

# **Neuromuscular Fatigue**

Edited by A.J. Sargeant and D. Kernell

*Abstract:* Among Health Scientists, it has become customary for muscle fatigue to be described and evaluated in terms of the force that can be produced by a muscle. In the past decade this approach has been expanded to the assessment of voluntary contractions. Of particular concern is the use of the failure point of a muscle to produce a desired force as the event in time when the muscle becomes fatigued. This paper points out the limited usefulness and possible faulty interpretation of this approach. It is suggested that muscle fatigue is more correctly (and usefully) viewed as a continual function of contraction time. A technique that accomplishes this need is described and is referred to as the EMG spectral compression technique. This technique is based on the well-known fact that the frequency spectrum of the EMG signal is continuously compressed during a sustained contraction. The median frequency of the EMG signal is recommended as the preferred variable for tracking spectral compression. It is shown that the median frequency is affected mostly by the pH in the muscle, which depends on the amount of net Lactate that is produced and removed, as well as some other unknown factor(s). It is argued that this objective and non-invasive, non-painful technique provides superior means for assessing and monitoring muscle fatigue in humans performing voluntary contractions. It also provides convenient means for studying some biochemical modifications within the muscle without invading the muscle.

### **Muscle performance**

Muscles are physiological force actuators. Thus, among those trained in the Health Sciences it has been teleologically reasonable to evaluate the performance of muscles by the behavior of the force that they generate. Therefore, it is not surprising that early attempts at measuring the deterioration in the capability to maintain a desired performance level (muscle fatigue) would be based on a decrease in the force output of the muscle.

Pursuing this line of thought, Burke *et al.* (1971) described a fatigue test for classifying the performance of muscle fibers which could be stimulated directly via an electrical pulse train (40 Hz; 1/3 s on, 2/3 s off) applied to the nerve innervating the muscle. The performance of the muscle fiber was assessed by observing the amplitude of the force twitch. When the amplitude began to decrease, fatigue was considered to begin. This approach made reasonable use of the force variable as an index of muscle fatigue, but introduced a notion of fatigue that was related to an indication of a deterministic failure in the behavior of the monitored variable. This approach has been widely accepted and as a consequence, the notion of fatigue has become synonymous with the concept of failure as used in engineering and physics, two disciplines that have dealt with the concept of fatigue for considerable time. Following the lead of Burke, Edwards (1981) extended this concept of fatigue to voluntary contractions in the human by describing fatigue as the inability to sustain a voluntary contraction at a predetermined force level. More recently, Bigland-Ritchie (1984) has advanced the concept of monitoring the rate of decline of force output during a sustained maximal voluntary contraction. Although this approach improves on the use of force as the variable of measure, it remains susceptible to the capability and/or willingness of the individual to continue to elicit maximal effort during a test. Such tests are uncomfortable and questionably objective.

The notion of *equating fatigue to a failure point* carries with it some practical disadvantages. For example, fatigue would be detectable only after it has occurred. This approach would have little use in clinical and ergonomics applications where it is often desirable to have indications that precede failure to produce the desired force so that appropriate remedies can be made or evaluations can be

taken. Conceptually, the notion of the failure point also relates to the occurrence of a catastrophic event. During a sustained contraction, a complex system which has numerous individual processes simultaneously operating to achieve an end goal is at work. Consider for example the metabolite usage, the Lactate accumulation (with its corresponding pH changes), modifications in the Calcium ion release mechanism, Potassium ion losses, Creatine Phosphate usage, decrease in the firing rates, motor force-twitch potentiation and other mechanisms. All of these processes undergo continual modification as a function of time during a contraction. Each process fatigues at its own rate and most, if not all, affect the ability of the muscle to produce force. Thus, failure to produce force may occur either by a catastrophic event in a dominant process or as a combination of the fatigue characteristics of the individual processes.

