

MYOELECTRICAL MANIFESTATIONS OF LOCALIZED MUSCULAR
FATIGUE IN HUMANS

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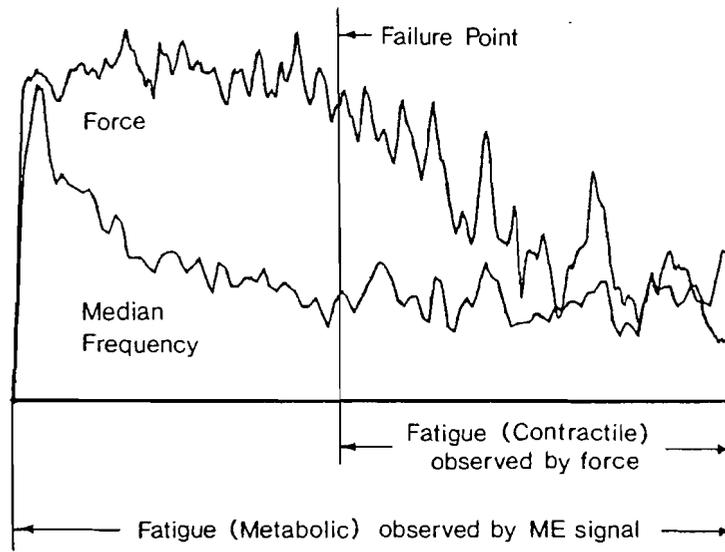
I. THE CONCEPT OF FATIGUE

The concept of fatigue as applied to monitoring or measuring the deterioration of a performance of the *human operator* has been ambiguous and often misapplied. Mention the word "fatigue" to a group of health specialists and life scientists, and many diverse and divergent descriptions and explanations will emerge. In man, the issue of fatigue is complex due to the various physiological and psychological phenomena which contribute to it and which demonstrate it. In general, most if not all currently used methods for measuring fatigue in the active human are, by their very nature, inherently doubly subjective. That is, they rely on the cooperation of the individual performing a prescribed task and on the disposition of the observer when assessing the performed task. (It is usually possible for the observer to induce the subject to make an exertion beyond the initial presumption of his capability and/or interest.)

Another source of confusion arises from the fact that far too many health specialists and life scientists appear to have accepted the concept of fatigue as being associated with, demonstrated by, or represented by an event occurring at or over an identifiable period of time. For example, it is common to think of when an individual fatigues or to indicate that an individual is fatigued when a particular task cannot be performed or maintained at a specific time. Such a notion of fatigue is inconsistent with that which has been successfully employed by engineers and physical scientists, who have considered the concept of fatigue as a time-dependent process. For example, consider a steel girder that supports the main structure of a bridge. It may well remain in place with no apparent, externally visible structural modification for over 50 years; then suddenly, in one instant a fracture develops, the girder fails and the bridge collapses. If one, observing from a distance, were to look at the main structure of the bridge for a sign of fatigue, none would be easily noted during the 50-year period. What would be noted in this mode of observation would be the *failure point*. All the while, however, the crystalline structure of the steel girder was undergoing an alteration caused by chemical and physical processes. In order to monitor the progression of this alteration, specimens of data from within the girder itself or externally observable modifications related to the internal alteration are required.

An analogy in terms of muscle fatigue in the human body would be the task of maintaining a muscle contraction constant for as long as possible. Throughout this task, the involved muscles are continuously fatiguing, but at some instant in time the *failure point* will occur when the desired force output may no longer be maintained and contractile fatigue becomes observable.

Access to biochemical and physiological data within the muscle or the nervous system could reveal time-dependent changes indicative of a fatigue process, even though the ex-



LOCALIZED MUSCULAR FATIGUE

FIGURE 1. Distinction between contractile fatigue and metabolic fatigue. In this case, the force was exerted during an isometric contraction of the first dorsal interosseous muscle. The task consisted of maintaining the force output at 50% of the maximal value for as long as possible, and when this was no longer possible, the subject attempted to produce as much force as possible. The failure point denotes the time when the force output was no longer maintained at the desired average value. The median frequency value was calculated from the power density spectrum of the myoelectric signal. The time duration of the contraction was 150 sec.

ternally observable mechanical performance would not be altered until the *failure point*. An example of this is presented in Figure 1, which demonstrates the force output of the first dorsal interosseous muscle during an attempt at maintaining a 50% maximal voluntary contraction (MVC) output constant and the median frequency of the power density spectrum of the myoelectric (ME) signal from the same muscle. (It will be explained later that the median frequency, as well as other characteristic frequencies of the power density spectrum, appear to provide an appropriate representation of biochemical events within the muscle.) Note that even when the force output remains relatively constant, the median frequency is continuously decreasing in value.

This notion of a fatigue process and failure point may well prove useful in describing physiological, biochemical, and mechanical events in the human body.

This chapter will present a review of the published information concerning the relationship of the ME signal detected on the surface of the skin and the fatigue related events which occur in the muscle. An attempt will be made to explain and unify some of the seemingly disjointed data from different disciplines.

II. BACKGROUND

Over a half century ago, Muscio⁹⁶ argued that the then current interpretation of the word "fatigue" was too general in meaning for scientific use and should be abandoned. This timely advice induced involved professions to subdivide the concept of fatigue into subsets. This approach was exemplified by Bills¹⁰ who suggested that fatigue be divided into three major categories. The first was subjective fatigue, characterized by a decline of alertness, mental concentration, motivation, and other psychological factors. The second was objective

fatigue, characterized by a decline in work output. The third was physiological fatigue, characterized by changes in physiological processes. These categories have been further subdivided into areas with identifiable origins and symptoms (see Simonson and Weiser,¹²²).

One type of physiological fatigue is induced by sustained muscular contractions. It is associated with such external manifestations as the inability to maintain a desired force output, muscular tremor and localized pain. The effects of this fatigue are localized to the muscle or group of synergistic muscles performing the contraction. This category of fatigue has been termed *localized muscular fatigue* by Chaffin.²⁰ Although this term originally had its roots in the the field of Ergonomics, it was subsequently popularized by a research group at Chalmers Institute of Technology and Sahlgren Hospital in Sweden. However, according to Merton⁸⁹ and various other investigators, even this category of fatigue may have its source peripherally (in the muscle tissue or neuromuscular junction) or centrally (in the brain and spinal cord).

In the study of localized muscular fatigue, analysis of the ME signal, detected on the surface of the skin over a muscle, has been extensively employed. Since the historic work of Piper¹⁰⁹ in 1912, the frequency components of the surface ME signal have been known to decrease when a contraction is sustained. Cobb and Forbes²³ noted this shift in frequencies toward the low end with fatigue, and also observed a consistent increase in amplitude of the ME signal recorded with surface electrodes. Many other investigators have also noted an increase in ME signal amplitude (Knowlton et al.,⁵⁹ Scherrer and Bourguignon,¹¹⁹ Zhukov and Zakharyants,^{143a} Lippold et al.,⁸¹ DeVries,³³ Kadefors et al.,⁵⁶ Kuroda et al.,⁶⁷ Lloyd,⁸² Vredendregt and Rau,¹⁴³ Stephens and Usherwood,¹²⁷ Viitasalo and Komi,¹⁴² Stulen and De Luca,¹²⁹ Clamann and Broecker,²² Maton,⁸⁷ Hagberg,³⁰ and others). The frequency shift (towards the lower frequencies) has also been observed often and in a variety of muscles throughout the human body (Kogi and Hakamada,⁶⁰ Sato,¹¹⁸ Kadefors et al.,⁵⁶ Kwatney et al.,⁶⁸ Lindström et al.,⁷⁵ Johansson et al.,⁵⁴ Chaffin,²⁰ Lindström et al.,⁷⁴ Viitasalo and Komi,¹⁴¹ Lindström et al.,⁷⁷ Givens and Teeple,³⁶ Komi and Tesch,⁶² Petrofsky and Lind,¹⁰⁵ Bigland-Ritchie et al.,⁹ Hagberg,⁴⁰ Inbar et al.,⁵¹ Kranz et al.,⁶⁵ Palla and Ash,¹⁰² Stulen and De Luca,¹³² Mills,⁹⁰ Hagberg and Ericson,⁴¹ De Luca et al.,³² and others). These two phenomena, which are pictorially represented in Figure 2, are in fact related. Lindström et al.⁷⁴ and De Luca²⁹ explained the interrelationship by noting that during a sustained contraction the low-frequency components of the ME signal increase and, hence, more ME signal energy will be transmitted through the low-pass filtering effect of the body tissue. Therefore, the magnitude of the two related phenomena is dependent on many factors, such as force level of contraction, time into the contraction, the type of electrode used to obtain the ME signal, the thickness of the subcutaneous tissue, and the particular muscle investigated.

A minor digression is necessary at this point. It is commonly observed that the spectral shift is most dramatic near the beginning of a sustained contraction, whereas the amplitude of the ME signal shows a more pronounced increase near the end of a sustained contraction. Such divergent behavior of these two measurements would seem to indicate that they might have separate origins, were it not for the fact that the firing rates of the motor units decrease, even during constant-force contractions. This decrease in the firing rate is more pronounced near the beginning of the contraction. The decreasing firing rates will decrease the amplitude of the ME signal and thus offset the increase induced by the frequency shift.

Most of the work in this area has been performed on data obtained during constant-force contractions. Three explanations have been proposed to account for the increase in amplitude and the frequency-shift of the ME signal observed during a sustained, constant-force, isometric contraction. They are motor unit recruitment, motor unit synchronization and changes in the conduction velocity of muscle fibers. To this list should be added the regularity (coefficient of variation) of the motor unit discharge.

