Muscle Fatigue and Time-Dependent Parameters of the Surface EMG Signal

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 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 one 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 central nervous system could reveal time-dependent changes indicative of a fatigue process, even though the externally observable mechanical performance would not be altered until the failure point. An example of this is presented in Figure 8.1, which demonstrates the force output of the first dorsal interosseous muscle during an attempt at maintaining a 50% maximal voluntary contraction (MVC) constant, along with the median frequency of the power density spectrum of the EMG signal from the same muscle. (It will be explained later that the median frequency, as well as other characteristic frequencies of the power density
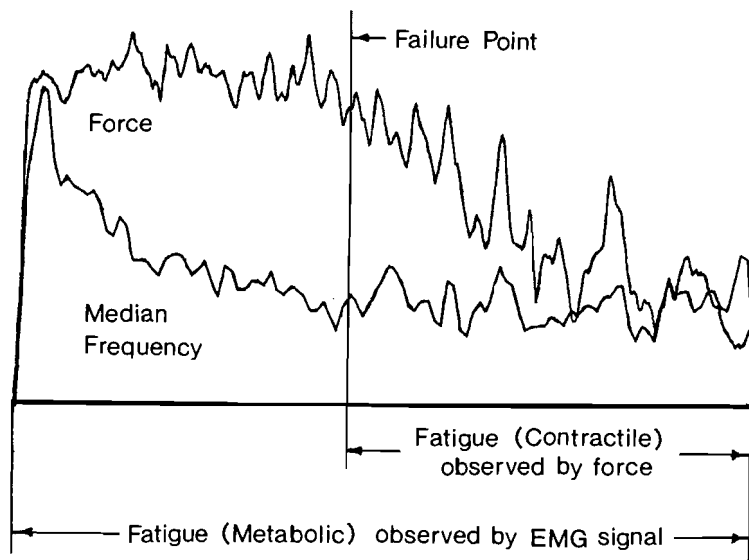
LOCALIZED MUSCULAR FATIGUE

Figure 8.1. 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 EMG signal. The time duration of the contraction was 150 s.

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 EMG signal detected on the surface of the skin and 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.

BACKGROUND

Over one-half century ago, Muscio (1921) 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 professionals to subdivide the concept of fatigue into subsets. This approach was exemplified by Bills (1943), 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, 1976).

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 (1973). Although this term originally had its roots in the field of ergonomics, it was subsequently popularized by a research group at Chalmers University of Technology and Sahlgren Hospital in Sweden. However, according to Merton (1954) 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 EMG signal, detected on the surface of the skin over a muscle, has been extensively employed. Since the historic work of Piper (1912), the frequency components of the surface EMG signal have been known to decrease when a contraction is sustained. Cobb and Forbes (1923) noted this shift in frequencies toward the low end with fatigue and also observed a consist-