The use of force during a voluntary contraction as an index of muscle fatigue introduces at least three additional confounding factors. Firstly, in voluntary contractions the force output of an individual muscle is not often directly accessible. Without surgical intervention, one can only measure the net torque at a joint. That is, the difference between the torques produced by the agonist muscles and the torque produced by the antagonist muscles. Thus, the monitored torque may not faithfully represent the torque (or force) of the muscle of interest, depending on the behavior of the antagonist muscles. This issue is particularly troublesome in the presence of pain or injury where muscles may alter their load sharing during a task. Secondly, during a submaximal contraction, it is possible to maintain the torque (or force) output acceptably constant in a macroscopic sense, but there are time-dependent physiological and biochemical processes that microscopically alter the means for generating force during a sustained contraction. Some of these are: 1) some motor units might become derecruited, 2) the force twitches of motor units potentiate, i. e., the integral of their force increases, and 3) the firing rates of most active motor units decrease. This later phenomenon was first reported by Person and Kudina (1972) and independently by De Luca and Forrest (1973). Later, De Luca (1979) proposed " ... if the firing rate of the motor unit decreases and there is no significant recruitment, a complimentary mechanism must occur to maintain the constant-force output. One possible mechanism is the potentiation of force twitch tension of the motor units as a contraction progresses." An additional caveat was added by De Luca et al. (1982a) "...that this behavior is likely to be due either to post-tetanic twitch potentiation or to a concurrent reduction in the force output of agonist and antagonist muscles which maintains the monitored force output of a joint constant". Now this concept has been adopted into the motor control literature. Thirdly, during a voluntary contraction the failure point is a function of both physiological and psychological factors, and it is difficult to know accurately the causal relationship of each to the failure point.

The psychological factor could be removed by activating the muscle via electrical stimulation instead of a voluntary effort. However, a muscle contraction induced by electrical activation generates force by decidedly different means. For a review of electrical stimulation of muscles see Merletti, Knaflitz and De Luca (1992). By contemporary methodologies, the electrical stimulation is delivered as a train of pulses at a fixed frequency, usually 30-40 Hz, whereas in actuality the firing rates of the motor units are quasi-random. Also, the recruitment order of the motor units is decidedly different than in the natural activation. Although the order is not completely reversed, whether the motor point of the muscle is stimulated (Knaflitz, Merletti and De Luca: 1990) or the nerve is stimulated (Gorman & Mortimer, 1983, among others). But most importantly during electrical stimulation, the higher threshold motor units, which decrease their force output more quickly than others, are stimulated at a much higher rate than occurs during natural activation. During a voluntary contraction, the higher threshold motor units fire at lower rates than earlier recruited, lower threshold motor units ( De Luca et al., 1982b). *The central nervous system appears to be designed to compromise between activating the muscle to generate the maximal force possible and sustaining a contraction for a finite amount of time.*

If not force, then what?

The other obvious variable that has been commonly used to assess the performance of muscles is the surface electromyographic (EMG) signal. This approach presents several advantages: 1) The EMG

signal can be detected from a specific muscle designated for study, unlike the force variable which cannot be easily isolated for an individual muscle. However, if it is desired to study a group of muscles, multiple channel detection techniques may be used to obtain the required EMG signals simultaneously. 2) The EMG signal provides information that directly and objectively relates to the state of the muscle studied; it reflects the factors that are directly related to the anatomy or architecture, the physiology and the biochemistry of the muscle. 3) It circumvents the subjectivity of the voluntary-force variable monitored during a voluntary contraction. 4) Detection techniques are not painful and are not invasive, rendering them useful for clinical and ergonomics purposes.

Early attempts at using the EMG signal as an index of muscle fatigue relied on the amplitude of the signal, with inconsistent success. It has been known for over four decades (Knowlton *et al.*, 1951) that the amplitude of the EMG signal generally increases during sustained sub-maximal constant-force contractions. This phenomenon is due to the increased time duration of the motor unit action potentials (MUAP) during a sustained contraction. It will be seen below that this behavior is more reliably and clearly displayed in the behavior of the frequency spectrum of the EMG signal. Also, amplitude measurements of the EMG signal are prone to be affected by various sources of noise and impedance matching complications. The electrical noise can originate from the ambient surroundings, improper grounding of the electronic equipment, low-quality amplifiers, electrically-unstable connection to the skin by the electrodes caused by ineffective mechanical coupling of the electrode to the skin, movement of the electrode with respect to the skin causing motion artifacts, and other more esoteric sources.

An alternative and more reliable approach is to study the behavior of the EMG signal in the frequency domain. This is known as the EMG spectral technique. Modifications in the EMG signal during a sustained contraction which are difficult to quantify in the time domain can become considerably easier to quantify in the frequency domain. However, this technique is not without its own set of complications; they will be discussed in a section near the end of the paper.