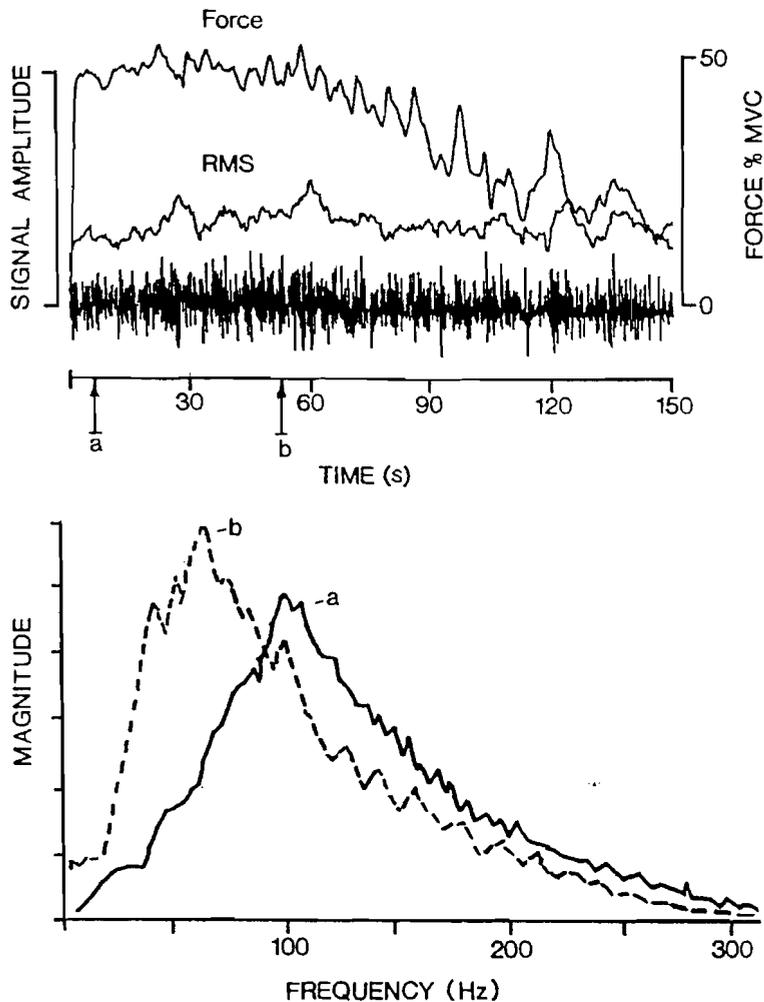


FIGURE 2. (Top) Myoelectric signal amplitude and force during an attempted constant-force contraction in the first dorsal interosseous muscle. (Bottom) Power density spectra of the myoelectric signal at the beginning and at the end of the constant-force segment of the contraction.

Edwards and Lippold,³⁴ Eason,^{33a} Vredendregt and Rau,¹⁴³ and Maton⁸⁷ have attributed the increase in amplitude to recruitment of additional motor units. They postulated that as a contraction progressed, additional motor units would be required to maintain the force output constant. Although this is plausible, current available information does not support this postulate as being a necessary cause of the observed phenomenon. For example, increases in amplitude and frequency-shift into lower frequencies has been observed in the first dorsal interosseous muscle during 80% MVC (Merletti et al.,⁸⁸ Stulen¹³¹), and it has been clearly established (De Luca et al.,³⁰ Milner-Brown et al.⁹¹) that at this contraction level, the first dorsal interosseous muscle does not recruit any motor units. In any case, the author has not been able to find one report in the literature that without doubt and ambiguity presented evidence that new motor units are recruited during constant-force contractions of some particular muscle. Arguments for motor unit recruitment during constant-force contractions, such as those presented by Maton,⁸⁷ are not convincing without proof that the force output of the muscle under investigation remains invariant. It is important to note that, in all reported experiments in which the ME signal detected from one muscle has been scrutinized for motor unit recruitment during sustained constant-force contractions, the force output of the muscle has been obtained by monitoring the torque at the joint controlled by

the muscle under investigation. Implicit or explicit assumptions have been made that invoke a linear proportionality between these two parameters. However, such is not necessarily the case, because the stiffness of agonist and antagonist muscles may vary without varying the net torque at a joint, but the increasing stiffness would increase the force output of the individual muscle being investigated. Therefore, monitoring the torque of the joint does not provide assurance that the force of an individual muscle remains invariant.

The above comment should not be misconstrued to argue against the existence of motor unit recruitment during an apparent constant-force contraction. This phenomenon may well exist, but it remains to be proven.

Synchronization, i.e., the tendency for motor units to discharge at, or nearly at, the same time has often been cited as the cause for both frequency shift and amplitude increase (Scherrer and Bourguignon,¹¹⁹ Missiuro et al.,⁹² Person and Mishin,¹⁰⁴ Lloyd,⁸² Chaffin,²⁰ Bigland-Ritchie et al.,⁹ and Palla and Ash,¹⁰² and others). However, synchronization of motor units has been reported to be more evident as the time duration of the contraction progresses (Lippold et al.,⁸⁰ Missiuro et al.,⁹² Lippold et al.⁸¹). Yet, the frequency shift is more pronounced at the beginning of a contraction (see Figure 1). Hence, the behavior of these two phenomena is not complimentary during a sustained contraction, indicating a lack of a powerful association.

Mathematical modeling of the ME signal has indicated that little, if any, frequency shift occurs as the result of motor unit synchronization (Trusgnich et al.,¹³⁷ Verroust et al.,¹⁴⁰ Blinowska et al.,¹² Jones and Lago⁵⁵). Nonetheless, mathematical modeling approaches can only provide limited insight into the modification of the power density spectrum due to synchronization of motor unit discharges because of the incomplete knowledge of the detailed behavior of motor unit discharges. In such circumstances, the indications provided by models are highly dependent on the assumptions made about the discharge statistics of the motor units and the shapes of the action potentials. The reports that have accounted for a modifying effect on the power density spectrum are consistent in indicating an increase in the low-frequency range of the spectrum, in the neighborhood of the firing rate values.

The analysis in the following section will address the effect of the discharge statistics (including firing rate and coefficient of variation of the inter-pulse intervals) on the power density spectrum. It will be seen that these properties also affect the low-frequency components. In fact, the effect of the firing rate, coefficient of variation of the inter-pulse intervals and synchronization are inextricably interwoven, and all have the potential of modifying the energy distribution of the lower frequency compartment of the power density spectrum. But, their effect and interaction is complex and not well understood. However, all current indications strongly suggest that any resulting modification of the spectrum has the potential of being inconsistent in nature. Although they are not suitable candidates for explaining the dramatic frequency-shift throughout the whole bandwidth of the ME signal, their effect on the low-frequency end of the spectrum cannot be disregarded.

The relevance of the third explanation concerning the conduction velocity of the muscle fibers will also become evident in the following section.

III. PROPERTIES OF THE POWER DENSITY SPECTRUM OF THE MYOELECTRIC SIGNAL

A systematic investigation of the behavior of the frequency shift of the ME signal requires an analysis of its power density spectrum. This task requires a mathematical model for the spectrum, which in turn requires a mathematical model for the ME signal. The latter task may be approached by considering that the ME signal, detected by electrodes placed inside the muscle or on the skin that is superficial to the muscle, consists of the temporal superposition of the motor unit action potential trains (MUAPT's) which are generated by the

individual motor units of a contracting muscle. A schematic representation of this concept along with the noise disturbances introduced during ME signal recording is presented in Figure 3.

Let us denote the MUAPT as $u(t)$; then the ME signal may be expressed as:

$$m(t) = \sum_{i=1}^p u_i(t) \quad (1)$$

where p represents the total number of MUAPTs in the ME signal. It follows that the power density spectrum of the ME signal may be expressed as:

$$S_m(\omega) = \sum_{i=1}^p S_{u_i}(\omega) + \sum_{\substack{i,j=1 \\ i \neq j}}^q S_{u_i u_j}(\omega) \quad (2)$$

where

$S_{u_i}(\omega)$ = the power density spectrum of the MUAPT, $u_i(t)$.

$S_{u_i u_j}(\omega)$ = the cross-power density spectrum of MUAPTs $u_i(t)$ and $u_j(t)$.

This spectrum will be non-zero if the firing rates of any two active motor units are correlated.

q = the number of MUAPTs with correlated discharges.

For details of this mathematical approach, refer to De Luca and Van Dyk.²⁸ De Luca et al.³¹ have shown that many (if not all) of the concurrently active motor units have, during an isometric muscle contraction, firing rates which are greatly correlated. It is not yet possible to state that all concurrently active motor units are correlated (although all of the observations of De Luca et al.³¹ support this point); therefore, q is not necessarily equal to p , which represents the total number of MUAPTs in the ME signal.

The above equation may be expanded to consider the following facts:

1. During a sustained contraction the characteristics of the MUAP shape may change as a function of time, t . For example, Broman,¹⁵ Kranz et al.,⁶⁵ and Mills⁹⁰ have all reported an increase in the time duration of the MUAP.
2. The number of MUAPTs present in the ME signal will be dependent on the force of the contraction, F .
3. The detected ME signal will be filtered by the electrode before it can be observed. This electrode filtering function will be represented by $R(\omega, d)$, where d is the distance between the pick-up points of a bipolar electrode.

Note that the recruitment of motor units as a function of time during a constant force contraction has not been considered; however, the required modification to the equation is trivial and the concept may easily be accommodated. The concept of "motor unit rotation" during a constant force contraction (i.e., newly recruited motor units replacing previously active motor units), which has at times been speculated to exist, has also not been included. No account may be found in the literature which has provided evidence of this phenomenon by definitively excluding the likelihood that the indwelling electrode has moved relative to the active muscle fibers and, in fact, records from a new motor unit territory in the muscle. For a review of these details, see De Luca.²⁹ Hence,

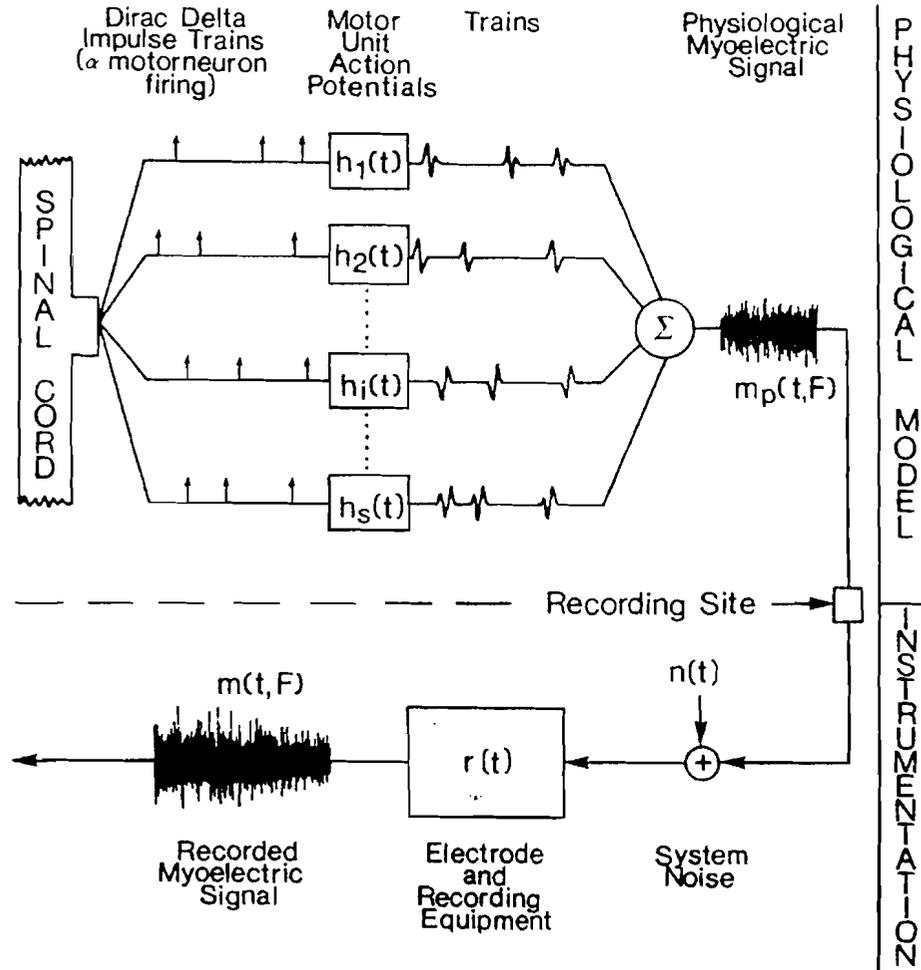


FIGURE 3. Generalized model of myoelectric signal generation and recording.

