the sources of, and the influences on, the signal. This task is daunting and complicated because the current state of knowledge does not enable us to consider quantitatively the cause and effect of all the processes and phenomena that influence the EMG signal. Even if the influences could be completely characterized, the analytical rendition would be complicated by the anisotropy and inhomogeneity of the tissues between the muscle membranes and the detection electrode. The analysis would be further confounded by the uniqueness of the microanatomical structures of each detection location. It is conceivable that in the future, elegant and complex models will be used in conjunction with means for describing the anatomy, physiology, and electrical field properties to allow a direct consideration of this problem. For the time being, we are restricted to general considerations, simplistic models, and simplistic analyses.

In an attempt to describe the factors that influence the EMG signal, I have found it useful to group them into the following categories: causative, intermediate, and determin-

istic factors. The interrelationships of the factors along with their effects on the EMG signal and the interpretation of the signal characteristics are displayed in Figure 1. Any attempt such as this to collect known influences on the EMG signal will be constrained in its ability to express all possible interactions. The following attempt represents a first pass that is subject to improvement. The interaction is organized so as to describe a "flow-of-influence" among the factors, the EMG signal, and the force.

The *causative* factors have a basic or elemental effect on the signal. These are divided into two groups: extrinsic and intrinsic. The extrinsic causative factors are those associated with the electrode structure and its placement on the surface of the skin above the muscle. They include the following:

- 1. Electrode configuration, which describes
- the area and shape of the electrode detection surfaces, which determine the number of active motor units detected by virtue of the number of muscle fibers in their vicinity, and
- the distance between the electrode detection surfaces, which determines the bandwidth of the differential electrode configuration.
- Location of the electrode with respect to the motor points in the muscle and the myotendinous junction, which influences the amplitude and frequency characteristics of the detected signal.
- 3. Location of the electrode on the muscle surface with respect to the lateral edge of the muscle, which determines the amount of crosstalk that may be detected by the electrode.
- 4. Orientation of the detection surfaces with respect to the muscle fibers, which affects the value of the measured conduction velocity of the action potentials and, consequently, the amplitude and frequency content of the signal.

The influence of electrode location on the amplitude and frequency spectrum of the signal is displayed in Figure 2.

The intrinsic causative factors are the physiological, anatomical, and biochemical characteristics of the muscle. Unlike the extrinsic factors, they cannot be controlled due to limitations of current knowledge and technology. They include the following:

- The number of active motor units at any particular time of the contraction, which contributes to the amplitude of the detected signal.
- Fiber type composition of the muscle, which determines the change in the pH of the muscle interstitial fluid during a contraction.
- Blood flow in the muscle, which determines the rate at which metabolites are removed during the contraction.
- Fiber diameter, which influences the amplitude and conduction velocity of the action potentials that constitute the signal.
- Depth and location of the active fibers within the muscle with respect to the electrode detection surfaces; this relationship determines the spatial filtering, and consequently the amplitude and frequency characteristics, of the detected signal.
- The amount of tissue between the surface of the muscle and the electrode, which affects the spatial filtering of the signal.

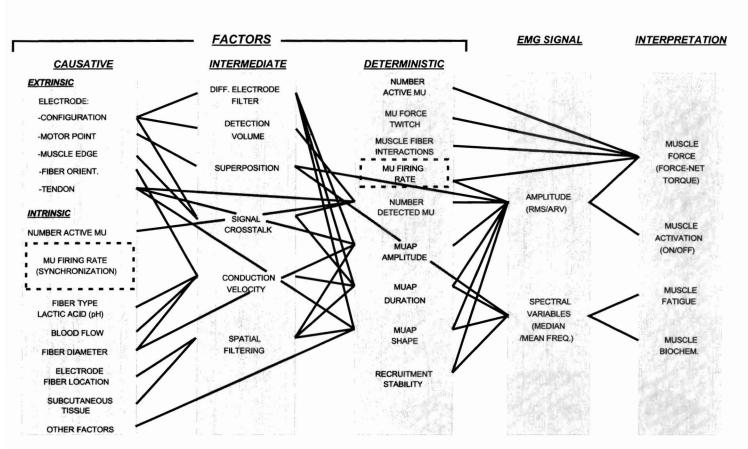


Figure 1 — Schematic diagram of the factors that affect the EMG signal. The arrangement of factors is designed to demonstrate the flow of the influences and interactions among the factors.

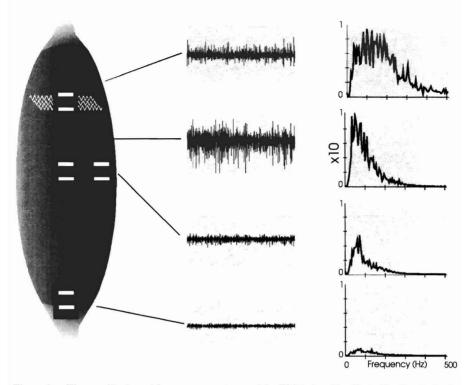


Figure 2 — The amplitude and frequency spectrum of the EMG signal is affected by the electrode location with respect to the innervation zone (top electrode), myotendinous junction (bottom electrode), and lateral edge of the muscle (middle right electrode). The preferred location is in the midline of the muscle belly between the nearest innervation zone and the myotendinous junction. In this location, the EMG signal with the greatest amplitude is detected.

• Other factors that are yet to be identified, such as the length of the depolarization zone and ionic fluxes across the membrane.

In this category, I have also included firing characteristics of the motor units (which include the behavior of the motor unit firing rates and any interaction among the firing rates, such as synchronization of motor unit firings) and the motor unit twitch. These latter causative factors are presented in a dashed-line box in Figure 1, because although they are causative, they are also deterministic in that they affect the EMG signal directly.

The *intermediate* factors represent physical and physiological phenomena that are influenced by one or more of the causative factors and in turn influence the deterministic factors. These include the following:

- Band-pass filtering aspects of the electrode, which are inherent characteristics of a differential electrode configuration.
- Detection volume of the electrode, which determines the number and weight of the motor unit action potentials that compose the signal.
- Superposition of action potentials in the detected EMG signal, which influences characteristics of the amplitude and frequency of the signal.

- Crosstalk from nearby muscles, which contaminates the signal and may mislead interpretation of the signal information.
- Conduction velocity of the action potentials that propagate along the muscle fiber membrane; the conduction velocity affects amplitude and frequency characteristics of the signal.
- The spatial filtering effect due to relative position of the electrode and the active muscle fibers.

The latter two factors are especially important because they dramatically affect the signal characteristics. As the distance between the active fibers and the electrode detection surfaces varies, two important concerns arise. First, the spatial filtering characteristics of the detection arrangement change, thus altering the amplitude and frequency characteristics of the motor unit action potentials (MUAPs) that are within the detection volume of the electrode. Second, the relative movement of the electrode and the active fibers may be sufficient to place a new set of active motor units within the detection volume of the electrode and to remove some of the motor units from the detection volume. This consideration requires that if the muscle fibers change length during a contraction, then the electrode position must change similarly. With current detection techniques, it is difficult to satisfy this requirement because the electrode is affixed to the skin surface, which does not change length in concert with the muscle fibers during a contraction. Thus, for practical reasons, signal stability can only be approached if the contraction remains isometric. If signal stability is not a consideration for the analysis being performed, such as determining activation time, then the limitation of the isometric contraction need not be a concern.

The *deterministic* factors have a direct bearing on the information in the EMG signal and the recorded force. These include the following:

- The number of active motor units.
- Motor unit force-twitch.
- Mechanical interaction between muscle fibers.
- Motor unit firing rate.
- The number of detected motor units.
- Amplitude, duration, and shape of the MUAPs.
- Recruitment stability of motor units.