ent increase in amplitude of the EMG signal recorded with surface electrodes. Many other investigators have also noted an increase in EMG signal amplitude (Knowlton et al, 1951; Scherrer and Bourguignon, 1959; Zhukov and Zakharyants, 1960; Lippold et al, 1960; deVries, 1968; Kadefors et al, 1968; Kuroda et al, 1970; Lloyd, 1971; Vreden-bregt and Rau, 1973; Stephens and Usherwood, 1975; Viitasalo and Komi, 1977; Stulen and De Luca, 1978a; Clamann and Broecker, 1979; Maton, 1981; Hagberg, 1981; 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, 1962a and b; Sato, 1965; Kadefors et al, 1968; Kwatney et al, 1970; Lindström et al, 1970; Johansson et al, 1970; Chaffin, 1973; Lindström et al, 1974; Viitasalo and Komi, 1977; Lindström et al, 1977; Givens and Teeple, 1978; Komi and Tesch, 1979; Petrofsky and Lind, 1980b; Bigland-Ritchie et al, 1981; Hagberg, 1981; Inbar et al, 1981; Kranz et al, 1981; Palla and Ash, 1981; Stulen and De Luca, 1981; Mills, 1982; Hagberg and Ericson, 1982; De Luca et al, 1983; and others). These two phenomena, which are pictorially represented in Figure 8.2, are in fact related. Lindström et al (1970) and De Luca (1979) explained the interrelationship by noting that during a sustained contraction the low-frequency components of the EMG signal increase and, hence, more EMG 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 EMG 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 EMG 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 EMG 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 EMG 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
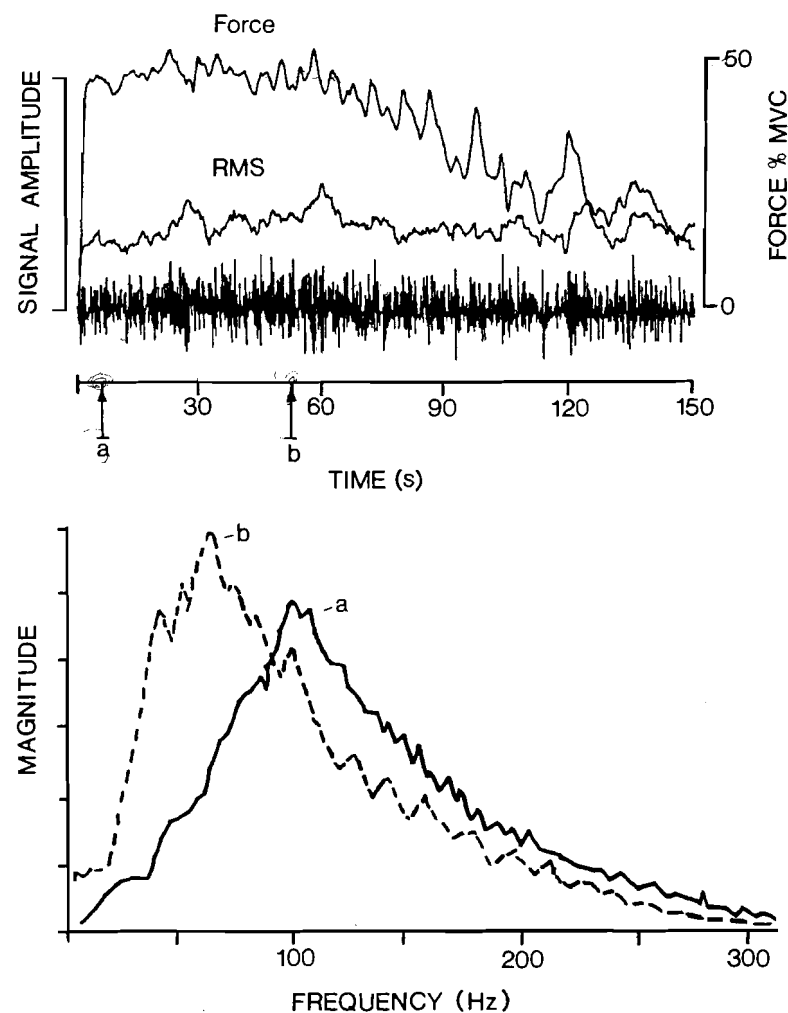
Figure 8.2. (*Top*) EMG signal amplitude and force during an attempted constant-force contraction in the first dorsal interosseous muscle. (*Bottom*) Power density spectra of the EMG signal at the beginning and at the end of the constant-force segment of the contraction.

list should be added the regularity (coefficient of variation) of the motor unit discharge.

Edwards and Lippold (1956), Eason (1960), Vredenbregt and Rau (1973), and Maton (1981) 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, currently

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 have been observed in the first dorsal interosseous muscle during 80% MVC (Stulen, 1980; Merletti et al, 1984), and it has been clearly established (Milner-Brown et al, 1973; De Luca et al, 1982a) that at this contraction level, the first dorsal interosseous muscle does not recruit any motor units. In any case, we have 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 (1981), 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 EMG 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 by increasing the force output of the individual muscle being investigated. Therefore, monitoring the torque of the joint does not provide ensurance 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 of both frequency shift and amplitude increase (Scherrer and Bourguignon, 1959; Missiuro et al, 1962a and b; Person and Mishin, 1964; Lloyd, 1971; Chaffin, 1973; Bigland-Ritchie et al, 1981; and Palla and Ash, 1981; 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, 1957; Missiuro et al, 1962a; Lippold et al, 1960). Nevertheless, the freqeuncy shift is more pronounced at the beginning of a contraction (see Fig. 8.1). 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 EMG signal has indicated that little, if any frequency shift occurs as the result of motor unit synchronization (Trusgnich et al, 1979; Verroust et al, 1981; Blinowska et al, 1980, Jones