$$S_m(\omega, t, F) = R(\omega, d) \left[\sum_{i=1}^{p(F)} \dot{S}_{u_i}(\omega, t) + \sum_{\substack{i,j=1 \\ i \neq j}}^{q(F)} S_{u_i u_j}(\omega, t) \right] \quad (3)$$

The notation used in this equation may appear to be awkward in that it contains functions of time and frequency, when the frequency variable in turn represents a transformation from the time-domain. This notation is used to describe functions that have time-dependent frequency properties. From a practical point of view, such equations are useful to describe the properties of "slow" nonstationary stochastic processes.

Now, a major question arises concerning the cross-power density term. Does it really vary as a function of time during a sustained contraction so as to singly influence the power density spectrum of the ME signal? There are three eventualities that may influence its time-dependency: (1) the characteristics of the shape of the MUAP $u_i(t)$ and $u_j(t)$ change as a function of time, (2) the number of MUAPTs which are correlated varies as a function of time, (3) the degree of cross-correlation among the correlated MUAPTs varies. A change in the shape of the MUAP of $u_i(t)$ and $u_j(t)$ would not only cause an alteration in the cross-power density term, but would also cause a more pronounced modification in the respective auto-power density spectra. Hence, the power density spectrum of the ME signal would be altered regardless of the modifications of the individual cross-power density spectra of the MUAPTs. There is to date no evidence to directly support the other two points. In fact, De

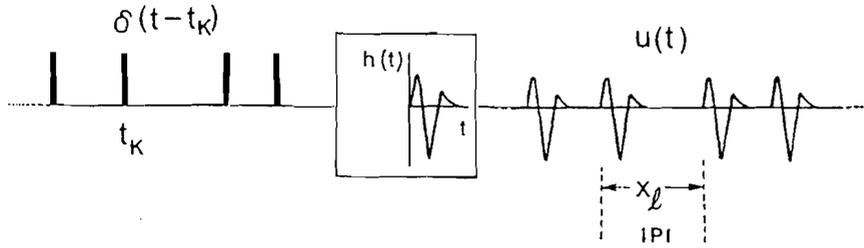


FIGURE 4. Model for a motor unit action potential train.

Luca et al.^{30,31} have presented data which indicate that the cross-correlation of the firing rates of the concurrently active motor units does not appear to depend on either time during, or force of, a contraction. This apparent lack of time-dependent cross-correlation of the firing rates is not inconsistent with previously mentioned observations indicating that the synchronization of the motor unit discharges tends to increase with contraction time. These two properties can be unrelated.

Now, let us consider a mathematical representation for the MUAPT. De Luca²⁸ reported that the MUAPT could be conveniently expressed as a series of Dirac Delta impulses (which denote the time occurrence of a motor unit firing) passed through a filter whose impulse response is the shape of an individual motor unit action potential (MUAP). The impulse response of the filter may be constructed to be time-variant in order to reflect any change during a sustained contraction. A graphic clarification of this representation is presented in Figure 4. Reports have appeared in the literature which argue in favor of the existence of minimal (if any) dependence among the interimpulse intervals (IPIs) of a particular MUAPT (Claman,²¹ Masland et al.,⁸⁵ De Luca and Forrest,²⁵ Kranz and Baumgartner,⁶⁴ and others). It could be argued that these reports do not firmly conclude that the IPIs of a MUAPT are statistically independent. In fact, it is easy to envisage specific circumstances and situations where the Ia-alpha motoneuron reflex loop may inject some dependence. However, the overwhelming majority of the available data supports an approach for modelling the IPI train as a renewal process which provides considerable mathematical and practical convenience. Therefore,

$$u_i(t) = \sum_{k=1}^n h_i(t) * \delta(t - t_k) = \sum_{k=1}^n h_i(t - t_k) \quad (4)$$

where

$$t_k = \sum_{\ell=1}^k x_\ell \quad \text{for } k, \ell = 1, 2, 3, \dots, n$$

$u_i(t)$ = the MUAPT

$h_i(t)$ = the MUAP

$\delta(t)$ = the Dirac Delta function

t = time

t_k = the time locations of the MUAPs

x = the IPI

n = the total number of MUAPs in a MUAPT

i, k, ℓ = integers which denote specific events

The power density spectrum of the MUAPT may be expressed as:

$$S_{u_i}(\omega) = S_{\delta_i}(\omega) \cdot |H_i(j\omega)|^2 \quad (5)$$

where

$S_{\delta_i}(\omega)$ = power spectrum of $\delta_i(t)$, the impulse train

$H_i(j\omega)$ = the Fourier transform of $h_i(t)$

Pursuing the above rationale, LeFever and De Luca⁷³ and, independently, Lago and Jones⁷⁰ derived the following expression for the power density spectrum of the MUAPT:

$$S_{u_i}(\omega, t, F) = \frac{\lambda_i(t, F) \cdot \{1 - |M(j\omega, t, F)|^2\}}{1 - 2 \cdot \text{Real} \{M(j\omega, t, F)\} + |M(j\omega, t, F)|^2} \{|H_i(j\omega)|^2\}$$

for $\omega \neq 0$ (6)

where

λ_i = the firing rate of the motor unit

$M(j\omega, t, F)$ = the Fourier transform of the probability distribution function $p_n(x, t, F)$ of the IPIs

More recently, Blinowska et al.¹¹ have derived similar expressions. The above expressions clearly indicate that the power density spectrum of the ME signal may be constructed from the energy spectra of the MUAPs of the motor units that contribute to the ME signal and the firing statistics of these motor units. In addition, the contribution of the firing statistics has a multiplicative effect on the energy spectrum of the MUAP. By representing $h_i(t)$ by a Fourier series, LeFever and De Luca⁷³ were able to show that the multiplicative factor is nearly constant above 40 Hz and that the IPI statistics of a MUAP only noticeably alter the shape of an individual MUAPT power density spectrum between 0 and 40 Hz.

This point may now be readily verified empirically. By using the computer assisted decomposition technique developed by LeFever and De Luca⁷³ and Mambrito and De Luca,⁸⁴ it is possible to obtain highly accurate IPI measurements of MUAPT records many seconds long. The Fourier transform of the IPIs may then be computed directly. Figure 5 presents the magnitude of such Fourier transforms for MUAPTs that were all detected during isometric constant-force contractions maintained at 50% MVC in the first dorsal interosseous muscle. The time duration of the MUAPT segment that was analyzed was 5 sec.

In the plots containing data from more than one MUAPT, the Fourier transforms were calculated separately for each MUAPT and were then averaged to obtain the displayed function. The associated histograms represent the sum of the individual histograms for each MUAPT. The data associated with the histogram represent the average of the weighted values for the individual MUAPTs. The statistics of the IPIs of each motor unit are given in Table 1. The coefficient of variation which is the ratio of the standard deviation to the mean value is a measure of the regularity with which the motor unit is discharging. The smaller the coefficient of variation, the sharper and higher will be the peak corresponding to the firing rate in the magnitude of the Fourier transform.

Figure 5A presents the data from one motor unit. The coefficient of variation of the IPIs is relatively low, and the firing rate peak (35 pulses/sec) is evident. The harmonics of the firing rate are not distinctly present. The fluctuations beyond 40 Hz are due to the random nature of the IPIs. Hence, the function remains essentially constant beyond 40 Hz.

Figure 5B presents the average of the magnitude of the Fourier transform of the IPIs of 3 MUAPTs concurrently active during the same muscle contraction. In this case where the coefficients of variation are slightly lower than that of the previous case, the peak associated with the average firing rate remains clearly distinguishable and the subsequent fluctuations of the function are less pronounced. On the other hand, Figure 5C which also presents the average of the magnitude of the Fourier transform of the IPIs of three MUAPTs concurrently active during another similar contraction from the same muscle displays a considerably different behavior. In this latter case the coefficient of variation of the MUAPTs is almost double that in Figure 5B. And, in fact, the summed histogram of the IPIs is broader, a result of the greater separation of the means of the individual histograms of these three MUAPTs.

Figure 5D presents the average of the magnitude of the Fourier transform of the IPIs of 36 MUAPTs obtained from nine separate but similar contractions, each from a different subject. In this case, the average coefficient of variation is comparable to that of the three MUAPTs in Figure 5C. The coefficients of variation of the individual MUAPTs vary considerably, rendering the relatively broader summed histogram in Figure 5D. In this latter set of data, the peaks corresponding to the firing rates are greatly depressed.

It is apparent from the examples presented in Figure 5 and Table 1 that the waveform of the modulating function $S_{\delta_i}(\omega, t, F)$ may or may not have a peak at the frequency value corresponding to the firing rate, depending on the regularity with which the motor unit discharges, i.e., the coefficient of variation of the IPIs. When more than one MUAPT is present, the presence of a peak in the Fourier transform of the IPIs will also depend on the amount of separation between the mean of the individual IPI histograms. (This latter parameter determines how the peaks and valleys in the initial part of the Fourier transform superimpose and cancel out; note the different results of Figures 5B and 5C.) In general, when many MUAPTs having a wide range of individual coefficients of variation are present, the peaks will be less pronounced and the magnitude of the Fourier transform is, for practical intents and purposes, constant above 40 Hz.