When one studies the rich and convoluted interactions between the many factors that influence the information content of the EMG signal, it is reasonable to ask if there is any hope of using the EMG signal in a constructive fashion to describe the state of the muscle. The answer is a confident "yes" for some applications and a guarded "maybe" for other applications. For example, we can have confidence in measurements where an electrode, which does not detect significant crosstalk from adjacent muscles, is placed on the muscle surface between the innervation zone and the myotendinous junction for the purpose of (a) determining, in a particular subject, when the muscle turns "on and off" or (b) describing if the muscle force output is increasing or decreasing over a period of time when the fatigue processes of the muscle do not significantly affect signal characteristics. If, however, the circumstances change from this specific condition, interpretation becomes complicated and caution is required.

The general map of interactions provided in Figure 1 represents most known interactions that attempt to describe all circumstances. If we are interested in a particular relationship, contraction, or phenomenon, the interactions reduce to specific subsets of the complete map.

Detection and Processing of the EMG Signal

Is the EMG signal detected and recorded with maximal fidelity? How should the EMG signal be analyzed?

Although the technical considerations of how to detect and process the EMG signal are not central issues in this paper, it is nonetheless useful to review some of the essential details. For a more complete description, see *Muscles Alive* (5th ed.) (Basmajian & De Luca, 1985).

The first issue concerns electrode configuration. Because the EMG signal is low in amplitude with respect to other ambient signals on the skin surface, it is necessary and convenient to detect the EMG signal with a differential configuration. That is, two detection surfaces are used and the two detected signals are subtracted prior to being amplified. In this differential configuration, the shape and area of the detection surfaces and the distance between the detection surfaces are important factors because they affect the amplitude and frequency content of the signal. The differential arrangement acts as a comb band-pass filter to the electrical signal seen by the detection surfaces. (In actuality, if the interdetection surface spacing is set so as not to alias the EMG signal, the spectrum of the EMG signal should fit in the low end of the band-pass filter. Thus, for practical purposes, the differential electrode behaves as a high-pass filter.)

The distribution of frequencies in the spectrum as well as the bandwidth is affected by the distance between detection surfaces. This fact is often not given sufficient consideration. Also, the shapes and areas of and the distance between the detection surfaces determine the number of muscle fibers seen by the electrode, thus affecting the signal amplitude; that is, the greater the number of fibers covered by the detection surface, the greater the amplitude of the EMG signal. The distance between the detection surfaces need not span a large portion of the muscle in order to detect a signal that represents the whole surface of the muscle, because the muscle fibers of a motor unit are somewhat randomly scattered throughout the cross-section of a muscle; thus, any location on the muscle contains fibers that represent motor units which generate a force throughout the muscle. From a practical point, the distance between the detection surfaces cannot be too small because the detection surfaces may be shunted electrically if the surface of the skin becomes moist with sweat, which is conductive. The electrical shunting decreases the signal amplitude, deteriorates the signal to noise ratio, and may filter out the higher frequency components.

There is presently no agreement on the configuration and dimensions of a standard surface electrode. This is partially due to the different perceived requirements of an electrode. With the intention of initiating a discussion to generate a consensus on this point, I have included below a number of specifications that have been honed from two decades of practice.

The other issue concerns how the EMG signal is processed. In the time domain, two parameters are commonly used: the root-mean-squared (RMS) value and the average rectified value. Both are appropriate and provide useful measurements of signal amplitude. For EMG signals detected during voluntarily elicited contractions, the RMS value may be more appropriate because it represents the signal power and thus has a clear physical meaning. On the other hand, the average rectified value is a measure of the area under the signal and hence does not have a specific physical meaning. For additional details, refer to De Luca and Van Dyk (1975) and De Luca and Knaflitz (1992).

Recommendations

- 1. Differential electrode configuration
 - Detection surfaces to consist of two parallel bars, each 1.0 cm long, 1–2 mm wide, spaced 1.0 cm apart.
 - A bandwidth of 20-500 Hz with a roll-off at least 12 dB/octave.
 - A common mode rejection ratio > 80 dB.
 - Noise $< 2 \,\mu V RMS (20-500 \,\text{Hz}).$
 - Input impedance > $100 \text{ M}\Omega$
- Locate the electrode on the midline of the muscle belly, between the myotendinous junction and the nearest innervation zone, with the detection surface oriented perpendicularly to the length of the muscle fibers. Use electrical stimulation or surface electrical mapping to locate the innervation zones.
- 3. Use the RMS value of the signal to measure the amplitude of the voluntarily elicited EMG signal.

The Activation Timing of Muscles

How should the EMG signal be analyzed? Where does the detected EMG signal originate?

For determining the activation timing of muscles, it does not matter if the contraction is isometric or anisometric. It is only relevant to determine if any segment of the muscle in the vicinity of the electrode is active. This is effectively accomplished by determining if the EMG signal originates from the muscle of interest and if the signal amplitude supersedes the amplitude of the noise in the detection and recording equipment.

The issue of crosstalk from other adjacent muscles is important in this case because the amplitude of the signal being analyzed is low and near the noise level. It has been shown (De Luca & Merletti, 1988) that in the leg, as much as 17% of electrical activity from nearby muscles may be detected on the surface of the muscle of interest. Therefore, if an adjacent muscle is active rather than the one directly below the electrode, a crosstalk signal can be detected and misinterpreted as originating from the muscle of interest. The likelihood of detecting a crosstalk signal may be reduced considerably by placing the electrode in the midline of the muscle belly. However, even this precaution may not ensure that the minimally perceived signal originates from the muscle of interest.

An approach that has been suggested for determining if a signal originates from an adjacent muscle is to cross-correlate the signal detected above a muscle with the signal separately detected above an adjacent muscle, the concept being that if the signals have a cross-correlation value less than 0.3, for example, crosstalk is not present. This approach is not sound because the tissues between and within the various muscles are anisotropic and inhomogeneous. These properties of the conduction volume cause multiple diffractions of the electric field vectors at the discontinuities and generate multiple paths of differing impedances between the source and the detection sites, causing the signal to be scrambled in the frequency domain and hence uncorrelated. Thus, signals originating in other muscles would not be interpreted as crosstalk, yielding a false-negative error. Another argument against the cross-correlation approach stems from the fact that the firing rates of motor units in different muscles contracting to perform a specific task can be considerably cross-correlated (De Luca & Mambrito, 1987). Thus, the surface EMG signals from such simultaneously contracting muscles could be substantially cross-

correlated without the presence of crosstalk, yielding a false-positive error. This is an important consideration when coactivation of muscles is suspected.

Muscle function testing has been suggested as a means to determine the presence of crosstalk. This approach consists of placing electrodes above the muscle of interest, stabilizing the joint in a position favorable for specific muscle action, and requesting the subject to make specific contractions which should activate muscles adjacent to the one under consideration but which will not activate that muscle itself. Thus, if a signal is detected during the procedure, it would be crosstalk from adjacent muscles. The uncertainty of this approach lies in the fact that one cannot know definitively if the subject is activating the nearby muscles during the tests, thus yielding a false-negative error.

By current knowledge, there is only one way to reduce and possibly eliminate crosstalk in the EMG signal detected with surface electrodes: the double differential technique (Broman, Bilotto, & De Luca, 1985; De Luca & Knaflitz, 1992). This technique consists of using a surface electrode having three detection surfaces equally spaced apart. Two differential signals are obtained, one from detection surfaces 1 and 2 and another from detection surfaces 2 and 3; then a differential signal is obtained from these two. Thus, the EMG signal undergoes two levels of differentiation. This procedure considerably decreases the pick-up volume of the three-bar electrode, thus filtering out signals from further distances that often correspond to those emanating from other muscles. Figure 3 presents an example in which use of the double differential technique eliminates the crosstalk signal. The top right panel presents the single and double differential signals detected at the flexor carpi ulnaris muscle during a 50% maximal voluntary contraction (MVC) wrist extension isometric contraction. Note that the single differential signal is present, whereas the simultaneously detected double differential signal (which originates from farther away) is indistinguishable from the noise. In contrast, the bottom right panel shows that when the flexor carpi ulnaris muscle is activated at the 5% MVC level, both single and double differential signals are detected because the source for both is near the electrode placed on top of the muscle.