### **The EMG spectral variable technique**

It is well known that during a sustained contraction the surface EMG signal undergoes a translation (slowing) in the time domain. This phenomenon can be seen in the top right-hand quadrant of Figure 1, where examples of the EMG signal detected at the beginning and end of an isometric constant-force contraction are shown. If the Fourier transforms of the two signals are taken, the slowing of the EMG signal is seen as a compression in the frequency domain. That is, the amplitude of the low-frequency components increases and that of the high-frequency components decreases. (To be precise, the spectrum also undergoes a mild change in shape that is not explainable by a compression in the frequency domain. See Brody *et al.* (1991) and Merletti, Knaflitz and De Luca (1992).) A convenient means of tracking the frequency compression is to monitor the median frequency of the spectrum. For an overview see Figure 1 and for details see a review article by De Luca (1985).

The cause of the frequency compression provides reason for using the spectral technique as an index of muscle fatigue. Through mathematical modeling and experimentation we have established that the control properties of the motor units ( firing rate and synchronization) do not affect the frequency spectrum appreciably (De Luca 1985). The now well known decrease in the firing rates of the motor unit during constant force contractions cause only a minor shift in the frequency spectrum below 40 Hz.

Synchronization among the discharges of concurrently active motor units has been, at times, used to explain the modification in the frequency spectrum. Recent work at our Center has indicated that only approximately 4-5 % of motor unit discharges are synchronized, and that these discharges occur in bursts of mostly one or two consecutive firings at sporadic intervals throughout the contraction. These observations strongly suggest that synchronization cannot be an influential factor in modifying the frequency spectrum.

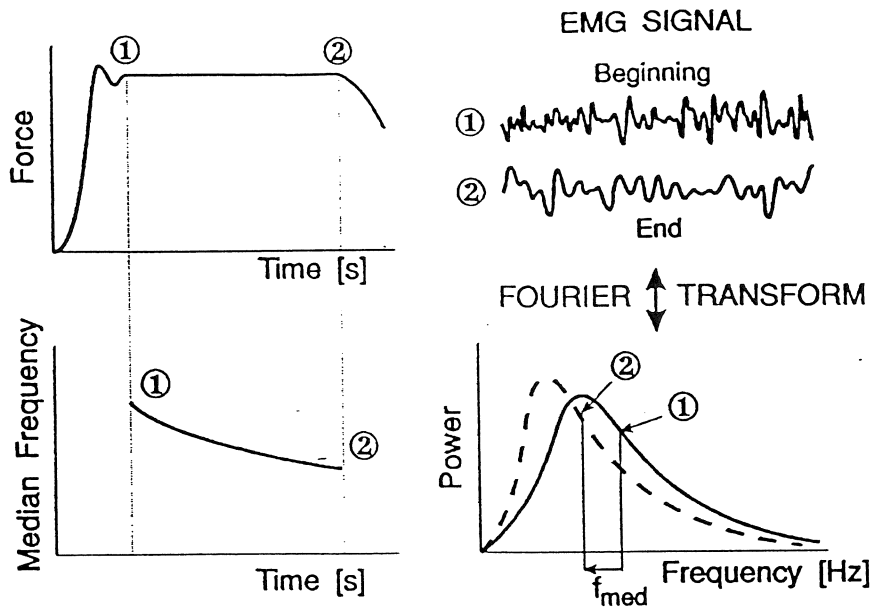


Figure 1. The EMG signal at the beginning (1) and the end (2) of a constant-force isometric contraction (top left quadrant), along with the corresponding frequency spectral compression and the time course of the median frequency during the sustained contraction. This is a schematic representation consistent with the behavior of the real data.