It should be noted that the two parameters that have been identified as affecting the presence of the firing rate peak are both related to synchronization. That is, the smaller the coefficient of variation and the closer the average firing rate values, the greater the probability of two or more motor units discharging during a specific time interval. It should also be immediately added that physiological events may also occur that may render the MUAPTs dependent and thereby introduce a third parameter to the concept of synchronization.

The examples of Figure 5 and Table 1 indicate that the pattern, and possibly the energy content of the power density spectrum below 40 Hz, may be altered by the statistics of the discharge properties of the motor units. Therefore, in order to explain the complete shift of the frequency spectrum, our attention should be directed at the spectral representation of the MUAPs as indicated by Equation 5.

It is important to note that modifications in the total spectral representation of the MUAPs can only result from a modification in the characteristics of the shape of the MUAP *per se*. Such modifications have their root cause in events that occur locally within the muscle. Broman¹⁵ and De Luca and Forrest²⁵ were the first to present evidence that the MUAP increases in time duration during a sustained contraction. More recently, Kranz et al.⁶⁵ and Mills⁹⁰ have provided further support.

This approach was pursued by Lindström,⁷⁴ who derived a different expression for the power density spectrum of the ME signal detected with bipolar electrodes. He approached the problem by representing the muscle fibers as cylinders in which a charge is propagated along its length. The simplified version of his results may be expressed as:

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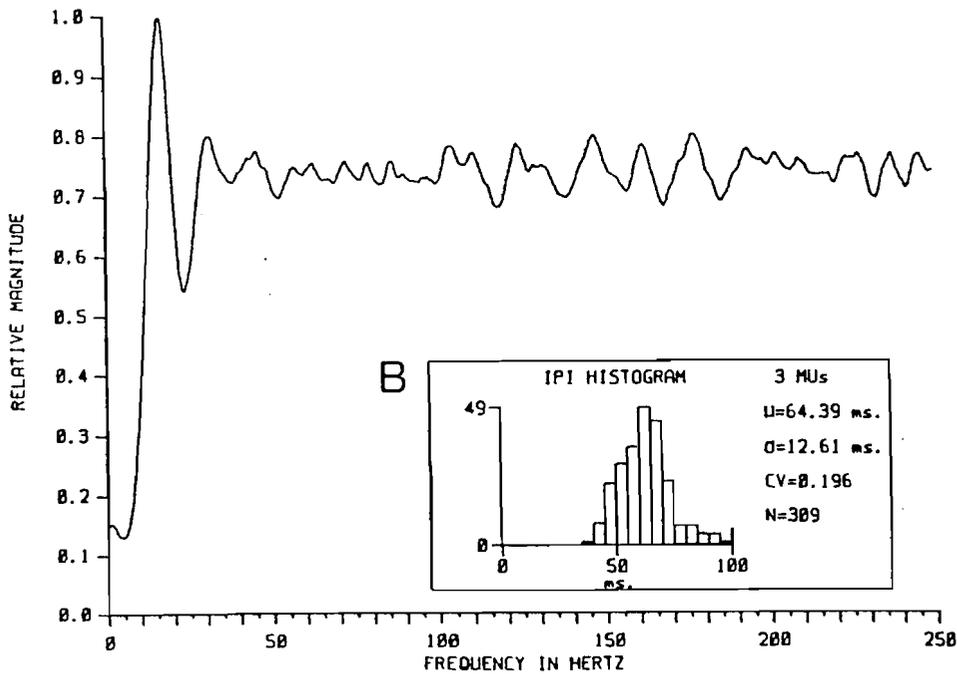
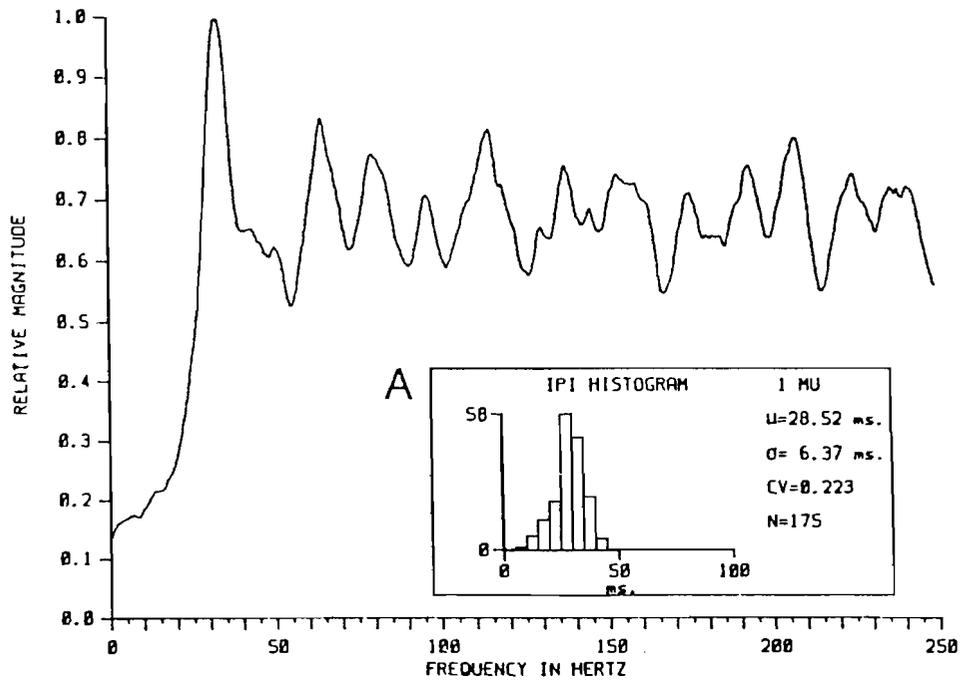


FIGURE 5. Magnitude of the Fourier transforms and histograms of the inter-pulse intervals of the motor unit action potential trains obtained from the first dorsal interosseous muscle during constant-force isometric contraction performed at 50% maximal force. The functions displayed in the following plots are expressions of the mathematical function of Equation 6: (A) one motor unit firing, the coefficient of variation of its inter-pulse intervals is 0.223; (B) three concurrently active motor units, the average coefficient of variation of their inter-pulse intervals is 0.196; (C) three concurrently active motor units, the average coefficient of variation of their inter-pulse intervals is 0.341; (D) thirty-six motor units pooled from nine separate constant-force contractions, the average coefficient of variation of the inter-pulse intervals is 0.304

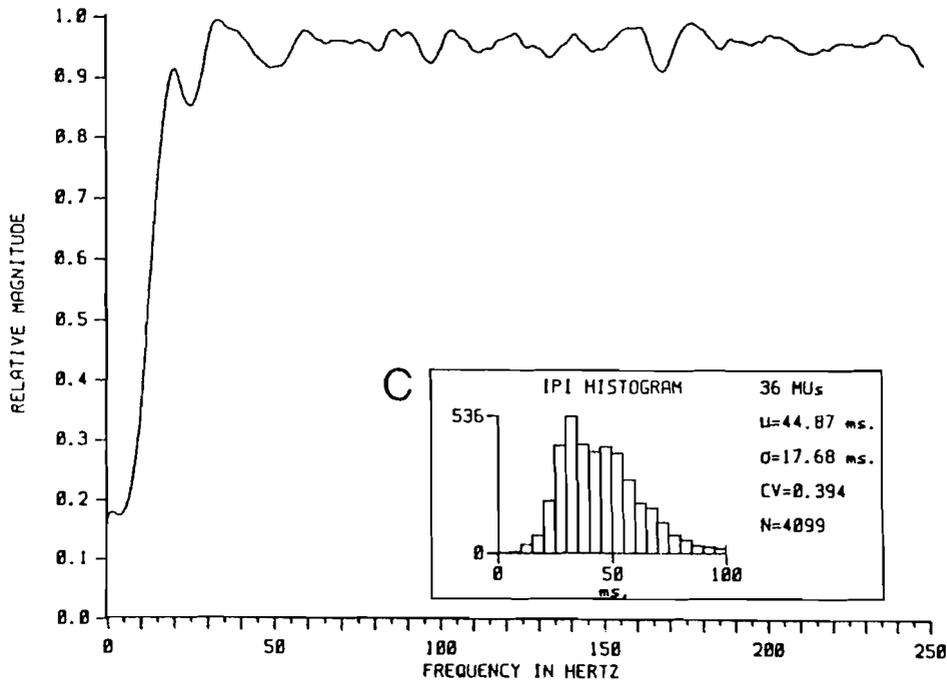


FIGURE 5 C

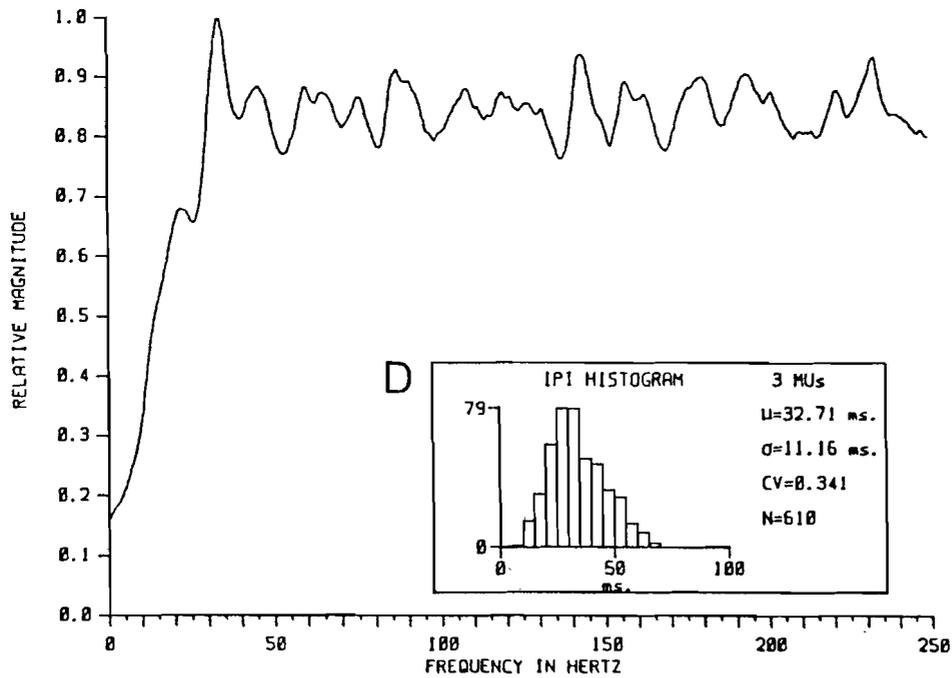


FIGURE 5 D

$$S_m(\omega) = R(\omega, d) \left[\frac{1}{v^2} G \left(\frac{\omega d}{2v} \right) \right] \quad (7)$$

where

- v = the average conduction velocity of active muscle fibers contributing to the ME signal
- G = the shape function which is implicitly dependent on many anatomical, physiological and experimental factors
- d = the distance between the pick-up contacts of the bipolar electrode

Table 1
INTER-PULSE INTERVAL
STATISTICS OF MUAPTS^a

	μ (ms)	s.d. (ms)	c.v.
Group A	28.5	6.4	0.22
Group B	58.5	9.3	0.20
	61.8	10.0	0.16
	69.3	13.7	0.16
Group C	29.7	8.3	0.28
	31.3	11.8	0.38
	43.4	11.2	0.26
Group D	28.5	5.6	0.20
	:	:	:
	42.7	18.7	0.44
	:	:	:
	87.2	10.7	0.12

^a Inter-pulse statistics of MUAPTs obtained during isometric constant-force contractions sustained at 50% of maximal force in the first dorsal interosseous muscle. This is the detailed data of the functions and histograms plotted in Figure 5. (μ = mean, s.d. = standard deviation, c.v. = coefficient of variation.)