and Lago, 1982). Nonetheless, mathematical modeling approaches can only provide limited insight into the modification of the power density spectrum caused by the synchronization of motor unit discharges because of the incomplete knowledge of the detailed behavior of the 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 range of the firing rate values.

The review presented in the following section will address the effect of the discharge statistics (including firing rate and coefficient of variation of the interpulse 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 interpulse 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 are complex and are 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 EMG 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.

FACTORS AFFECTING THE FREQUENCY SHIFT OF THE EMG SIGNAL

A systematic investigation of the behavior of the frequency shift of the EMG 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 EMG signal. Such an analysis has been developed in Chapter 3. The equation describing the power density spectrum is repeated here for convenience.

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

where

$t = \text{time}$

$F = \text{force}$

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

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

$R(\omega, d)$ = the electrode filtering function

p = the total number of MUAPTs that comprise the signal

q = the number of MUAPTs with correlated discharges

d = the distance between the detection surfaces of the bipolar electrode

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

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

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

where

λ_i = the firing rate of the motor unit

$S_{\delta_i}(\omega)$ = The power density function of the impulse train $\delta_i(t)$, which represents the time events of the MUAPs.

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

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

These equations emphasize the description of the power density spectrum in terms of the statistical properties of motor unit discharges. Although the waveforms of the MUAPs are represented in $H_i(j\omega)$, any factors which may modify the waveform during a sustained contraction are not represented. One such factor is the conduction velocity of the EMG signal along the muscle fibers. This relationship is expressed in the following alternative representation:

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

where

v = the average conduction velocity of active muscle fibers contributing to the EMG signal

G = the shape function which is implicitly dependent on many anatomical, physiological, and experimental factors.

From the above equations and with reference to the related discussion in Chapter 3, it is possible to draw the following observations concerning the behavior of the spectrum:

1. A decrease in the firing rates of the motor units will contribute to a shift of the power density spectrum towards frequencies. This will occur because the probability distribution function (or histogram) will become increasingly skewed. A pictorial explanation of this point is presented in Figure 3.7.

The energy contribution discharge characteristics in the power density spectrum is limited to below 40 Hz.

2. A modification in the discharge characteristics of the motor units may also effect the power density spectrum because the cross-correlation terms will be effected. Factors such as synchronization and regularity (coefficient of variation) of motor unit discharge play a prominent role in determining the behavior of the cross-correlation values. It has been shown (in Chapter 3) that these discharge characteristics have a tendency to increase the energy in the low-frequency part of the spectrum. Their effort is also limited to below 40 Hz.

3. Any modification in the waveform of the MUAPs will be reflected in the Fourier transforms of the waveforms and thus in the power density spectrum. The waveform would have a larger time duration as the conduction velocity decreases during a sustained contraction because the time to traverse the electrode environment would be longer. This would cause a simultaneous increase in the low-frequency components and a decrease in the high-frequency components. Evidence for the increase in the time duration of the MUAP has been provided by Broman (1973), De Luca and Forrest (1973a), Broman (1977), Kranz et al (1981), and Mills (1982).

The first two observations concern control properties of the motor units, which may be either central and/or peripheral in origin. Their involvement in the spectral shift is realistic because it is known that during a sustained contraction, the firing rate of motor units decreases, and other statistical parameters may be altered. (Refer to the discussion in Chapter 5 for details.) The third observation is associated with biochemical and physiological events which occur in the muscle tissue per se. Their involvement in the spectral shift is also realistic and will be discussed in the following section.