If the double differential technique cannot be used, then there are two ways to test the signal for crosstalk. The first is to calculate the frequency spectrum of the EMG signal that is suspected to contain a crosstalk signal and that of an EMG signal believed to originate from the muscle of interest. A crosstalk signal will have a lower frequency spectrum because it originates further away and will be subject to additional low-pass filtering due to spatial filtering. This effect may also be seen in the bottom left panel of Figure 3. The power spectrum of the single differential signal detected during the 5% MVC of the flexor carpi ulnaris muscle has a greater bandwidth than the single differential signal detected on the same muscle during a 50% MVC isometric extension contraction of the wrist. The lesser bandwidth of the latter signal is consistent with the additional spatial filtering effect on a signal originating at a greater distance, as would be expected for a crosstalk signal.

The second approach consists of placing surface electrodes on all the adjacent muscles and wire electrodes in the deep muscles to monitor them for lack of activity. This approach is cumbersome, involves the use of several surface electrodes, and requires the use of wire electrodes. Using wire electrodes negates the original advantage of surface electrodes. If wire electrodes are to be used, use them to detect the signal from the muscle of interest, because they are far less susceptible to crosstalk.

It is also necessary to consider the delay between muscle activation as detected by the EMG signal and muscle activation as determined by muscle force generation. For biomechanical studies, the time course of the force is the relevant parameter. The delay between the EMG signal and the force is a variable that depends on several factors, including fiber-type composition of the muscle, firing rate dynamics of the muscle, and vis-

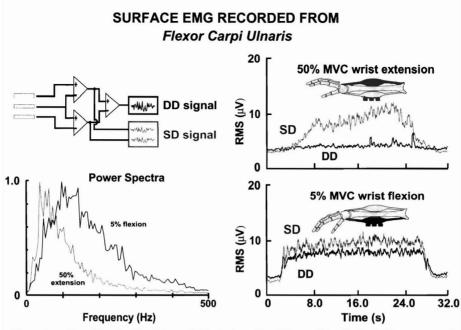


Figure 3 — Use of the double differential technique (top left panel) to eliminate the presence of crosstalk (top right panel) while detecting the signal from the muscle of interest (bottom right panel). Note the decreased bandwidth (bottom left panel) of the single differential signal originating from the extensor muscles as compared to that of the single differential signal originating from the flexor carpi ulnaris directly below the electrode. This indicates the spatial filtering effect on signals that travel greater distances to reach the electrode.

coelastic properties of the muscle and tendon tissues (including their length). The relatively more aerobic, slower twitch, slower fatiguing muscle fibers have a relatively slower rise-time in their force twitch and tetanization force than the relatively more glycolytic, faster twitch, faster fatiguing muscle fibers. Thus, a muscle consisting of a greater percentage of fast-twitch muscle fibers will have a shorter time delay between the EMG signal and the force. The firing rate dynamics of the motor units determine the rate with which the motor units will generate tetanic force. The viscoelastic properties of the muscle and tendon tissue determine the rate of force increase at the tendon attachment to the bone. The overall delay between the excitation of the muscle and the force buildup can be considerable, on the order of a few hundred milliseconds, as shown in Figure 4.

In addition to the physiologically caused delay between force and the EMG signal, other considerations must be taken into account when estimating the on–off times of the EMG signal. There is a physiological limit to the accuracy of the resolution of the estimate. Consider the case where the electrode is located 4 cm from the center of the innervation zone. With an average conduction velocity of 4 m/s, the signal will require 10 ms to reach the electrode. (Such a time delay is realistic in the larger muscles of the limbs.) Thus, the resolution need not be greater than 10 ms when comparing the on–off times among different muscles, among different subjects, and when the electrode is relocated. Also, as the muscle fatigues, the conduction velocity decreases and the arrival time of the EMG signal increases.

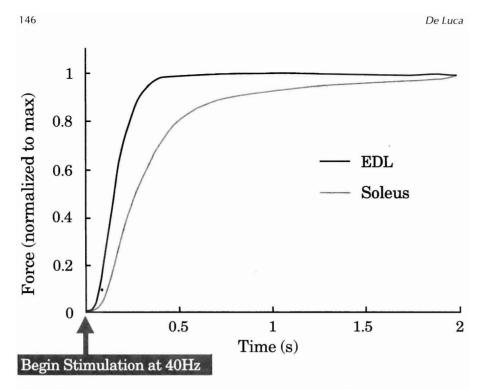


Figure 4 — Tetanic force produced by the soleus muscle (slow-twitch, highly aerobic, slow-fatiguing) and the extensor digitorum longus muscle (fast-twitch, highly glycolytic, fast-fatiguing), both from the rat. Both muscles were stimulated supramaximally, via their nerve, with a 40 Hz train of pulses having a width of 0.2 ms. Note the dramatic difference in the delay of the force rise-time.

If the activation time is to be determined in real time, then knowledge of the detection and recording-system noise is required. This implies that an epoch of noise must be recorded before the EMG signal is activated so that an estimate of the noise level is available. Figure 5 presents an example of the EMG signal initiating and becoming distinct from the background noise. The top trace represents the recorded EMG signal and the bottom trace represents the RMS value processed with an averaging window of 25 ms. The amplitude of the noise may be estimated by treating the noise as a stochastic variable. There are several ways of doing so. A simple approach is to calculate the value of the noise amplitude at two standard deviations from the mean value. This value captures 95% of the amplitude of the noise signal and is commonly used as a descriptor of stochastic signals. The time value at which the EMG signal exceeds this level or recedes below it, for a minimally defined amount of time (say 20 ms or twice the typical physiologically limited resolution), may be considered as the on or off time of the muscle. This point is indicated as t_{a} in Figure 5. Note that more sophisticated approaches are possible by prewhitening the raw EMG signal and constructing decision-based criteria for establishing when the raw signal surpasses the noise threshold. This approach requires relatively sophisticated processing, and any improvement in the accuracy of the estimate may not be meaningful given the physiological limitation of the time resolution.

If the determination may be made off-line, then a more physiologically precise estimate of the on-off times may be made by calculating the intersection of the timevarying mean value of the noise and the EMG signal. These values may be obtained by

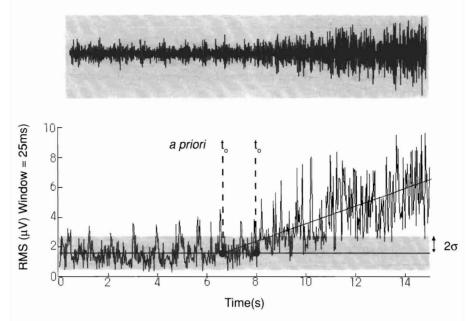


Figure 5 — Example of background noise and initiation of EMG signal activation from the erector spinae muscle of the back during a slowly increasing isometric extension of the back. Note the two different identifications of initiation time depending on availability of a priori knowledge of the stochastic properties of the noise and the behavior of the signal amplitude.

calculating the linear regressions of both. This point is indicated as a priori t_o in Figure 5. Note that the a priori intercept will occur earlier because of the additional information available in the off-line estimate. In the example, the discrepancy between the two estimates is on the order of 1.5 s. This is a rather large value, found only when the EMG signal increases very slowly, as was the case in the gradual isometric extension of the erector spinae muscles represented in Figure 5. In such cases, this discrepancy can completely override the physiologically induced delay between the force and the EMG signal. However, if the muscle were to have begun contracting more rapidly and if the amplitude of the noise of the electrode were lower, this discrepancy would be less than 200 ms, comparable to the value of the physiological delay.

It is apparent that any attempt at estimating the precise time at which a muscle begins and ends activation is fraught with difficulties that cannot be fully addressed with current knowledge and that require further study.

Recommendations

- 4. Measure the activation time in all types of contractions.
- 5. Limit the resolution of the on-off times to 10 ms when comparing among muscles and subjects.
- Be sure that the signal originates from the muscle of interest by testing for crosstalk. Use the double differential technique, the frequency spectrum technique, or the multiple-electrode technique.