One of the factors which is incorporated in the G function is the filtering effect of the tissue between the source (active muscle fibers) and the detecting electrode. Note that this distance depends on the location (depth) of the muscle fibers within the muscle plus the thickness of the fatty and skin tissues beneath the electrode. The form of this expression consists of modified Bessel functions of the second kind (Lindström⁷⁴). A plot of the expression for the "distance" or "tissue" filtering function is presented in Figure 6. It has the characteristics of a low-pass filter with a cutoff frequency that is inversely related to the distance D between the recording electrode and active muscle fibers.

The expression in Equation 7 may be generalized by introducing the time and force dependent effects on the ME signal; then:

$$S_m(\omega, t, F) = R(\omega, d) \left[\frac{1}{v^2(t, F)} G\left(\frac{\omega d}{2v(t, F)}\right) \right] \quad (8)$$

This representation of the power density spectrum explicitly denotes the inter-connection between the spectrum of the ME signal and the conduction velocity of the muscle fibers. Such a relationship is implicit in Equations 4 and 5, because any change in the conduction velocity would directly manifest itself in a change in the time duration of $h(t)$ as seen by the two recording contacts of a stationary bipolar electrode. If the conduction velocity were to decrease, the depolarization current would take more time to traverse the fixed distance along the fibers in the vicinity of the recording contacts and the detected MUAPs would have longer time durations. Hence, the frequency spectrum of the MUAPs and the ME signal, which they comprise, will have a relative increase in the lower frequency components and a decrease in the higher frequency components. In other words, a shift toward the low-frequency end would occur. Equation 8 also explains the amplitude increase of the ME signal as the conduction velocity decreases.

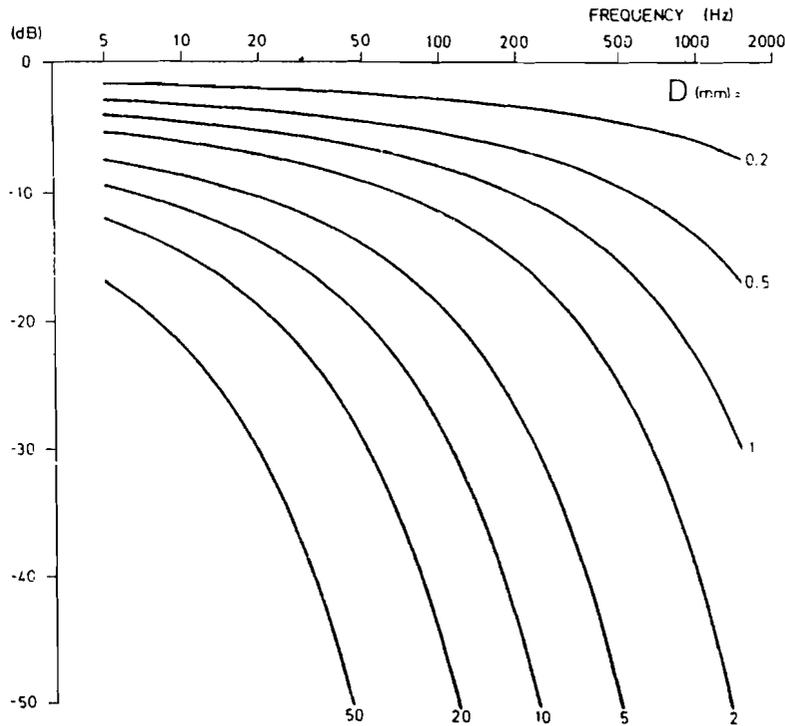


FIGURE 6. Representation of the distance filter function. The parameter D indicates the distance from an active fiber to the recording electrode. These curves were obtained by designating the conduction velocity along the muscle fiber to be 4 m/s and the diameter of the muscle fiber to be 100 μm . (From Lindström, L., Chalmers Institute of Technology, Gothenburg, Sweden, with permission.)

Lindström⁷⁴ was able to show that for a bipolar electrode located between the innervation zone and the tendon of a muscle the electrode filter function would have the following form:

$$R(\omega, d) = K \sin^2 \left(\frac{\omega d}{2v} \right) \quad (9)$$

where K is a scaling factor representing the various gain factors of the electrode and the electrode-skin interface. The representation of this function for an inter-electrode spacing, d , of 2 cm is presented in Figure 7. Note that when the relationship:

$$f_{\text{dip}} = n \frac{v}{d} \quad n = 1, 2, \dots \quad (10)$$

is satisfied, the value of the filter function $R(\omega, d)$ will be zero. This function has a multiplicative effect on the power density spectrum of the ME signal; therefore, it too will be zero at the above frequency values. Thus, if the inter-electrode spacing is known, it may be possible to calculate the average conduction velocity of the muscle fibers whose action potentials contribute to the detected ME signal.

These “dips” in the power density spectrum have been reported by several investigators (Lindström et al.,⁷⁴ Broman,¹⁵ Agarwal and Gottlieb,¹ Hogan,⁵⁰ Lindström and Magnusson,⁷⁷ Trusgnich et al.,¹³⁷ and others), but they are not always prominent. Consider the case in which the detected ME signal consists of several MUAPs whose respective MUAPs all contribute substantial energy to the power density spectrum, but whose muscle fibers have different conduction velocities. Then a filter function somewhat similar to that shown in Figure 7 would apply for every MUAP, but the location of the zero points of the function

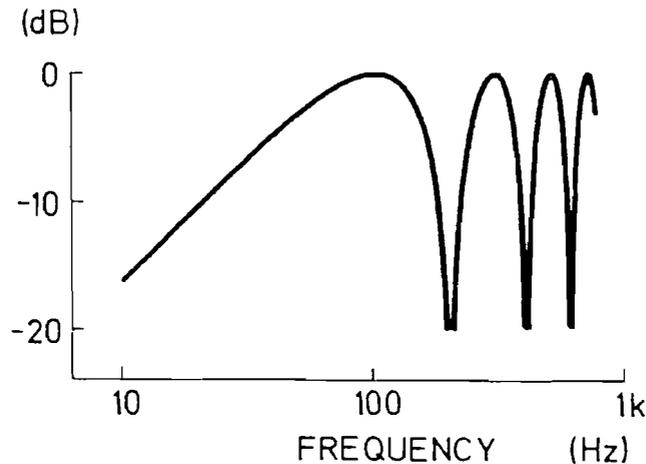


FIGURE 7. Filter function of a differential surface electrode. The separation between the two pick-up areas of the electrode was 2 cm and the conduction velocity of the muscle fiber was 4 m/s. (From Lindström, L., Chalmers Institute of Technology, Gothenburg, Sweden, with permission.)

would be different, in accordance with Equation 9. Hence, the sum of the electrode filter functions would constitute a function with no clearly demonstrable dips, and the power density spectrum would not contain a discernible dip.

The opposite is true when there is one MUAPT in the ME signal which contributes considerably more energy to the ME signal or when most of the motor units which contribute to the ME signal have nearly similar conduction velocities.

In a sense, the presence of the dips is analogous to the presence of the "peaks" at the low-frequency end of the spectrum. Both will be present when a MUAPT in the ME signal contributes considerable energy to the signal. But, it is possible for only one of these indicators to be present, depending on the firing statistics of the concurrently active motor units which contribute to the detected ME signal.

At this point, it is necessary to recollect some consistently observed properties of the motor units and of the ME signal as a function of time and force during a contraction. As the force output of the muscle increases, the number of active motor units increases and the firing rates of all the active motor units generally increase or remain nearly constant. As the time of a sustained contraction increases, the time duration of MUAPs increases, and the spectrum of the ME signal shifts towards the low-frequency end. The ramifications of these factors interpreted through the explanations provided by the mathematical models are all graphically illustrated in Figure 8.

IV. FACTORS AFFECTING THE WAVEFORM OF THE MOTOR UNIT ACTION POTENTIAL

It is now apparent that a considerable amount of the frequency shift of the power density spectrum of the ME signal is caused by a change in the spectral characteristics of the MUAPs which comprise the ME signal. Such changes may only occur if the waveform of the MUAPs change. The waveform may be altered by either varying the shape of the waveform or by scaling the waveform by linear operators. In the latter case, the shape of the waveform remains unaltered but characteristics of the shape are altered. Figure 9 presents a schematic diagram incorporating the currently known factors that directly determine or influence the waveform of the MUAPs. One factor, the tissue filtering, determines the actual MUAP shape; the other factor, conduction velocity of the muscle fibers, modifies the characteristics of the waveform.