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 EMG signal is caused by a change in the spectral characteristics of the MUAPs which comprise the signal. Such changes may only occur if the waveform of the MUAPs changes. 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. This distinction between the meaning of waveform and shape is important in subsequent discussions. Note that a linear multiplication in either the time scale or amplitude scale does not change the shape. Modifications in the shape are induced by nonlinear transformations. Figure 8.3 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.
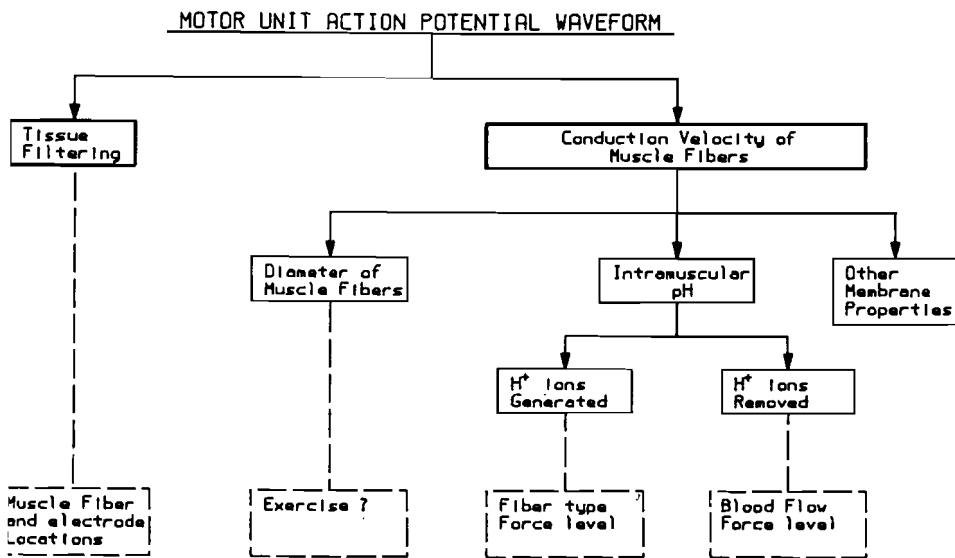
MOTOR UNIT ACTION POTENTIAL WAVEFORM

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                    ┌────────────────────────────┐
                    │ Conduction Velocity of      │
                    │     Muscle Fibers           │
                    └────────────────────────────┘
    ┌──────────┐        ┌──────────┐  ┌──────────────┐  ┌────────────┐
    │ Tissue   │        │ Diameter │  │ Intramuscular│  │ Other      │
    │ Filtering│        │ of       │  │    pH        │  │ Membrane   │
    └──────────┘        │ Muscle   │  └──────────────┘  │ Properties │
                        │ Fibers   │                    └────────────┘
                        └──────────┘
                              ┌──────────┐   ┌──────────┐
                              │ H⁺ Ions  │   │ H⁺ Ions  │
                              │ Generated│   │ Removed  │
                              └──────────┘   └──────────┘
  ┌───────────┐  ┌──────────┐  ┌──────────┐  ┌──────────┐
  │ Muscle    │  │ Exercise?│  │ Fiber    │  │ Blood    │
  │ Fiber     │  │          │  │ type     │  │ Flow     │
  │ and       │  │          │  │ Force    │  │ Force    │
  │ electrode │  │          │  │ level    │  │ level    │
  │ Locations │  │          │  │          │  │          │
  └───────────┘  └──────────┘  └──────────┘  └──────────┘
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Figure 8.3.   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 Fig. 2.16); 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 (Lindström, 1970). Of these two factors, the depth measure of the muscle fiber location is an important concern when additional motor units are recruited or decruited (such as when the force output of the muscle is varied) because their centers of electrical activity are most likely situated 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.

The conduction velocity of the muscle fibers is monotonically related to the diameter of the muscle fibers and is greatly affected by the intramuscular pH. The 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 (1981).) The relationship between conduction velocity and pH is less well documented and not as uniformly familiar.