Problems for Resolution

- 1. Elimination or substantial reduction of crosstalk from other muscles is required. Until means to do so are available, a way to easily identify the presence of crosstalk signals is necessary. (This is an essential requirement for all applications of the surface EMG signal.)
- 2. An agreed-upon method for determining the on-off times of a muscle activation is required.

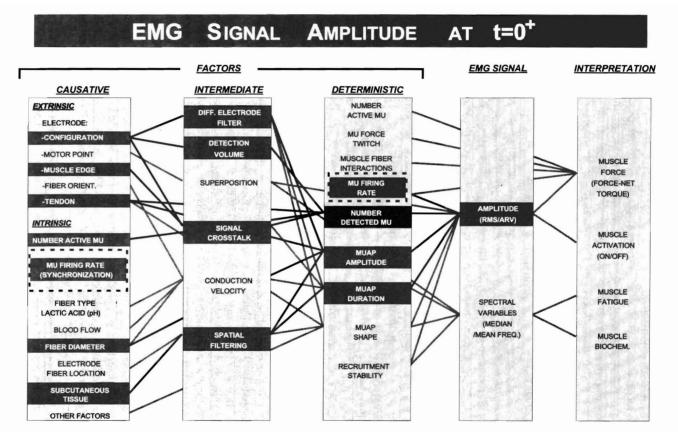
The Force/EMG Signal Relationship

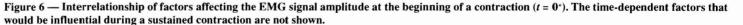
Where does the detected EMG signal originate? Is the EMG signal sufficiently stationary for the intended analysis and interpretation? Where does the measured force originate?

The force–EMG signal relationship has bound the disciplines of electromyography and biomechanics in an inextricable manner ever since they began their evolution toward quantification. A closed-form and/or simple equation describing this relationship would be desirable and extremely useful. However, such a simple form does not exist between force and the surface EMG signal, as they are currently measured, during a contraction. This is an application fraught with problems. The observation that the EMG signal amplitude generally increases as the force and/or contraction velocity of the muscle increases only provides a qualitative indication of a relationship between the variables. This qualitative relationship may prove useful if only qualitative descriptions of the muscle state are required. For example, if one needs to know whether a muscle generates more force during a substantially different task, it is possible to answer this question qualitatively by analyzing the EMG signal. However, if one asks precisely how much the force varies between two tasks, it is not possible to answer this question quantitatively with precision.

Many factors cause the relationship to be nonrigid. These have been presented in Figure 1 and are specifically outlined in Figure 6. The effect of all these factors can be neutralized by normalizing the amplitudes of the EMG signal and the force among contractions in which the electrode is not moved and if the relative distance between the electrode and the active muscle fibers remains fixed. If the electrode is moved, then the characteristics of the spatial filtering between the active fibers and the detection surfaces will change; also, the location with respect to the innervation zones, the myotendonous junction, and the midline of the EMG signal. If the force–EMG signal relationship is to be compared among different subjects, then all the intrinsic and some of the extrinsic factors that have been described previously can potentially affect the relationship. Among these, the amount of subcutaneous fatty tissue becomes a major concern, and any comparison should be made with caution.

The issue of crosstalk is omnipresent when dealing with surface EMG signals. As in other considerations of the signal, all possible efforts must be made to eliminate crosstalk or at least reduce its presence. With this problem aside, the next concern deals with the stationarity of the EMG signal. Two factors have the greatest effect on signal stationarity: (a) stability of the electrode position with respect to active muscle fibers (any such movement would affect the amplitude of the MUAPs and possibly bring the electrode to the territory of an active motor unit not previously detected), and (b) stability of the motor unit activation pattern. (For additional information on motor unit stability, see De Luca, Foley, and Erim, 1996.) The first factor may be substantially controlled by limiting the





muscle contraction to be as isometric as possible. For this reason, the isometric contraction is the most studied contraction even though it is less physiologically interesting than the anisometric contraction, which we use to perform the movement tasks that enable us to interact with our environment.

In an anisometric contraction, various mechanical, physiological, anatomical, and electrical modifications occur throughout the contraction that affect, in substantial ways, the relationship between signal amplitude and muscle force. For example, the force–length relationship of the muscle fibers varies nonlinearly, and the shapes of the MUAPs that construct the EMG signal are altered because the relative position of the electrode fixed on the skin surface changes with respect to the contracting muscle fibers. These effects are further aggravated if the displacement is accelerated, because the time delay between the signal and the force may limit use in some applications.

If it is absolutely necessary to process an EMG signal detected during an anisometric contraction, the researcher must make every attempt to limit the analysis to a near-isometric epoch of the record and extrapolate the interpretation of the analysis based on results from this epoch. If anisometric contractions are repetitive, such as those found in gait and cycling, then a fixed epoch in the period of the contraction should be chosen for analysis, and all comparisons should be made in this epoch. In this fashion, it may be possible to reduce the role of the signal nonstationarities with respect to the role of other effects being studied. In all cases, such analyses and comparisons should be performed with the utmost concern for the inherent limitations of this approach.

If a quantitative relationship between the EMG signal and force is required, then the contraction must be isometric. However, even under this constraint the relationship between force and EMG signal remains problematic. It is generally agreed that when the EMG signal is sufficiently smoothed, the relationship is monotonic, but the linearity appears to differ among muscles (assuming that there are no technical and other confounding factors such as crosstalk) (see Figure 7). However, because the amplitude of the surface EMG signal is a random variable, the instantaneous value of the amplitude is not monotonic with respect to the force value. Furthermore, the estimate of the signal amplitude will vary as a function of force due to intrinsic anatomical and physiological factors. More specifically, the relationship is nonrigid for two reasons:

1. In most muscles, the detection volume of the electrode is smaller than the volume of the muscle. Therefore, the number of MUAPs detected by the electrode is less than the number active in the muscle. Consider the likely case where the electrode only detects one tenth the active motor units and a new motor unit is activated in the detection volume of the electrode. Then, on average, the energy of the EMG signal will be increased by one unit and the force output of the muscle by one tenth of a unit. For a fixed-size electrode, this effect will be more pronounced in larger muscles. Additionally, if the newly recruited motor unit is located close to the electrode, then the relative increase of the EMG signal will be greater than the corresponding increase of the force, because the new MUAP will contribute more than an average unit of energy to the EMG signal. However, if the newly recruited motor unit is located far away from the electrode, then the force will increase but the amplitude of the EMG signal will not.

2. As the muscle force output increases beyond the level of a newly recruited motor unit, the firing rate of the recruited motor unit will increase but the force contribution of the motor unit will saturate. Each MUAP will continue to provide energy to the EMG signal, while the force contribution saturates to a near constant value. This nonlinear relationship causes the amplitude of the EMG signal to increase more than the force output. Consequently, the control strategy, described in terms of the mixture of firing rate dynam-

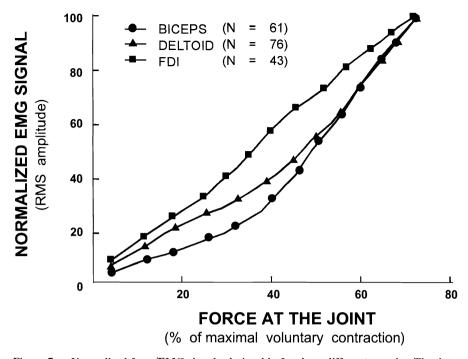


Figure 7 — Normalized force/EMG signal relationship for three different muscles. The data have been greatly smoothed, with a window width of 2 s. Note the difference in linearity of the relationship among the muscles.

ics and recruitment range that the central nervous system (CNS) uses to control different muscles, can also affect the EMG–force relationship. For example, the first dorsal interosseous muscle has firing rates with considerable dynamic range and recruits all its motor units below 50% of its maximal voluntary contraction. Larger muscles recruit their motor units through a wider range of force levels and their firing rates exhibit less dynamic ranges.

These two major factors are displayed graphically in Figure 8. The impact of all these effects can be reduced by filtering, but then the instantaneous and intimate relationship between force and the EMG signal is lost.