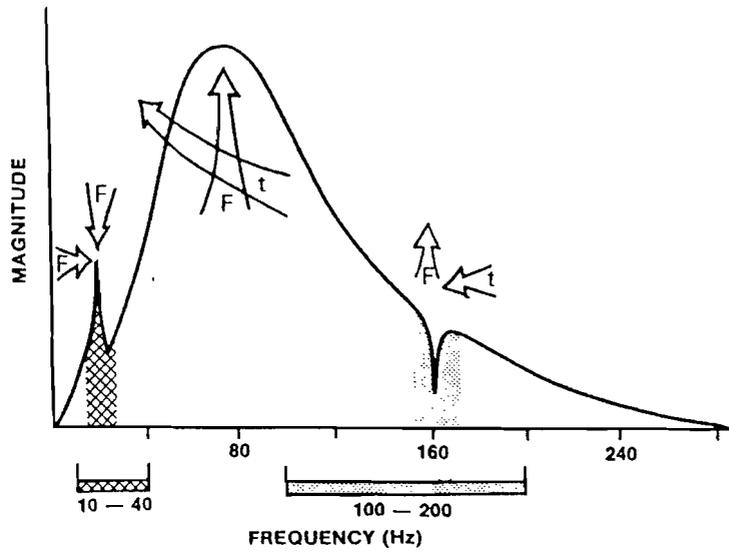


FIGURE 8. Diagrammatic representation of the frequency spectrum of the differentially detected myoelectric signal with a graphic representation of the modifications which occur as a function of time and force of contraction. The shape has been purposefully exaggerated so as to accentuate interesting segments. The direction of the arrows indicates the direction of the modification on a particular segment of the spectrum either increasing force or time. (For example, an arrow with the letter *F* indicates that as the force increases, the segment to which it is pointing will be modified in the direction of the arrow.) The peak in the low frequency components is associated with the firing rates of motor units; the dip in the high frequency components is associated with the conduction velocity along the muscle fibers. The bars on the frequency axis indicate the range over which the peaks and dips may occur.

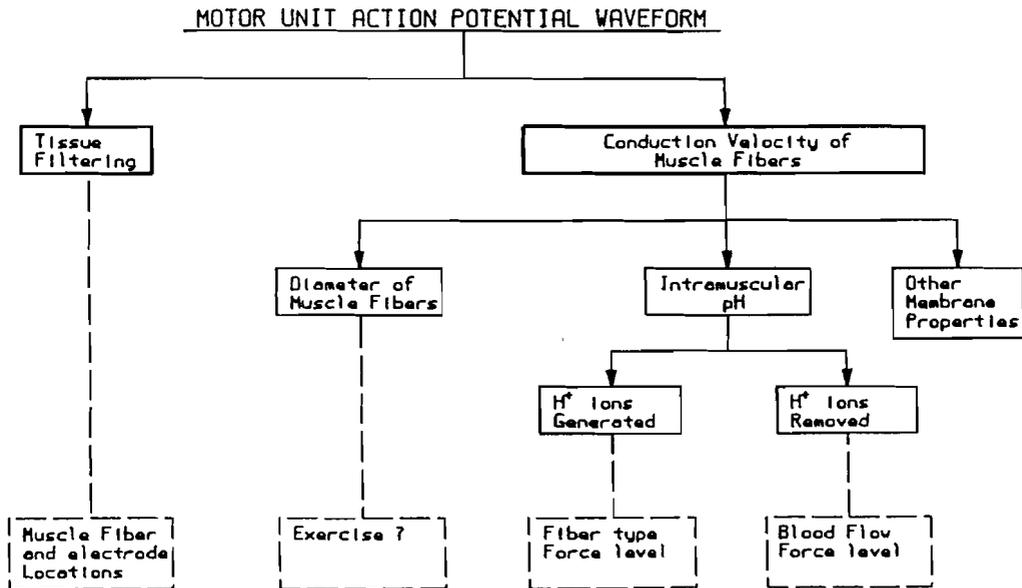


FIGURE 9. Factors affecting the waveform of the motor unit action potentials.

The amount of tissue filtering is determined mainly by three factors: the relative three-dimensional arrangement of the muscle fibers of an active motor unit, the distance between the surface electrode contacts and the active muscle fibers (see Figure 6), and the location

of the electrode on the surface of the muscle as a function of the distance between the innervation zone and the tendon of the muscle (Lindström⁷⁴). Of these two factors, the depth measure of the muscle fiber location is an important concern when additional motor units are recruited or de-recruited, such as when the force output of the muscle is varied, because their centers of electrical activity are most likely located in differing locations within the muscle. The distance between the active fiber and the electrode describes the current path between these two points, which is not necessarily the shortest distance depending on the degree of homogeneity of the tissues.

It is generally accepted that the conduction velocity of the muscle fibers is greatly affected by the diameter of the muscle fibers and the intramuscular pH. This relationship between conduction velocity and muscle fiber diameter has been known for many years and is widely accepted. Recent reports have provided conflicting results concerning modifications in the diameter of muscle fibers due to endurance exercise. For a review of this literature, the reader is referred to Salmons and Henriksson.¹¹⁷ The relationship between conduction velocity and pH is less well-documented and not as uniformly familiar.

It has also been speculated that the conduction velocity might decrease with the augmentation of potassium ions in the extracellular fluid during a contraction. The mechanisms by which the potassium ions and hydrogen ions affect the conduction velocity may possibly be closely related. The hydrogen ion mechanism has received relatively more attention; therefore, it will be discussed here. The biochemical-mechanical processes which result in a muscle fiber contraction have as a byproduct the formation of lactic acid and pyruvic acid which pass through the muscle fiber membrane into the surrounding interstitial fluid. The amount of hydrogen ions that accumulates inside and outside the muscle fiber membrane will also be dependent on the rate of hydrogen ion removal either by physical transport or by chemical reaction. The relative and absolute effects of both processes are likely to differ in muscles having considerably different fiber type constituency and tissue consistency. Generally, during repetitive muscle fiber twitches (as would be the case in sustained contractions), the hydrogen ion concentration increases and the pH decreases. Many reports verifying this point have been published. The earlier work was performed in animals (Ogata,⁹⁹ Beatty et al.⁵). In the past decade, numerous relevant investigations on human muscles *in situ* have been reported. The reader is referred to Ahlborg et al.,² Hermansen and Osnes,⁴⁹ Sahlin et al.,¹¹⁴ Tesch and Karlsson,¹³⁵ Sahlin et al.,¹¹⁵ Tesch et al.,¹³⁶ Harris et al.,⁴⁶ Viitasalo and Komi,¹⁴² among other reports. Two of these reports by Tesch^{135,136} and his colleagues present data indicating that more lactate is accumulated in muscles that consist mostly of fast-twitch fibers than those that consist mostly of slow-twitch fibers. This observation is consistent with the suggestion that higher activities of glycolytic enzymes, such as LDH and M-LDH, would favor a rapid lactate formation in fast-twitch fibers (Sjodin,¹²³ Tesch et al.¹³⁶).

It has been postulated that hydrogen ions play a significant role in the generation of action potentials in excitable membranes. They affect the process possibly by causing physical changes in the arrangement of membrane proteins and/or via their electric field generated by their charge (Bass and Moore⁴). Experimental evidence for this concept has been provided by Jennische⁵³ who demonstrated that the membrane potential decreased as the pH increased in both soleus and gastrocnemius muscles of the cat. Also Tasaki et al.¹³⁴ and Orchardson¹⁰⁰ demonstrated that the membrane excitability decreased when intracellular pH decreased. The conduction velocity is directly related to the membrane excitability. Therefore, introduction of acidic byproducts in the membrane environment may be expected to cause a decrease in the membrane conduction velocity. Such behavior in the pH and conduction velocity has been reported in a preliminary report of a study performed on the first dorsal interosseous muscle (Stulen,¹³¹ De Luca et al.³²). However, the interaction between pH and conduction velocity is not firmly established and additional research in this area would be useful.

It is the net amount of hydrogen ions in the membrane environment that is of importance. Therefore, in addition to the amount of hydrogen ions that are formed during a muscle contraction, it is also necessary to consider any mechanism that removes them from the membrane environment. This point was demonstrated by Mortimer et al.⁹³, who studied the decrease in conduction velocity in ischemic muscles of cats. In one set of experiments, a muscle was made ischemic by arterial clamping; in another set, a muscle was perfused at normal physiological pressure with nitrogen-bubbled dextran. In both cases, the muscle was not oxygenated. Yet, the decrease in conduction velocity was significantly greater under ischemic conditions than in the dextran-perfusion state. Thus, they concluded that the net accumulation of metabolic byproducts caused the decrease in conduction velocity.

The removal of acidic byproducts will be a function of many factors, the most obvious being the functional capacity of the vascularization in the muscle and the force level of the contraction. As the force output of the muscle increases from zero, the oxygen demand of the muscle increases, requiring an increase in the blood flow. However, the intramuscular pressure also increases, eventually resulting in occlusion of the arterioles and diminution of blood flow in the muscle. Mortimer et al.⁹⁴ and Bonde-Petersen et al.¹³ employed Xenon 133[®] clearance techniques to demonstrate that blood flow in the biceps and other muscles of the arm was dependent on the level of contraction. The data in both investigations showed that blood flow peaked at about 25% MVC and decreased to below resting values for contractions performed above 50% MVC. This observation has been qualified by Reis et al.,¹¹⁰ Bonde-Petersen and Robertson¹⁴ and Petrofsky et al.,¹⁰⁸ who all demonstrated that muscles consisting mainly of slow-twitch fibers are more heavily dependent on their blood supply for their ability to generate force. Belcastro and Bonen⁶ found that the rate of lactic acid removal was increased during mild self-regulated recovery exercise. It has also been found that sustained exercise will tend to decrease the amount of necessary blood flow into a muscle (Varnauskas et al.¹³⁹ and Saito et al.¹¹⁶), while apparently increasing the capillary density in the muscle (Andersen and Henriksson,³ Ingjer⁵²).

The details of the above discussion are represented in the block diagram of Figure 9. In summary, the waveform of the MUAPs detected by a surface electrode will be a function of the particular muscle that is contracting and the force level of the contraction. These two variables determine the fiber type, number, firing rate and location of the motor units that are involved, as well as the state of the blood flow. (Some evidence exists that exercise may be a contributing factor by possibly altering the fiber diameter and blood flow.) During sustained, fatiguing contractions, two factors will have the greatest effect on the motor unit action potential waveform. In constant-force contractions, where the number of active motor units is essentially fixed, the dominant factor is the amount of acidic byproducts which remain in the muscle fiber membrane environment. During force-varying contractions, the effect of tissue filtering of the newly recruited motor units also plays a prominent role.