The biochemical-mechanical processes which result in a muscle fiber

contraction have as a by-product 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, 1960; Beatty et al, 1963). In the past decade, numerous relevant investigations on human muscles in situ have been reported. The reader is referred to the reports of Ahlborg et al, 1972; Hermansen and Osnes, 1972; Sahlin et al, 1975; Tesch and Karlsson, 1977; Sahlin et al, 1978; Tesch et al, 1978; Harris et al, 1981; and Viitasalo and Komi, 1981, among others. Two of these reports by Tesch 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, 1976; Tesch et al, 1978).

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 the electric field generated by their charge (Bass and Moore, 1973). Experimental evidence for this concept has been provided by Jennische (1982), who demonstrated that the membrane potential decreased as the pH increased in both soleus and gastrocnemius muscles of the cat. Also Tasaki et al (1967) and Orchardson (1978) demonstrated that the membrane excitability decreased when intracellular pH decreased. The conduction velocity is directly related to the membrane excitability. Therefore, introduction of acidic by-products 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, 1980; De Luca et al, 1983). However, the interaction between pH and conduction and 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 (1970), 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 by-products caused the decrease in conduction velocity.

The removal of acidic by-products 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 (1971) and Bonde-Petersén et al (1975) 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 (1967), Bonde-Petersén and Robertson (1981), and Petrofsky et al (1981), all of whom 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 Bonen (1975) 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, 1970; Saito et al, 1980), while apparently increasing the capillary density in the muscle (Andersen and Henriksson, 1977; Ingjer, 1979).

The details of the above discussion are represented in the block diagram of Figure 8.3. 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, in which the number of active motor units is essentially fixed, the dominant factor is the amount of acidic by-products 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.

PARAMETERS USED TO MEASURE THE FREQUENCY SHIFT

A cautionary note is in order on the technical details associated with the detection and recording techniques used to acquire the EMG signal. (For additional and specific details, refer to the material in Chapter 2.) Special attention should be given to the combined bandwidth of the electrode, amplifier, and recording device in order to acquire a faithful representation of the EMG signal. Ideally, for surface electrodes, the bandwidth (3 dB points) should be 0–500 Hz. However, the DC coupling may cause complications. Therefore, for practical purposes it is recommended that the low-3 dB point be set at 20 Hz. The reader is reminded that the estimate of parameters of the frequency spectrum of the EMG signal discussed in this section will be affected by the recording bandwidth. Wherever possible, in the discussion of this and following sections, it will be assumed that a sufficiently wide bandwidth has been used to acquire the signal.

Several investigators have attempted to use the increase in amplitude of the EMG signal as an empirical measure of localized muscle fatigue (deVries, 1968; Currier, 1969; Lloyd, 1971; Viitasalo and Komi, 1977; Hara, 1980; Petrofsky and Lind, 1980a; Hagberg, 1981; Maton, 1981). These investigators have used either the rectified integrated or the rms value of the EMG signal. Although, as discussed in prior sections, the amplitude is indeed a reflection of the frequency shift (Lindström et al, 1977; De Luca, 1979). Furthermore, the amplitude 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. Regardless of these technical limitations, any measure of the total energy content of the EMG signal cannot provide the best representation of the frequency shift. The reader is reminded that the spectrum of the EMG 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 EMG signals (Kadefors et al, 1968; Johansson et al, 1970). 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. Alternately, 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, 1979; Muller et al, 1978; Bellemare and Grassino, 1979 and 1982; Schweitzer et al, 1979; Bigland-Ritchie et al, 1981).

The main attraction of the ratio parameter is that it presents dramatic changes in value of the frequency shift. However, Stulen and De Luca (1981) have shown through mathematical calculation that the ratio parameter has several drawbacks which make it inferior to other special parameters. This point has been demonstrated empirically by Schweitzer et al (1979) and Hary et al (1982). The ratio parameter is sensitive to the shape of the EMG signal spectrum. This is a hindrance, because in some muscles the frequency components of the EMG 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 Luca, 1981). In fact, this nonlinear relationship explains the misinterpretation of Bigland-Ritchie et al (1981), who inappropriately arrived at the correct conclusion that the decrease in the conduction velocity does not provide sufficient cause for the change in the frequency spectrum of the EMG signal. This interpretation was subsequently remedied by an elegant study from the same laboratory (Bellemare and Grassino, 1982).