The other important issue to address concerns the origin of the measured force associated with the muscle or group of muscles being studied. The CNS regulates the torques at a joint during purposeful muscle contractions. During isometric contractions, the moment arm of the muscle and the instantaneous center of rotation of the joint remain constant (for practical purposes); thus, the net torque may be reasonably and directly related to the net force acting in the joint. But even in this simplified situation, the origin of the contributing forces must be understood before the measured net force is associated with the force produced by an individual muscle or a group of muscles. There are at least two points to be considered. First, does a muscle generate force homogeneously throughout its volume? This is a relevant consideration when an EMG signal detected at a specific location is compared with the force generated by the muscle throughout its volume. Second, what is the role of the agonistic and antagonistic muscles acting at the joint being considered? The contribution of the force of the synergistic and

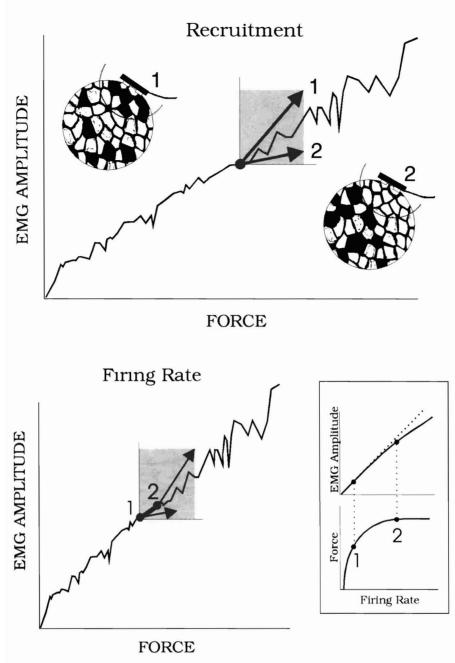


Figure 8 — (Top) Recruitment of a motor unit. A quanta of force is contributed to the muscle contraction; however, the contribution to the EMG signal amplitude depends on the proximity of the detection surfaces of the electrode to the nearest fibers of the recruited motor unit. (Bottom) A newly recruited motor unit will increase its firing rate as the force demand increases. The tetanization force increases rapidly as a function of the increasing firing rate, whereas the contribution to the amplitude of the EMG signal increases less rapidly.

antagonistic muscles to the muscle from which the EMG signal is detected, as well as any ligaments acting on the joint, must be negligible if parameters of the detected EMG signal are to be associated with the absolute value of the force from the monitored muscle. Alternatively, their contributions must remain constant to enable a relative comparison. This latter point introduces the notion of muscle substitution during a sustained contraction. This phenomenon has not been sufficiently studied and is not well understood. It may present a real concern in pathological cases where the CNS may employ, more readily, alternative strategies to accomplish a task when normally preferred options are limited or unavailable.

All the concerns associated with force measurement during isometric conditions are magnified in degree and augmented in number when one deals with anisometric contractions. At least three facts require careful consideration: (a) At most joints, when the muscle length changes so does the length of the moment arm, and in some joints, so does the instantaneous center of rotation (De Luca & Forrest, 1973), (b) from physiological considerations, as the length of the muscle fibers changes so does the force generated by the fiber, and (c) the inertial components of the net torque must be taken into account. These facts present an enormous challenge to any attempt at identifying the force originating from a specific muscle. Current knowledge falls far short of providing quantitative interpretations of the force generated within a muscle or a specific group of muscles from torque measurements in dynamic conditions.

Comparison Among Subjects, Muscles, and Contractions

Throughout this section, many concerns have been raised about the variability of the detected EMG signal and the measured force as a function of experimental conditions, repeated contractions within a subject, and contractions among different subjects. To enable comparison of data collected in these various conditions, the EMG signal is often normalized with respect to force (or torque). However, even with this practice, variations in the relationship can be found in the same muscle among subjects. When comparing among subjects, the lengths of moment arms in individual subjects may present a problem because they may vary while the EMG signal remains related to the force produced by the muscle. There is at least one other important reservation to the use of normalization: Its predilection to render similar the data from different subjects tends to suppress distinctions in the data that would be associated with abnormal or pathological cases. This is an important concern when the EMG signal is to be used for analysis of clinical data. When a comparison among subjects is required, the force (torque) as well as the amplitude of the EMG signal is normalized to provide a basis of comparison among the differing force capabilities of the subjects' muscles. Figure 7 provides an example of the different normalized and smoothed relationships for three different muscles.

It has been common practice to normalize force (or torque) with respect to the maximal isometric force that a subject can generate at the monitored joint. The concerns and pitfalls of this approach are apparent. The two most obvious are whether a subject actually generates maximal force when asked to do so and whether the monitored force is in some direct manner related to the muscles being studied. It has been suggested that it may be possible to verify whether a subject is generating maximal force by supramaximally stimulating, at a high frequency (whenever possible), the nerve to the muscle(s) during an attempted maximal force contraction. No noticeable increase in force output as a consequence of stimulation might indicate that maximal force was being produced. This reasoning is not convincing, because the force output of a muscle during a synchronous

volley of firings, due to the stimulation of a nerve, does not necessarily produce the same total force as when the muscle is being asynchronously activated by the CNS.

One practical, although by no means foolproof, means for estimating the maximal force value is as follows: Constrain the joint of interest with restraints that impede, to the fullest extent possible, the force contribution of muscles not directly involved with the force to be measured. This is not a simple task. It requires comprehensive knowledge of the biomechanics and anatomy of the muscles and joints being considered as well as some aptitude for designing a suitable restraining device. This task should be executed or supervised by experienced individuals; even then, such attempts are susceptible to subjectivity and outright failure. When the joint is constrained in a satisfactory fashion, ask the subject to produce three attempts at maximal contraction. The contractions should be brief, no more than 5 s in duration. Smooth the force signal with a filter having a sliding window of 1 s duration. Then measure the greatest value of the force record. A rest period of at least 2 min between each contraction is required for recovery. The contractions should be brief so that the force contribution of the fast-fatiguing fibers does not diminish as the force builds to the maximal value, but sufficiently long to be filtered as described. Let the subject choose his or her own manner of producing the force; in this fashion the CNS may optimize its control strategy, if it is so designed. Accept the largest value as the maximal value. At the NeuroMuscular Research Center, we have been satisfied with this approach. But clearly a more objective or more physiologically sound approach must be developed.

Recommendations

- A quantitative comparison of proportional relationship between the EMG signal and force should be limited to well-regulated isometric contractions in which the joint being tested is constrained to limit the torque contributions of muscles other than the muscle of interest.
- 8. The force and the EMG signal are not monotonically related in an instantaneous sense. Analyses relying on an instantaneous relationship should treat the relationship as stochastic. To obtain a near-monotonic relationship between the force and the EMG signal, filter the signal with a window width of 1 s.
- 9. When obtaining measurements from constant-force isometric contractions, confirm that there are no motor units active on the verge of their threshold. This situation is to be avoided particularly in cases where the force level is low and the amplitude of the marginally recruited MUAP is high.
- 10. Avoid anisometric contractions. If data from such contractions are to be analyzed quantitatively, use contractions that have the least amount of shortening and the slowest velocity, and interpret the results with caution.
- 11. If repetitive anisometric contractions must be analyzed, choose an epoch of data from a fixed portion on the cycle of activity and monitor the signal parameters as the cycles progress.
- 12. When normalizing the amplitude of the EMG signal, do so at values less than 80% MVC. Above this level, the EMG signal and the force (torque) are exceptionally unstable and do not provide a suitable reference point.
- 13. Measure the MVC by choosing the greatest value of three consecutive attempts at reaching the maximal value, with a rest period of at least 2 min between contractions. Let the subject choose his or her own force rate to reach maximal value. The posture of the subject should be similar if not identical to that assumed during the actual test. Repeat this measurement each time the experimental conditions change.