V. PARAMETERS USED TO MEASURE THE FREQUENCY SHIFT

Several investigators have attempted to use the increase in amplitude of the ME signal as an empirical measure of localized muscle fatigue (De Vries,³³ Currier,²⁴ Lloyd,⁸² Viitasalo and Komi,¹⁴¹ Hara,⁴⁴ Petrofsky and Lind,¹⁰⁵ Hagberg,³⁹ Maton⁸⁷). These investigators have used either the rectified integrated or the rms value of the ME signal. As discussed in prior sections, the amplitude is indeed a reflection of the frequency shift (Lindström et al.,⁷⁸ De Luca²⁹). The amplitude also varies with the type of electrode used to detect the signal, the placement of the electrode, as well as the time dependent properties of the conducting gels that are commonly used to interface electrodes to the skin. These technical limitations notwithstanding, any measure of the total energy content of the ME signal cannot provide the best representation of the frequency shift. The reader is reminded that the

spectrum of the ME signal is modified by a concurrent increase in the low-frequency components and a decrease in the high-frequency components. These effects tend to offset each other so that the total power of the spectrum will have a reduced sensitivity to any frequency shift in the spectrum.

Some of the earliest attempts at identifying a single parameter of the power density spectrum for representing the frequency shift involved the calculation of the rms value of band-passed ME signals (Kadefors et al.,⁵⁶ Johansson et al.⁵⁴). However, this approach was apparently replaced by the "ratio" parameter which displayed more dramatic changes that seemed to be related to the frequency shift. The ratio parameter is the ratio of the rms of the low-frequency components to the rms of the high-frequency components (or the inverse). The separation point between the high- and low-frequency regions may be any convenient characteristic frequency, such as the mean or median of the spectrum, chosen at the initiation of the contraction. Alternatively, it has been calculated by taking the ratio of the rms values of the signal passed through two band-pass filters, one located in the low-frequency end of the spectrum, the other in the high-frequency end. The ratio parameter is convenient to monitor and has been used by several investigators (Gross et al.,³⁷ Muller et al.,⁹⁵ Bellemare and Grassino,^{7,8} Schweitzer et al.,¹²¹ and Bigland-Ritchie et al.⁹).

The main attraction of the ratio parameter is that it presents dramatic changes in value of the frequency shift. However, Stulen and De Luca¹³² have shown through mathematical calculation that the ratio parameter has several drawbacks which make it inferior to other spectral parameters. This point has been demonstrated empirically by Schweitzer et al.¹²¹ and Hary et al.⁴⁵ The ratio parameter is sensitive to the shape of the ME signal spectrum. This is a hindrance, because in some muscles the frequency components of the ME signal may vary during force-varying contractions, due to the recruitment of motor units that have significantly different action potential shapes. Furthermore, this parameter is dependent on the initial value of the characteristic frequency chosen to divide the spectrum or on the center frequency and bandwidths of the band-pass filters chosen. For the case where the median frequency is used as the partitioning frequency, a statistical analysis has shown that the ratio parameter estimate is biased and has a covariance approximately 30% greater than the covariance of the estimate of the median frequency. Also, the ratio parameter is not linearly related to the conduction velocity of the muscle fibers (Stulen and De Luca¹³²). In fact, this nonlinear relationship explains the misinterpretation of Bigland-Ritchie et al.⁹ who inappropriately reached the correct conclusion that the decrease in the conduction velocity does not provide sufficient cause for the change in the frequency spectrum of the ME signal. This interpretation was subsequently remedied by an elegant study from the same laboratory (Bellemare and Grassino⁸).

Other parameters have also been proposed, but have not been widely used. De Luca and Berenberg²⁷ proposed a polar representation consisting of a plot of the low-frequency rms value vs. the high-frequency rms value. In such a representation, the plot remains on a line of slope 1 when no frequency shift is present and curves continuously towards the low-frequency rms axis as the shift progresses, thereby describing a characteristic pattern. Sadoyama and Miyano,¹¹³ noting that the frequency shift appeared to be a quasi-exponential function of time during a sustained contraction, derived an expression which they termed "measure H". This expression consisted of the integral of the spectral function multiplied by the log of the spectral function. This parameter has the advantage of providing a more linear representation of the frequency shift as a function of time. Hagg,⁴¹ Inbar et al.,⁵¹ and Masuda et al.⁸⁶ have proposed yet another parameter, the number of zero-crossings of the ME signal. This parameter has the advantage of being relatively simple to implement in hardware. However, the number of zero-crossings may be severely affected by noise, a deficit whose impact may be reduced by introducing a non-zero threshold to the signal. A more important disadvantage of this approach is introduced by the fact that the number of

zero-crossings is approximately linearly dependent on the force of the contraction during relatively low efforts (Lindström and Petersén⁷⁹).

Since, according to Equation 7, all the frequencies are scaled by the same factor, a frequency shift may be observed by tracking any characteristic frequency. Three have been used by various investigators. They are the median-frequency, the mean-frequency, and the mode-frequency. The median-frequency is the frequency at which the power density spectrum is divided into two regions with equal power; the mean-frequency is the average frequency; and the mode-frequency is the frequency of the peak of the spectrum. All three are (in a mathematical sense) linearly related to the conduction velocity of the muscle fibers (Stulen and De Luca¹³²).

The mean-frequency has been used by Herberts et al.,⁴⁸ Lindström et al.,⁷⁴ Lindström and Magnusson,⁷⁷ Broman and Kadefors,¹⁷ Hagberg,³⁸ Komi and Tesch,⁶² Lynne-Davies et al.,⁸³ Ortengren et al.,¹⁰¹ Hagberg,⁴⁰ Hagberg and Ericson,⁴¹ and Ladd et al.⁶⁹ The median-frequency has been used by Stulen and De Luca,^{128,129} Sabbahi et al.,¹¹² Petrofsky,¹⁰⁷ Petrofsky and Lind,¹⁰⁵ Inbar et al.,⁵¹ Palla and Ash,¹⁰² Stulen and De Luca,¹³³ De Luca et al.,³² Merletti et al.,⁸⁸ van Boxtel et al.,¹³⁸ Sadoyama et al.,¹¹³ and Kranz et al.⁶⁵

Of these characteristic frequencies, the mode-frequency is the least useful. Although superficially it might appear to be a useful parameter, because even for relatively poor signal-to-noise ratios it is always theoretically possible to obtain the best estimate. However, this is not the case, because the ME signal is a stochastic signal which does not have a smooth and sharply defined region near the peak value of its spectrum; hence, the variance of the spectrum would strongly influence the estimation accuracy of the mode. This point has been confirmed empirically by Schweitzer et al.¹²¹ They found that the coefficient of variation for the estimate of the mode-frequency was five times greater than that of the mean-frequency for ME signal obtained from the human diaphragm.

Recently, Stulen and De Luca¹³² have shown that the median-frequency provides a reliable, consistent and relatively unbiased estimate of a parameter of the spectrum that is related to the muscle fiber conduction velocity. In general, the estimate of both the median and mean frequencies provides an acceptably good representation of the frequency shift. Both are superior to other parameters (Stulen and De Luca,¹³² Hary et al.⁴⁵). However, both have relative advantages and disadvantages, depending on the quality of the ME signal, the shape of the spectrum and other related factors. These two frequency parameters offer the additional advantage that the calculation of their estimate may be implemented in analog circuitry, allowing them to be obtained on-line and in real-time (Broman and Kadefors,¹⁷ and Stulen and De Luca^{128,133}).

VI. BEHAVIOR OF CHARACTERISTIC FREQUENCIES

It is now apparent that according to available information, the median- and mean-frequencies are the preferred characteristic frequencies for monitoring the frequency shift. The median-frequency (Stulen and De Luca¹³²) and the mean-frequency (Lindström⁷⁴) have both been mathematically demonstrated to be linearly related to the average conduction velocity of the muscle fibers. Sodayama et al.^{113a} have provided experimental verification for a linear relationship.

Both these characteristic frequencies have been shown to decrease as a function of time during a sustained contraction (Figure 1). During sustained constant-force contractions, the rate of decrease has been found to be either quasi-linear (Petrofsky,¹⁰⁷ Petrofsky and Lind,^{105,106} Inbar et al., Mills⁵¹) or quasi-exponential (Lindström et al.,⁷⁷ Stulen,¹³¹ Hagberg,³⁹ Stulen and De Luca,¹³³ De Luca et al.³²). This apparent discrepancy may easily be attributed to either different muscles that may have been used to perform the measurements or to different processing schemes employed to calculate the characteristic frequencies. It should be noted

that an exponential response may appear to be quasi-linear if the time constant of the processing scheme or device is long relative to the time constant of the event being monitored.

The mean- or median-frequencies may decrease by more than 50% in value from the beginning to the end of a sustained isometric constant-force contraction. However, the amount of decrease appears to be dependent on the muscle being investigated; some reveal much less dramatic decreases than others. Stulen¹³¹ found that in the first dorsal interosseous muscle and the deltoid muscle the greatest decrease in the value of the median-frequency occurred at 50% MVC. This observation is consistent with that of Clamann and Broeker²² who found that the amplitude of the ME signal demonstrated the greatest increase during sustained 50% MVC contractions and with that of Tesch and Karlsson¹³⁵ who found that maximal lactate concentrations were found in muscles which contracted isometrically at 50% MVC to exhaustion.

After termination of a sustained contraction, the median- and mean-frequencies monitored in a muscle have been observed to recover (increase towards their initial value) within 4 to 5 min (Sabbahi et al.,¹¹² Stulen,¹³¹ Petrofsky and Lind,¹⁰⁵ Mills,⁹⁰ Merletti et al.⁸⁸). This behavior is consistent with that of the conduction velocity observed by Broman.¹⁵ These observations are consistent with the time required to remove lactic acid after the cessation of exercise that induces localized muscle fatigue (Harris et al.⁴⁶). Other reports such as that of Sahlin et al.¹¹⁵ described much longer lactate removal times after exercises which caused a systemic exhaustion, such as exercising on an ergometer until total exhaustion. The distinction between these two types of tasks is important. In the latter case, most of the skeletal muscles in the body would be involved. Hence, a relatively high concentration of lactate would be present in the systemic bloodstream, whereas in the former case, only a localized group of muscles would be primarily involved in the prescribed exercise; less lactate would be present in the bloodstream; and the time required for absorption would be less. The latter case is more indicative of localized muscular fatigue and may more accurately be associated with the characteristic frequency measurements made from one muscle.

The absolute values of the mean- and median-frequencies have been found to vary inconsistently as a function of force within any one muscle (Petrofsky and Lind,^{105,106} Stulen,¹³¹ Palla and Ash¹⁰²). In another study, Hagberg and Ericson⁴¹ found that the mean-frequency increased with force at relatively low levels of contraction; at levels exceeding 25% MVC the value of the mean-frequency became independent of force output. These results are consistent with the explanation presented in a previous section. That is, the relative position of the newly recruited motor units with respect to the recording electrode and the diameter of the muscle fibers of the newly recruited motor units determine the modification of the ME signal frequency spectrum caused by the additional MUAPs present in the signal.