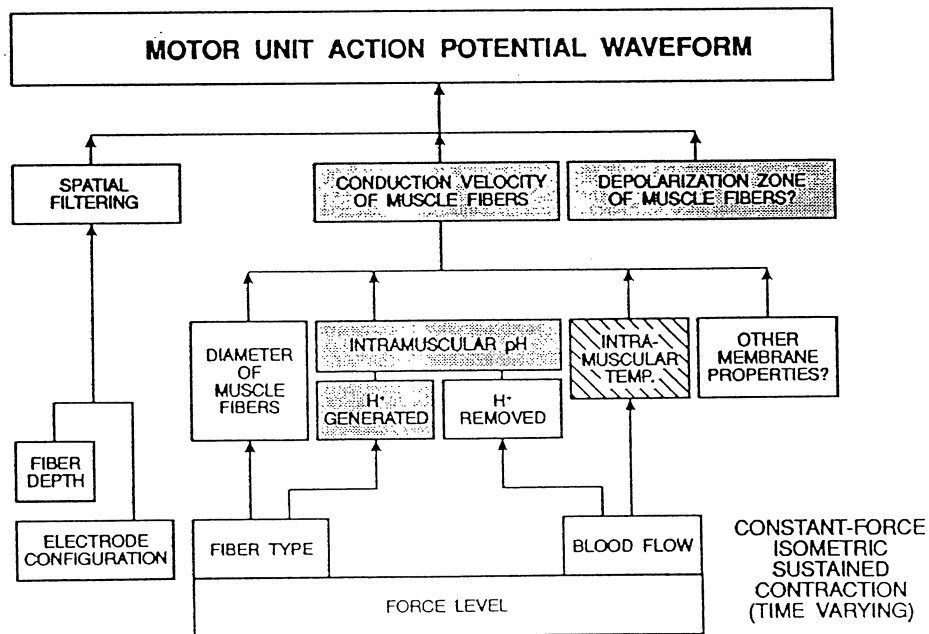


Figure 2. Factors which effect the shape of the motor unit action potential during a sustained contraction. The shaded boxes indicate factors that modify as function of time during a sustained constant-force isometric contraction above 30 % MVC. Temperature is less relevant than the other highlighted factors. Other indicated factors are relevant only during weaker contractions or non-isometric contractions.

The explanation for the frequency compression can be found in the behavior of the MUAP shape during a contraction. This is the likely source because we know from mathematical modeling that the

shape of the frequency spectrum of the EMG signal is almost exclusively due to the shape of the constituent MUAPs. For the past decade my colleagues (notably Serge Roy and Roberto Merletti) have been attempting to understand which factors dominate the behavior of MUAP shape. Our current understanding is summarized in Figure 2, which presents the known influences on the MUAP shape, and highlights those that are subject to modification during a sustained constant-force contraction. These influences progressively affect the MUAP shape during the accumulation of fatigue. The two dominant factors are the conduction velocity (CV) of the muscle fibers and some other, yet to be identified factor. We suspect the length of the depolarization zone to be that other factor (Merletti, Knaflitz, De Luca; 1992), but Stegeman (in this book) suspects otherwise.

The modeling work of Lindstrom *et al.* (1970) as well as our own modeling work (Stulen & De Luca, 1981) has shown that the CV of the muscle fibers directly affects the bandwidth of the frequency spectrum and inversely (by a square-root factor) the amplitude of the EMG signal. In the past decade there have been several reports from different laboratories which agree that the CV of muscles fibers decreases during a sustained contraction. Teleologically, the causal relationship between CV and EMG spectral compression can be seen in Figure 3. In this figure, the depolarization zone of a muscle fiber is seen moving past a pair of (differential) electrodes. The slower the CV, the longer will be the time taken by the depolarization zone to pass by the electrodes, and consequently the duration of the detected action potential will be longer. An increased duration of the action potential is reminiscent of the “slowing down” of the EMG signal mentioned at the beginning of this section and causes its frequency spectrum to compress. It should be noted that the arguable effect of the increased depolarization zone can be explained in a similar fashion. That is, if the depolarization zone increases during a sustained contraction, it will take more time for it to pass by the detection electrodes and the time duration of the action potential will increase correspondingly.