Problems for Resolution

- 3. Development of a method of surface detection that follows the movement of the muscle fibers. (This approach would improve the stationarity of the signal and provide a rational basis for quantitatively relating the detected EMG signal to the measured torque during anisometric contractions, although the effects of force–length and inertial forces remain and require consideration.)
- 4. Development of means for on-line measurement of the stationarity of the surface EMG signal. (This approach would provide a measure of signal stationarity while the signal is being detected.)
- 5. Development of a method by which the absolute force produced by a muscle may be meaningfully estimated with an accuracy of $\pm 5\%$ directly from the surface EMG signal detected above a muscle. Constant-force and force-varying isometric contractions should be addressed first. (This approach would require new means for detecting the EMG signal.)
- 6. How do muscle fibers transmit force throughout the muscle? (6a) When a muscle fiber contracts, is the force transmitted longitudinally along the fibers or is it also transmitted radially via interconnections among the fibers? (6b) There is mounting evidence that in most muscles the fibers do not traverse the length of the muscle. How is the force produced by a fiber transmitted to the tendon of the muscle?
- 7. Does a muscle generate force homogeneously throughout its volume? (This is a function of the homogeneity of the muscle tissues, the distribution of fibers within a motor unit, the distribution of motor units within the volume of the muscle, and the architecture of the muscle attachment to bone or other tissues. This knowledge is particularly necessary when considering muscles that are wide and short. In these cases, the resultant force vector may alter its angle of action as a function of force level. This knowledge would also be useful for refinements of biomechanical models.)
- 8. Description of the anisotropy and inhomogeneity of muscle, fascia, fat, and skin tissues. (This knowledge would be useful to describe how the amplitude and frequency spectrum of the EMG signal is altered by the tissues between the active fibers and the electrode.)
- 9. Refinement and further development of anatomically correct and detailed biomechanical models of the musculoskeletal system. (Such models will enable the calculation of force vectors and the instantaneous center of rotation of the joints, so that torques and forces may be calculated during motion.)

The EMG Signal as a Fatigue Index

Is the EMG signal detected and recorded with maximal fidelity? How should the EMG signal be analyzed? Where does the detected EMG signal originate? Is the EMG signal sufficiently stationary for the intended analysis and interpretation?

In the study of human biomechanics, it is often desirable to assess the fatigue of muscles that are involved in performance of a task. Physiologists have become accustomed to using the force output of a muscle as the index of muscle fatigue. In particular, the point at which a contraction can no longer be maintained (the failure point) is generally the point at which the muscle is said to fatigue. This approach implies that fatigue occurs at a specific point in time, a notion inconsistent with the concept of fatigue accepted by engineers and physical scientists. The use of the failure point carries with it some practical disadvan-

tages. For example, fatigue is detected only after it occurs. This approach has little use in clinical and ergonomics applications, where it is often desirable to have indications that precede failure so that appropriate remedies may be made. In addition, there are at least three other confounding factors:

- During a voluntary contraction, the force of an individual muscle is not often directly accessible, and the monitored torque may not faithfully represent the force of the muscle of interest.
- During a submaximal contraction, it is possible to maintain the torque acceptably constant, in a macroscopic sense, but there are time-dependent physiological and biochemical processes that microscopically alter the means for generating force during a sustained contraction. These processes include (a) recruitment and derecruitment of motor units (this has not been proven beyond doubt but remains plausible), (b) a decrease in the firing rates of most motor units, and (c) an increase in the amplitude of force twitches of motor units during sustained contractions.
- The failure point is a function of both physiological and psychological factors, and it is difficult to know accurately the causal relationship of each to the failure point.

The alternative and, in my assessment, preferable approach is to exploit the wellknown spectral modification property of the EMG signal detected during a sustained contraction. (The spectral modification manifests itself mostly as a compression accompanied by an alteration in the skewness of the shape.) Monitoring and quantifying the spectral modification during sustained contractions provide fatigue indices that describe the time course of the fatigue-related physiological and biochemical processes. Such an approach provides at least two advantages over the contractile fatigue approach:

- Contractile force can only be conveniently measured by monitoring the torque about a joint to which more than one muscle can contribute. In contrast, the EMG signal can be detected from individual muscles; thus, the spectral variable fatigue index can be used to describe the performance of individual muscles.
- Spectral modification progresses continuously from the onset of contraction, thus
 indicating the rate of the fatigue process early in the contraction. Contractile fatigue, as currently measured, requires the subject to expend considerable effort before fatigue is measurable.

Since the mechanisms that cause EMG spectral modification and those that generate the force both undergo changes during the progression of fatigue, it is inevitable to ask if a relationship exists between the two. The answer is undoubtedly "yes." The more interesting question is whether the relationship is causal; a considerable amount of work is required before this question can be answered. Nonetheless, the lack of proof of a causal relationship does not logically preclude the use of the spectral fatigue index, especially when empirical evidence reveals its usefulness.

The spectral modification may be monitored and quantified by tracking some characteristic indicators of the frequency spectrum, such as the median, mean, or mode frequency of the spectrum, or alternatively by calculating a ratio of low-frequency to highfrequency bandwidths, or by integrating the area corresponding to the decrease of the median frequency. I prefer the median frequency because it is less sensitive to noise, less sensitive to signal aliasing, and in most cases more sensitive to the biochemical and physiological processes that occur within the muscles during sustained contractions (De Luca, 1984; Merletti, Knaflitz, & De Luca, 1992). However, the estimate of the median frequency is more variable, largely due to instability of the EMG signal spectrum at lower frequencies. Nonetheless, a three-point digital filter reduces the variability of the estimate to that of the mean frequency. For a comprehensive review, refer to De Luca (1984). The spectral modification events described above are represented graphically in Figure 9.

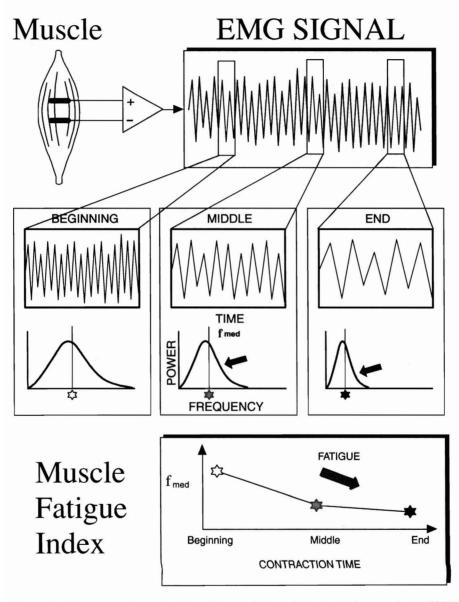


Figure 9 — Diagrammatic explanation of the spectral modification that occurs in the EMG signal during sustained contractions. The muscle fatigue index is represented by the median frequency of the spectrum.

It now remains to explain the factors that cause this phenomenon. Two main properties of the EMG signal can affect the frequency spectrum: the firing behavior of the motor units, and the shape of the MUAP. The dominant effect of the motor unit firing rate is limited to the frequency neighborhood of the average firing rate (15 to 25 Hz). The second harmonics of the firing rates are considerably smaller than the first harmonics and occur at double the frequency of the first harmonics, where they are overwhelmed by the energy of the MUAP shapes. The variance of the firing rates determines the broadness of the frequency peak representing the firing rate; therefore, any influence will be limited to the same region. Synchronization of motor unit firings, that much-abused property of the EMG signal that has been conveniently and improperly accused of contributing to the spectral modification and associated increase in the EMG signal amplitude, cannot significantly affect the spectral characteristics of the EMG signal for two reasons. First, it can be shown mathematically that interactions among the firing rates will contribute frequency components in the frequency region of the firing rate values. More importantly, synchronization has been shown to occur in small doses (less than 8% of firings) and in bursts of two to three sporadic firings (De Luca, Roy, & Erim, 1993). All these firing-related effects are reflected in the spectrum of the EMG signal at frequencies in the neighborhood of 15 to 25 Hz for most contractions in most muscles. In order to maintain signal stability, improve stationarity, and eliminate motion artifacts, the EMG signal should be detected with a low-frequency cutoff of 20 Hz, as has been recommended in this paper. Therefore, most of the contribution of the motor unit firing characteristics would not be included in the detected EMG signal.