Changes in median- and the mean-frequencies have also been shown to be affected by blood occlusion within the muscle. Recent studies by Hara,⁴⁴ Mills,⁹⁰ and Merletti et al.⁸⁸ have all indicated that the frequency shift of the ME signal is more pronounced when the blood in the contracting muscle is occluded by external compression. These results are consistent with the fact that when the blood is occluded, acidic byproducts accumulate in the environment of the muscle fiber membrane and decrease the conduction velocity of the muscle fibers. The median-frequency has also been shown to be affected by the muscle temperature. Petrofsky and Lind¹⁰⁵ found it to increase as the muscle temperature was increased from 10° to 40°C. Merletti et al.⁸⁸ found that it decreased linearly with decreasing muscle temperature during cooling. This latter observation is consistent with the known fact that the conduction velocity of the muscle fibers is proportionately related to temperature.

All the reported observations of the mean- and median-frequencies are consistent with the series of events displayed in the block diagram form in Figure 9. The only published work which argues against this thesis, with the weight of properly interpreted data, is that of Naeije and Zorn.⁹⁷ These investigators simultaneously measured the mean-frequency of

the ME signal and the average conduction velocity of the muscle fibers contributing to the ME signal from the biceps brachii of eight subjects. In four of the subjects, the decrease in the mean-frequency and conduction velocity were linearly correlated, and in the rest the mean-frequency decrease was not accompanied by a decrease in the conduction velocity. This apparent dichotomy might be resolved by considering the technique used to measure the conduction velocity. They used a cross-correlation technique to measure the difference in the time of arrival of the ME signal at two locations along the direction of the muscle fibers away from the innervation zone. This technique is not consistently reliable, especially when the cross-correlation value is less than 0.7 (personal observation). A possible explanation may be provided by the anisotropy of the muscle, fatty and skin tissues. The impedance of the path between a muscle fiber source and a location on the surface of the skin may vary with the location. Therefore, the arrival time of the signal to any point on the skin will be a function of the path taken by the signal to reach that point as well as the conduction velocity of the muscle fibers. Therefore, measurements of this kind require caution in their interpretation. In fact, similar experiments also involving the biceps brachii muscle reported by Sodoyama et al.^{113a} show no conflict between the behavior of the characteristic frequencies and the conduction velocity. In this latter report, considerable care was taken to resolve signal analysis conflicts.

VII. APPLICATIONS OF QUANTITATIVE MEASURES OF LOCALIZED MUSCULAR FATIGUE

The technique of monitoring the frequency shift of the ME signal for the purpose of measuring localized muscular fatigue has several advantages: it is noninvasive; it may be performed on muscle *in situ*; it may be performed in real-time; and it provides information relating to events which occur inside the muscle. In addition to this, Lindström and Petersén⁷⁹ have recently shown that the decrease of the mean-frequency is directly related to increased subjective sensation of perceived exertion during a sustained muscle contraction performed at moderate levels. Many applications of this approach for measuring muscle fatigue are envisioned; some are only concepts and still require experimental verification, whereas others have been already put into practice. The use of the ME signal frequency shift, however, is far from routine. Some of these applications are

Athletic training — The effects of athletic training and exercise on muscle fiber metabolism and architecture is currently an issue of considerable discussion. Numerous studies have been reported with a variety of conflicting results. For a review of these details, the reader is referred to an article by Salmons and Henriksson.¹¹⁷ As discussed in previous sections, these modifications within the muscle are theoretically accompanied by corresponding, observable changes in the frequency spectrum of the ME signal.

Industrial applications — The use of the frequency shift as an indicator of localized muscular fatigue has been applied in the field of Ergonomics (Broman et al.¹⁵, Kadefors et al.,⁵⁶ Petersén et al.¹⁰³). Herberts et al.⁴⁷ and Hagberg^{38,39} have studied the effect of elevated arm positions on localized muscular fatigue in the shoulder muscles, which is experienced by workers in a variety of work environments. Each study concluded that the exchange in the ME signal power density spectrum is useful for measuring the progression of fatigue as a function of arm position. In fact, Herberts et al.⁴⁷ suggest a preferred position to minimize the "sensation" of fatigue.

The technique may conceivably be used to distinguish between psychological fatigue derived from boredom and physiological fatigue derived from sustained effort in a work station. It is conceivable that this technique may prove useful in designing work stations in which individuals may comfortably and productively interact with their tasks.

Physical therapy — In rehabilitation programs involving muscle re-education and exercise, it is often necessary to assess the effectiveness of a prescribed physical therapy program. Manual muscle tests are currently the primary procedure for determining muscular strength and the progression or regression of strength. Yet, these tests are subjective and their accuracy depends on the training, skill, and experience of the clinician performing the examination (Kendall et al.⁵⁸). In a relatively recent report, Edwards and Hyde³⁵ stated that there are no quantitative methods for measuring muscle function in clinical use today for the diagnosis and management of patients complaining of weakness.

During a physical therapy session, it might be possible to assess the response of the impaired muscle(s) to treatment by measuring the frequency shift. If a characteristic frequency obtained from the impaired muscle decreases, then the muscle is indeed being exercised and is undergoing a fatigue process. If, on the other hand, the characteristic frequency does not change, it may indicate that the muscle is not being adequately exercised and/or the unaffected synergists are generating most of the force. When a muscle or a group of muscles is weakened, there is a tendency for subtle shifts in the pattern of muscle activity to occur to enable the synergistic muscles to generate the required force. This is known as "muscle substitution", and it denies the impaired muscle the intended exercise. Muscle substitution is difficult to detect by current manual testing, which depends greatly on the experience of the clinician. With the frequency shift technique, muscle substitution might be observed by noting a modification in the behavior of a characteristic frequency. For example, if a characteristic frequency obtained from a muscle decreases and then abruptly levels off or begins to increase without a decrease in force output, it may indicate that other muscles are now generating most of the force, allowing the impaired muscle to relax in a relative sense. This information alone would make the frequency shift technique a useful aid for the physical therapist.

The effectiveness of a prescribed treatment program could be determined by changes in the behavior of the characteristic frequency obtained during a series of treatments. Possibly, both the time constant of decay and/or the percentage decrease in the characteristic frequency obtained from a sustained contraction should increase with the number of trial sessions. If the muscle is severely atrophied at the start of the therapy, then the initial value of the characteristic frequency may also change significantly over several of the initial test sessions.

Diagnosis and prognosis of neuromuscular disorders — The effectiveness of the use of the ME signal in the assessment of neuropathic and myopathic disorders has often been investigated. The level of these investigations has included: single fiber electromyography promoted by Stålberg and his co-workers (Stålberg et al.¹²⁵ and Schwartz et al.¹²⁰), so-called quantitative electromyography (Buchthal et al.¹⁹) based on the temporal characteristics of the MUAPs and other factors observable with signals obtained with needle electrodes, macro electromyography based on information obtained by a needle electrode with considerably large pick-up area (Stålberg¹²⁶), and also frequency analysis of the ME signal (Larsson,⁷¹ Kopec and Hausman-Petrusewicz⁶³).

Larsson⁷¹ studied neuropathies induced by lesions of the peripheral motoneurons. His results suggest that the spectrum of the ME signal is shifted into lower frequencies in neuropathies with a clinical history of at least 6 months. Since a characteristic frequency is sensitive to the "average" shape of the MUAPs, changes in the characteristic frequency may be useful in following the development of the disorder. The opposite effect on the shape of the MUAPs is characteristic of myopathies. That is, they are generally shorter in duration than normal, and are more often polyphasic (Kugelberg⁶⁶). Each of these factors leads to a shift of the spectrum into higher than normal frequencies, which has been confirmed by Kopec and Hausman-Petrusewicz⁶³.

For a measure of the characteristic frequency to be useful in the diagnosis or prognosis of either type of disorder, the frequency shift must be measurably greater than that which

is normally expected due to the stochastic nature of the signal. However, if the disease is known to be present or is suspected, then the change in the characteristic frequency obtained from subsequent examinations may be useful in monitoring the progression or regression of the disorder.

Other clinical applications — One interesting application of the ME signal frequency shift has been in the assessment of diaphragm fatigue. In this particular case, contractile fatigue is inconvenient to measure and, moreover, provides a rather late assessment of the functional capability of the diaphragm. Researchers in Canada (Bellemare and Grassino,⁷ Gross et al.³⁷ and Solomon et al.¹²⁴) as well as researchers in the U.S. (Lynne-Davies et al.,⁸³ Schweitzer et al.¹²¹) have investigated this possible application. In particular Solomon et al. have indeed shown that metabolic fatigue, as indicated by the frequency shift of the ME signal, occurs before contractile fatigue. Hence, they proposed to use a measure of this shift to set the resistance to breathing for safely exercising the diaphragm in quadriplegic patients.

Ladd et al.⁶⁹ suggested that a measure of the mean-frequency could be used to monitor the progression of peripheral nerve regeneration. This possibility is based on the concept that different muscle fiber populations and/or different motor unit architectures would emerge throughout the regenerative process. Such a concept has merit. However, serious consideration should be given to the accompanying modifications of the vascular network in the re-innervating muscle, because it may influence the quantity of acidic byproducts which are retained in the muscle.

One area that has not yet been explored for possible consideration, but which from a logical perspective holds promise, involves peripheral vascular diseases. The diminished capacity of the vascular network to remove acidic byproducts from contracting muscles in limbs decreases the pH within the muscles (O'Donnell⁹⁸). Hence, the characteristic frequency would reflect this decrease by a corresponding decrease in its value.

Basic research — It is self-evident that the use of the frequency shift in this area of investigation holds a myriad of possibilities and applications. The process(es) of localized muscular fatigue are numerous and apparently complex, requiring a host of techniques for its study and analysis. The frequency shift is a prime candidate, because of its noninvasive nature and its direct and indirect relationship(s) to physiological, anatomical, and biochemical events and modifications within the muscle. To date, very little work of this nature has been reported in the literature. Preliminary reports by Sabbahi et al.¹¹² and Merletti et al.⁸⁸ provide some data indicating that the median-frequency of the ME signal is affected by a decrease in temperature and ischemic conditions in the muscle. Preliminary reports by Rosenthal et al.¹¹¹ and Hakkinen and Komi⁴³ have indicated that there might be a relationship between the fiber type composition of a muscle and the value of the median- and mean-frequencies. If this initial observation is substantiated, the technique could provide a noninvasive alternative to muscle biopsy.

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