Other parameters have also been proposed but have not been widely used. De Luca and Berenberg (1975) proposed a polar representation consisting of a plot of the low-frequency rms value versus 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 (1981), noting that the frequency shift appeared to be a quasiexponential 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 (1981), Inbar et al (1981), and Masuda et al (1982) have proposed yet another parameter, the number of zero crossings of the EMG 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 nonzero 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 Petersén, 1981).

Since, according to the equation relating the conduction velocity to the power density spectra, 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 Luca, 1981).

The mean frequency has been used by Herberts et al (1969), Lindström et al (1977), Lindström and Magnusson (1977), Broman and Kadefors (1979), Hagberg (1979), Komi and Tesch (1979), Lynne-Davies et al (1979), Ortengren et al (1979), Hagberg (1981), Hagberg and Ericson (1982), and Ladd et al (1982). The median frequency has been used by Stulen and De Luca (1978b, 1979), Sabbahi et al (1979), Petrofsky (1980), Petrofsky and Lind (1980a), Inbar et al (1981), Palla and Ash (1981), Stulen and De Luca (1982), De Luca et al (1983), van Boxtel et al (1983), Sadoyama et al (1983), Kranz et al (1983), and Merletti et al (1984).

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 EMG 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 (1979). 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 EMG signal obtained from the human diaphragm.

Recently, Stulen and De Luca (1981) 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, 1981; Hary et al, 1982). However, both have relative advantages and disadvantages, depending on the quality of the EMG 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, 1979; Stulen and De Luca, 1978b, 1982).

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 Luca, 1981) and the mean frequency (Lindström, 1970) have both been mathematically demonstrated to be linearly related to the average conduction velocity of the muscle fibers. Sadoyama et al (1983) have provided experimental verification for a linear relationship. Kranz et al (1983) have suggested that the change in conduction velocity may, in fact, account for nearly all the spectral shift seen in the signal.

Both these characteristic frequencies have been shown to decrease as a function of time during a sustained contraction (Fig. 8.1). During sustained constant-force contractions, the rate of decrease has been found to be either quasilinear (Petrofsky, 1980; Petrofsky and Lind 1980a and b; Inbar et al, 1981; Mills, 1982) or quasiexponential (Lindström et al, 1977; Stulen, 1980; Hagberg, 1981; Stulen and De Luca, 1982; De Luca et al, 1983). 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 quasilinear if the time constant of the processing scheme or device is long relative to the time constant of the event being monitored. The rate of decrease is a function of the contraction force; the higher the force, the greater the rate (see Fig. 8.4).

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
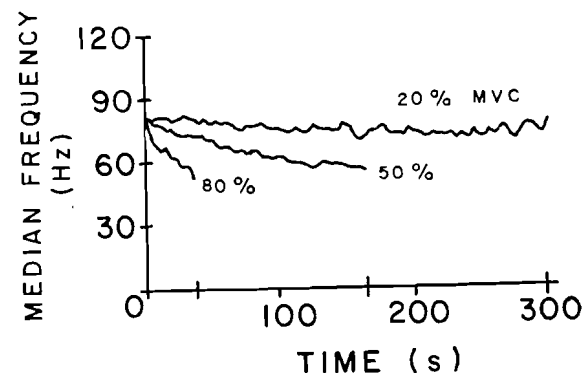
Figure 8.4. The median frequency as a function of time for contractions performed at 20, 50, and 80% of the maximal voluntary contraction level in the first dorsal interosseous muscle.

dependent on the muscles being investigated; some reveal much less dramatic decreases. Stulen (1980) 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 Broecker (1979), who found that the amplitude of the EMG signal demonstrated the greatest increase during sustained 50% MVC contractions and with that of Tesch and Karlsson (1977), 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 4–5 minutes (Sabbahi et al, 1979; Stulen, 1980; Petrofsky and Lind, 1980a; Mills, 1982; Merletti et al, 1984). This behavior is consistent with that of the conduction velocity observed by Broman (1973), and with the time required to remove lactic acid after cessation of exercise that induces localized muscle fatigue (Harris et al, 1981). Other reports, such as that of Sahlin et al (1978), described much longer lactate removal times after exercises which cause 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 blood stream. Whereas in the latter case, only a localized group of muscles would be primarily involved in the prescribed exercise, less lactate would be present in the blood stream, 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 value of the mean and median frequencies has been found to vary inconsistently as a function of force within any one muscle (Petrofsky and Lind, 1980a and b; Stulen, 1980; Palla and Ash, 1981). In another study, Hagberg and Ericson (1982) 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 EMG signal frequency spectrum caused by the additional MUAPTs present in the signal.