It remains for the shapes of the MUAPs to explain the spectral modification of the EMG signal. The shape of the compound action potential of a muscle during a sustained contraction illustrates that the time duration of the action potential increases as the time of the contraction increases. It can be shown mathematically that this behavior of the motor unit action potential shape compresses the spectrum of the EMG signal. Numerous reports in the literature attest to the increased time duration of the MUAP. Recently at the NeuroMuscular Research Center, we have shown that approximately 65% of this increase can be accounted for by the independently measured, concurrently decreasing conduction velocity of the action potential (Brody, Pollock, Roy, De Luca, & Celli, 1991). We have also shown that a decrease in conduction velocity is causally related to a decrease in the pH of a bath fluid surrounding the muscle. It is further known that during sustained contractions, the pH of the interstitial fluid decreases as lactic acid accumulates in the membrane environment.

To further explain the factors that alter the shape of the MUAP, I will refer back to Figure 1, which describes the interaction of all factors affecting the EMG signal, and isolate the ones that impact the MUAP shape at the beginning of a contraction. Some deal with electrode configuration, some with location and orientation, others with the architecture of the muscle and tissues between the active fibers and the electrode, and others with physiological and biochemical events. These factors are displayed in a block diagram in Figure 10. If an anisometric contraction is performed, all of these factors will influence the shape of the MUAP, and the causative influences are very difficult to identify. For this reason, I do not recommend analysis of the spectrum of the EMG signal detected during anisometric contractions. However, during an isometric contraction, the spatial filtering properties of the detection arrangement are fixed and remain constant. If the isometric contraction is maintained at constant force, recruitment of the motor units is unlikely to occur and the average size of the active muscle fibers remains constant. Thus, for a constant-force isometric contraction, the only factors that affect the MUAP are conduction velocity and the depolarization zone of the muscle fibers. Furthermore, if the contraction is sufficiently forceful to occlude blood flow in the muscle, the only known factor that affects conduction velocity is the amount of interstitial H⁺ and K⁺, with the causal influence of H⁺ being far more thoroughly studied. In most muscles in the limbs and back, blood flow becomes occluded at approximately 30% MVC. Muscle temperature plays an insignificant part. For most submaximal, short-term contractions (less than 15 s), the temperature increase in the muscle is no greater than 0.5 °C, which would increase conduction velocity by approximately 1.5% (Merletti, Sabbahi, & De Luca, 1984). However, there remains the effect of the size of the depolarization zone on conduction velocity; the amount of its influence is not yet known.

Thus, for constant-force isometric contractions above 30% MVC, spectral modification of the EMG signal is mostly due to the accumulation of lactic acid (the metabolic by-product of a muscle fiber contraction). This point should be made with some reservation because the possible effects of other ions and other factors have not been studied. Nonetheless, current knowledge indicates that by correctly analyzing the EMG signal, detected nonpainfully and noninvasively outside the body, researchers can quantitatively measure and monitor the amount of biochemical by-products produced in an individual muscle during a sustained contraction. This is an exciting association that leads to many practical applications.

It should be apparent that it is necessary to consider and follow some practical guidelines when using the spectral fatigue indexes. Consider the following points, which are apparent from the interactions shown in Figures 1 and 6:

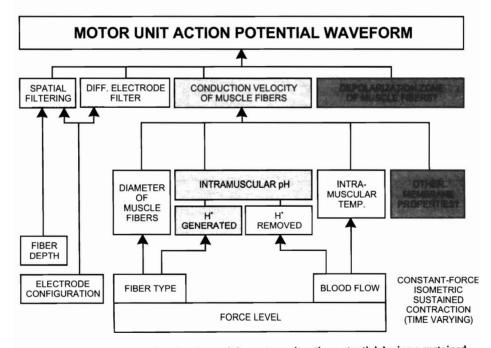


Figure 10 — Factors that affect the shape of the motor unit action potential during a sustained contraction. The shaded areas are indicative of known factors that are modified as a function of time during a sustained constant-force contraction above 30% MVC. The dark shaded areas indicate other possible influential factors whose effects have not yet been established.

1. Electrode configuration: The distance between the detection surfaces of an electrode determines the bandwidth of the filtering characteristics of the differential configuration. Therefore, if the median frequency, or any other measure of the frequency spectrum of the EMG signal, is to be compared among contractions, the same electrode configuration and dimensions must be used.

2. Electrode location: As previously recommended, the electrode should be placed between the myotendinous junction and the nearest innervation zone. This is particularly important when the spectral variables of the signal are to be measured. If a differential electrode is placed in the proximity of a motor point, it will detect the superposition of action potentials that have positive and negative phases and that pass by the electrode with relatively small time delays among them. The resulting short-time delay subtractions generate high frequencies that augment the energy content in the high-frequency end of the EMG signal spectrum and consequently yield a higher value for the median frequency. The innervation zones may be located by applying an electrical stimulation to the skin above the muscle and locating the points where the minimal amount of current is required to elicit a minimally perceivable muscle fibrillation. Alternatively, the surface above the muscle may be scanned with a surface electrode array during a low-level contraction to determine where the phases of the MUAP reverse (Masuda & De Luca, 1991).

3. Electrode orientation: The detection surfaces of the electrode should be oriented so that they intersect perpendicularly the length of the muscle fibers. The length of the fibers that traverse the distance between the detection surfaces determines the amount of time required for the action potentials to traverse the distance. Given that the inter-detection-surface spacing is fixed, the value of the apparent conduction velocity will be a function of the cosine of the angle between the fibers and the detection surfaces. As previously mentioned, the estimated value of the conduction velocity affects the frequency scaling of the spectrum and consequently the value of the spectral parameters. If the electrode is located near the myotendinous junction there is one other concern, that is, the truncation of the action potential as it reaches the higher impedance of the tendon. This truncation tends to introduce sharp features to the action potentials, which in turn increase the high-frequency contribution to the spectrum of the EMG signal.

4. Crosstalk: Because signals originating from other muscles generally travel through greater distances to reach the electrode, they are subjected to more spatial filtering (low-pass filtering). Hence, they will contain less energy in the higher frequencies. The inclusion of crosstalk signals in the detected EMG signal will skew the frequency spectrum toward the lower frequencies, thus reducing the value of the median frequency and possibly altering the rate of decrease during the contraction. This influence is particularly disruptive if the agonist and antagonist muscles alter their contribution to torque during the contraction, an occurrence that has special concern when pathology is present.

5. Subcutaneous tissue: Comparison of spectral parameters of EMG signals detected in similar locations among subjects, or from different locations within subjects, requires caution. The amount of subcutaneous (fatty) tissue between the electrode and the active fibers determines the amount of spatial filtering to which the signal is subjected. The greater the thickness of the tissue, the greater the low-pass filtering. Thus, additional subcutaneous tissue reduces the value of the median frequency.

6. Fiber diameter: The conduction velocity of the muscle fiber is proportional to (some power of) its diameter. Therefore, muscles with larger diameter fibers, such as those generally belonging to higher threshold motor units, will have greater average conduction velocities which, in turn, will shift the frequency spectrum toward the high-

frequency range and consequently increase the value of the median frequency. This effect can be noted when the force output of the muscle increases but the sensitivity is low (0.16 Hz/% MVC) (De Luca, Sabbahi, & Roy, 1986). Nonetheless, distinctions on the basis of fiber size would be expected as the result of exercise, when the muscle fibers hypertrophy; as the result of disuse, where the muscle fibers atrophy; and between muscles of different gender, because males generally have larger diameter fibers. Note that the latter three effects still require convincing experimental verification.

7. Signal stability: Even during a constant-force isometric contraction, the issue of EMG signal stability raises concerns that require close monitoring of the signal as it is detected. For example, if the force output of the muscle is not perfectly steady, motor units on the verge of recruitment and in the vicinity of the electrode could begin to fire and be detected. If such units have fibers that are near the muscle surface, they would contribute significant energy to the signal, and their frequency characteristics could alter the shape of the EMG signal spectrum unpredictably. Consequently, the value of the median frequency would also vary unpredictably.