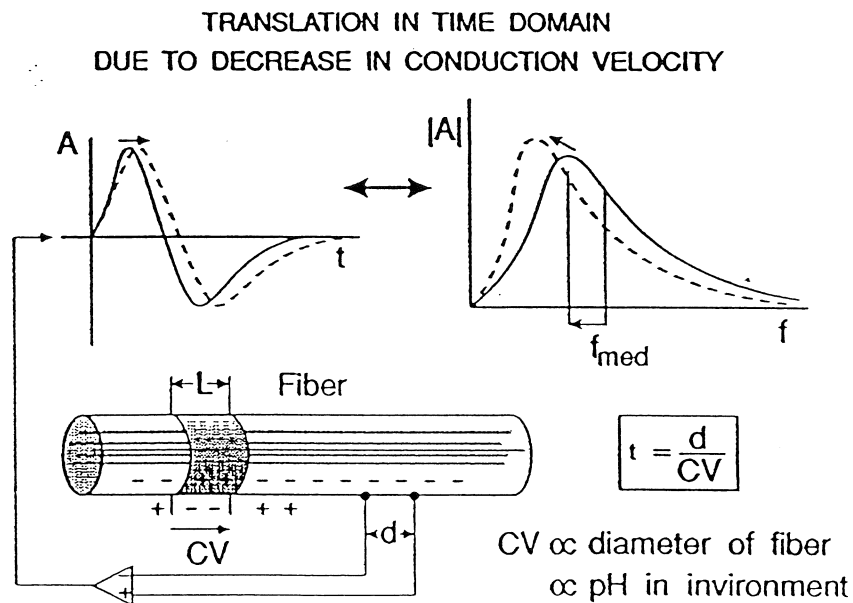
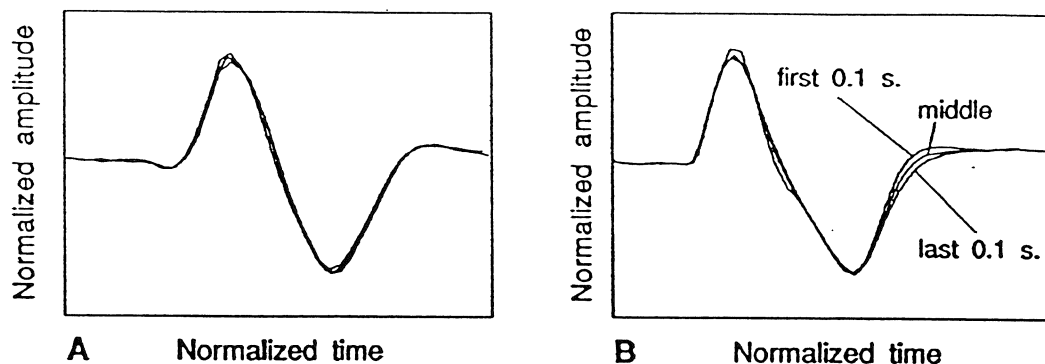


Figure 3. A schematic representation of the causal relationship between the conduction velocity along the muscle fibers and the time duration and spectrum of the detected action potential.

Now the question remains as to what causes the CV to decrease. Recent work in our Center (Brody *et al.*; 1991) demonstrated that the pH of the fluid surrounding the muscle fibers has a profound and causal effect on the CV. In this study, we placed a rat diaphragm nerve-muscle preparation in a bath of Ringer solution into which CO<sub>2</sub>, O<sub>2</sub> and N<sub>2</sub> could be aerated to change the pH of the solution. The pH was fixed at 6.6, 7.0 and 7.4. At each pH setting, the nerve was stimulated supramaximally for 3 s with a pulse train of 40 Hz. The EMG signal from the muscle was detected by a three-bar electrode and amplified with a double-differential configuration. This method

enabled us to separately and simultaneously measure the median frequency and the CV from the same set of EMG signals. In this fashion we found that at the beginning of a contraction (in the first 0.1 s, prior to any significant fatigue) the CV was directly related to the pH value. That is, when the pH was decreased, the time duration of the compound action potential increased and the amplitude decreased; the opposite occurred when the pH was increased. But, the change in the time duration and amplitude were completely accounted for by the corresponding change in the CV. See Figure 4a which compares the compound action potential shapes with the amplitude normalized by the inverse of the CV and the time duration by the CV at each pH level. However, at the end of the sustained 3 s stimulation, the change in the time duration and amplitude of the compound action potential could not be completely accounted for by the change in the CV at the different time intervals; indicating that some other(s) causative agent affected the shape of the compound action potential. See Figure 4b. In particular, the later part of the compound action potential could not be matched, indicating that the recovery phase is slowed down as a function of contraction time. One possible cause for this effect might be the leakage of Potassium ions from the membrane, however, this suggestion remains to be proven.



**Figure 4A.** The shape of the compound action potential detected from the stimulated rat diaphragm muscle at pH = 6.6, 7.0, 7.4. The amplitude and the time scales have been normalized by the conduction velocity value at the corresponding pH. Note the similarity of the shapes. ( See text for explanation.)

**Figure 4B.** The shape of the compound action potential at a set pH at the beginning (0 - 0.1 s), middle and end (2.9 - 3.0 s) of a stimulated contraction. The amplitude and time scales are normalized by the conduction velocity values at the corresponding times. Note that the normalized shapes are not identical. (See text for explanation.)