Changes in median and mean frequencies have also been shown to be affected by blood occlusion within the muscle. Recent studies by Hara (1980), Mills (1982), as well as our own work (Merletti et al, 1984) have all indicated that the frequency shift of the EMG 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 by-products 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 Lind (1980a) found it to increase as the muscle temperature was increased from 10 to 40°C. Merletti et al (1984) found it to decrease 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 8.3. The only published work which argues against this thesis, with the weight of properly interpreted data, is that of Naeije and Zorn (1982). These investigators simultaneously measured the mean frequency of the EMG signal and the average conduction velocity of the muscle fibers contributing to the EMG 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 EMG 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. 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. Sadoyama et al (1983) repeated essentially the same experiment, using the same muscle and the same cross-correlation technique. They were careful to resolve signal analysis conflicts, and their data showed a consistent simultaneous decrease in both the mean frequency and conduction velocity.

APPLICATIONS OF QUANTITATIVE MEASURES OF LOCALIZED MUSCULAR FATIGUE

The technique of monitoring the frequency shift of the EMG 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 that, Lindström and Petersén (1981) 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, although it is possible that the perceived sensation is not causally related to events occurring in the muscle per se. Many applications of this approach for measuring muscle fatigue are envisioned; some are only concepts and still require experimental verification, whereas others have already been put into practice. The use of the EMG signal frequency shift, however, is far from routine. Some of these applications are seen below.

Athletic Training. The effects of athletic training and exercise on muscle fiber metabolism and architecture are 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 (1981). As discussed in previous sections, these modifications within the muscle are theoretically accompanied by corresponding observable changes in the frequency spectrum of the EMG 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, 1973; Kadefors et al, 1976; Petersén et al, 1976). Herberts et al (1979) and Hagberg (1979, 1981a) 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 change in the EMG signal power density spectrum is useful for measuring the progression of fatigue as a function of arm position. In fact, Herberts et al (1979) 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 reeducation 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, 1971). In a relatively recent report, Edwards and Hyde (1977) 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 EMG 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 coworkers (Stålberg et al, 1975; Schwartz et al, 1976); so-called quantitative electromyography (Buchthal et al, 1957) based on the temporal characteristics of the MUAPs and other factors observable with signals obtained with needle electrodes; macroelectromyography based on information obtained by a needle electrode with considerably large pickup area (Stålberg, 1980); and also frequency analysis of the EMG signal (Larsson, 1975; Kopec and Hausman-Petrusewicz, 1966).

Larsson (1975) studied neuropathies induced by lesions of the peripheral motoneurons. His results suggest that the spectrum of the EMG 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 (Kugelberg, 1947). Each of these factors leads to a shift of the spectrum into higher than normal frequencies, which has been confirmed by Kopec and Hausman-Petrusewicz (1966).

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 EMG 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, 1979; Gross et al, 1979; and Solomon et al, 1979), as well as researchers in the USA (Lynne-Davies et al, 1979; Schweitzer et al, 1979) have investigated this possible application. In particular, Solomon et al (1979) have indeed shown that metabolic fatigue, as indicated by the frequency shift of the EMG 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 (1982) 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 reinnervating muscle, because it may influence the quantity of acidic by-products 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 by-products from contracting muscles in limbs decreases the pH within the muscles (O'Donnell, 1975); 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 (1979) and Merletti et al (1984) provide some data indicating that the median frequency of the EMG signal is affected by a decrease in temperature and ischemic conditions in the muscle. Preliminary reports by Rosenthal et al (1981) and Hakkinen and Komi (1983) 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.