8. Anisometric contractions: If the spectral technique is to be applied to dynamic contractions, which are often of interest in biomechanical studies, then the median frequency should be calculated only when the EMG signal is reasonably stationary and detected at the same phase during a repetitive dynamic contraction.

Recommendations

- 14. It is preferable to use the median frequency as the fatigue index calculated from the frequency spectrum of the EMG signal. The mean frequency could also be used.
- 15. When calculating spectral variables, limit the muscle contraction to one that is isometric, constant force, and greater than 30% MVC.
- 16. When comparing spectral variables among subjects, use the same electrode (especially with the same interdetection surfaces separation) and place it in a similar location with respect to the innervation zones and the insertion of the tendon. (Surface anatomical landmarks are generally not appropriate because the innervation zones may not be located in similar parts of the muscle among subjects.) Also consider the effect of the subcutaneous tissue.

Problems for Resolution

- 10. Is a single spectral variable, such as the median frequency, sufficient for monitoring the spectral modification of the EMG signal during a constant-force isometric contraction? (During a sustained contraction, it is possible that the shape of the frequency spectrum changes so that the signal does not undergo a simple frequency compression. If so, a variable that monitors the shape of the spectrum will be required.)
- 11. What factors, other than conduction velocity, affect the shape of the MUAP during sustained constant-force isometric contractions? (Consider the length of the depolarization zone and better document the influence of K⁺ ions that accumulate outside the muscle membrane during sustained contractions.)
- 12. Can a reliable quantitative spectral fatigue index that is causally related only to biochemical and physiological processes within the muscle be developed for force-varying isometric contractions and for anisometric contractions? (12a) Is it possible to account for the effect of the conduction velocity of progressively higher threshold motor units, and how variable is the change in average conduction velocity as

higher threshold motor units are recruited? (12b) Is it possible to account for the influence of nonphysiological and nonbiochemical factors that affect the EMG signal during anisometric contractions?

- 13. During a sustained contraction, is the behavior of the EMG signal spectral parameters related to the behavior of the force? Is the relationship causal? (13a) Does the behavior of the spectral parameters predict the behavior of the force? (These are basic questions that will provide an important link between electromyography and biomechanics.)
- 14. Can spectral modification be used to estimate the fiber type ratio in the muscle? (This technique could serve as a noninvasive "electrical biopsy." It could be used to estimate the fiber type composition of muscles as a function of exercise or to identify pathological and age-affected fiber-type composition.)
- 15. Can variables of the EMG spectrum be used to estimate the average diameter of muscle fibers? (This technique could be used to measure noninvasively muscle hypertrophy and atrophy.)
- 16. Is it possible to map electrically the surface above the muscle so that innervation zones, muscle fiber length, and orientation may be easily identified? (This technique would be useful in identifying locations for electrode placement and to study normal and abnormal muscle morphology.)

Issues for International Agreement

This paper has presented a cadre of issues that require consideration when EMG signal and measured force (torque) are used to describe the state of an active muscle. Many of the issues cannot be fully or substantially addressed with current knowledge, but fortunately some can. In these cases, recommendations have been presented and I put them forth to be scrutinized by the scientific community at large. These recommendations are offered in the spirit of beginning a broad-based accord for regulating the use of surface electromyography. They should also prove useful for teaching the practitioner how to use surface electromyography and for guarding against misuses and abuses that lead to incorrect interpretations.

I propose that the following topics have been sufficiently studied, have had a general impact on surface electromyography and biomechanics, and are appropriate for generating a consensus:

- 1. Electrode configuration and dimensions
- 2. Electrode placement and orientation
- 3. Means for processing the EMG signal for amplitude and spectral analysis
- 4. Means for determining the delay between force and the EMG signal
- 5. Procedure for determining MVC
- 6. Procedures for establishing repeatability of the EMG signal parameters
 - among contractions when the experimental conditions are fixed
 - among contractions when the electrodes are reapplied
 - among muscles
 - · among subjects

I propose that an international committee of respected, experienced, and practicing researchers in the fields of surface electromyography and biomechanics be organized to refine and/or modify the recommendations and sanction their approval.

References

Basmajian, J.V., & De Luca, C.J. (1985). Muscles alive (5th ed.). Baltimore: Williams & Wilkins.

- Brody, L.R., Pollock, M.T., Roy, S.H., De Luca, C.J., & Celli, B. (1991). pH induced effects on median frequency and conduction velocity of the myoelectric signal. *Journal of Applied Physiology*, 71(5), 1878-1885.
- Broman, H., Bilotto, G., & De Luca, C.J. (1985). A note on non-invasive estimation of muscle fiber conduction velocity. *IEEE Transactions on Biomedical Engineering*, 32, 341-344.
- De Luca, C.J. (1984). Myoelectric manifestations of localized muscular fatigue. CRC Critical Reviews in Biomedical Engineering, 11, 251-279.
- De Luca, C.J., Foley, P.J., & Erim, Z. (1996). Motor unit control properties in voluntary isometric isotonic contractions. *Journal of Neurophysiology*, **76**, 1503-1516.
- De Luca, C.J., & Forrest, W.J. (1973). Force analysis of individual muscles acting simultaneously on the shoulder joint during isometric abduction. *Journal of Biomechanics*, 6, 385-393.
- De Luca, C.J., & Knaflitz, M. (1992). Surface electromyography: What's new? Torino, Italy: CLUT. De Luca, C.J., & Mambrito, B. (1987). Voluntary control of motor units in human antagonist muscles:
- Reciprocal activation and co-activation. Journal of Neurophysiology, 58, 525-542.
- De Luca, C.J., & Merletti, R. (1988). Surface EMG crosstalk among muscles of the leg. Electroencephalography and Clinical Neurophysiology, 69, 568-575.
- De Luca, C.J., Roy, A.M., & Erim, Z. (1993). Synchronization of motor unit firings in human muscles. *Journal of Neurophysiology*, 70(5), 2010-2023.
- De Luca, C.J., Sabbahi, M.A., & Roy, S.H. (1986). Median frequency of the myoelectric signal: Effects of hand dominance. *European Journal of Applied Physiology*, 55, 457-464.
- De Luca, C.J., & Van Dyk, E.J. (1975). Derivations of some parameters of myoelectric signals recorded during constant-force isometric contractions. *Biophysical Journal*, 15, 1167-1180.
- Masuda, T., & De Luca, C.J. (1991). Recruitment threshold and muscle fiber conduction velocity of single motor units. *Journal of Electromyography and Kinesiology*, 1, 116-123.
- Merletti, R., Knaflitz, M., & De Luca, C.J. (1992). Electrically evoked myoelectric signals. CRC Critical Reviews in Biomedical Engineering, 19(4), 293-340.

Merletti, R., Sabbahi, M.A., & De Luca, C.J. (1984). Median frequency of the myoelectric signal. Effects of ischemia and cooling. *European Journal of Applied Physiology*, **52**, 258-265.

Acknowledgments

I owe a debt of gratitude to all who have influenced my thinking on the use and interpretation of the EMG signal. I have chosen not to refer to anyone directly because the material discussed in this paper has been influenced by printed statements from numerous sources, undocumented discussions with many colleagues and students, and personal impressions that I have accumulated by observing the work of many researchers. To provide a limited set of references would necessarily limit my expression of respect and appreciation to all who have influenced my thinking on this topic.

I am grateful to my associates Drs. J.J. Collins, M. Knaflitz, and S.H. Roy and Mr. L.D. Gilmore for numerous discussions on this topic and for their many comments on and suggestions for the material in this manuscript. I am particularly grateful to Dr. Z. Erim, who in addition to providing comments assisted me with the refinement of the paper. Thanks to Messrs. L. Andrus, E. Kupa, R. Buijs, P. Foley, and W. Conley for preparing the figures.

Financial support for this work was supplied by Liberty Mutual Insurance Co. and the Rehabilitation Research and Development Service of the Department of Veterans Affairs.