The amount of pH change is a function of the Lactate that is generated and removed from the muscle fiber environment. Work in progress in our Center indicates that, as anticipated, the fiber type influences the change in the median frequency, with greater changes seen in muscles which contain large proportions of anaerobic Type II fibers (Extensor Digitorum Longus) than those rich in Type I fibers (Soleus) of the rat. This observation was suspected in our earlier work (Merletti, Sabbahi & De Luca; 1984) where the median frequency was measured in the human First Dorsal Interosseous before, during and after ischemia was induced in constant-force isometric contractions at 20 % and 80 % of maximal voluntary contraction (MVC). In that study, the decrease in the median frequency during the contractions was dramatically higher in the 80 % MVC, where the Type II fibers are activated in considerably greater quantity. Thus, the force level of a contraction is critically important when using the EMG spectral technique for at least three reasons:

- 1) Larger diameter fibers (recruited in greater proportion at higher force levels) have larger CVs which produce MUAPs of shorter time duration and higher frequency bandwidths. This yields higher initial values of the median frequency at the beginning of a contraction. See Broman, Bilotto & De Luca (1985).
- 2) Anaerobic Type II fibers (recruited in greater proportion at higher force levels) increase the rate

of Lactate production and decreases the pH during a contraction. This yields higher rate of decrease for the median frequency during a contraction.

3) Blood flow (which is shut off at higher force levels) determines the amount of Lactate that remains in the environment of the muscle fibers. This affects the value of the median frequency. At force levels above 30 % MVC, in most muscles, the blood flow is occluded.

In addition to the concern over the force level, it is also important to monitor the length changes in the muscle during a contraction. Because the electrode is fixed on the skin above the muscle, any change in the muscle length will change the relative distance between the electrode and the active fibers. This would modify the spatial filtering characteristics and thus affect the shape of the MUAP with a corresponding effect on the median frequency.

It is recommended that the EMG spectral technique be used during constant-force isometric contractions above 30 % MVC. If it is to be applied to dynamic contractions, then the median frequency should be compared only when the EMG signal is reasonably stationary and detected at the same phase during a repetitive dynamic contraction. If the values of the median frequency are to be compared across individuals, then the amount of fatty tissue beneath the skin becomes a factor of concern because it affects the spatial filtering characteristics.

#### **Proper use of a new technique:**

When a new technique is used in the research and clinical environments, it is incumbent on the users to apply it with proper respect for and knowledge of its limitations and idiosyncrasies. All new technology has unexplored fringes that require careful considerations. Unchecked usage will provide inconsistent and possibly conflicting results. When using the EMG spectral technique the following technical considerations must be respected:

- 1) The electrode should be sufficiently small and placed well within the borders of the muscle so as to detect the EMG signal from the muscle in question and not crosstalk signal from adjacent muscles. Signals from adjacent muscles will be subjected to greater spatial filtering, thus reducing the value of the spectral variables of the detected signals. We have developed a special electrode, which in most cases, can satisfy the two conditions. The detection surfaces of our electrode consist of two parallel bars, each 1.0 cm long and 1.0 mm wide spaced 1.0 cm apart.
- 2) The spacing between the detection surfaces inversely scales the value of the spectral variables.
- 3) The orientation of the detection surfaces with respect to the muscle fibers also affects the value of the spectral variables.
- 4) The temperature of the muscle directly affects the spectral variables. Tests made for comparison should be made at similar temperatures or scaled appropriately.
- 5) Ambient electromagnetic radiation, motion artifacts, clipping of the signal during detection, and poor signal-to-noise ratio all adversely affect the value of the EMG spectral variables.

#### **Final note**

Given that both the EMG spectral variables and the force variable of the contractile mechanisms undergo changes during the progression of fatigue, it is inevitable to ask if a relationship exists between the two. The answer is undoubtedly yes. The more interesting question is if the relationship is causal. This issue is not clear at this time, and a considerable amount of work is required before meaningful statements can be made to illuminate this issue. Nonetheless the lack of proof of a causal relationship does not logically preclude the use of the spectral variables as a fatigue index, especially when empirical evidence reveals its usefulness. For example, work in progress at our Center (Roy, De Luca & Casavant, 1989) as well as corroborative work by others (Biederman et al., in press) this technique has been successfully adapted to objectively assess muscle impairment associated with lower back pain disorders. Results have demonstrated characteristic "patterns" of EMG median frequency that are reliably different for individuals with muscle insufficiency associated with lower back pain.



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