**Muscle Contraction**

Muscles are remarkably diverse in their size, shape and structure. The longer a muscle, the more it can shorten and higher its velocity of shortening. In contrast, the thicker a muscle, the more force (tension) it can develop. Regardless of their shapes and sizes, all muscles are made up of individual fibers but the number of fibers in muscles differs from a few hundred to a million, depending on the size of muscle [McComas, 1996].

For a muscle to contract, the necessary signals in the form of nerve impulses (action potentials) are sent by large cells, called motoneurons, lying in the spinal cord. There are usually a hundred or more motoneurons for each muscle. When an action potential spreads along the muscle fiber or cell, the fiber begins to contract after an initial latent period of time of about 3 ms [Guyton, 1971]. To effect contraction, the action potential causes electrical current to flow deep into the interior of the muscle fiber. The flow of electrical current through the walls of the transverse tubules causes the sarcoplasmic reticulum to release calcium ions into the surrounding sarcoplasm within a few milliseconds. These ions diffuse around the myofibrils contained within the sarcoplasm and there initiate the contractile process [Guyton, 1971]. The precise chemical and physical events that take place during the contractile process are not fully known, but in general it may be explained as follows. It is believed that in the presence of the troponin-tropomyosin complex, the normal actin filament of the relaxed muscle is inhibited from binding to the myosin filament. The theory of muscle contraction that is widely accepted today has to do with the configuration change in the actin molecule due to the binding of calcium ions to troponin. The binding of calcium ions to the troponin complex changes the normal relationship between the troponin and actin, and produces a new condition that leads to contraction. Such a condition causes the actin and the myosin filaments to bind to each other (Figure 1). Once the binding sites on actin have been made available, the crossbridge of myosin rapidly attaches to actin. It is usually hypothesized that, after the crossbridges formed, the stored adenosine triphosphate (ATP) in one of the heads of myosin, provides energy for the cross bridge to rotate forward while it is still attached to actin. Therefore these filaments slide along each other and cause shortening of the muscle. Although the exact mechanism by which the muscle shortening happens is not yet clear, it is believed that this is the mechanism by which the action potential causes muscle contraction [Enoka, 1994]. However, the calcium ions do not remain in the region of the myofibrils for more than a few milliseconds, because once the electrical current caused by the action potential is over, the sarcoplasmic reticulum almost immediately sequesters the calcium ions out of the sarcoplasm. Therefore, in effect, the action potential causes a short pulse of calcium ions in
the region of the myofibril, and it is during this time that the contractile process is activated. At the termination of this pulse of calcium ions, the muscle immediately relaxes.

**Figure 1** The myosin molecule and its arrangement in the thick filaments of the myofibrils. (A) A single molecule, consisting of a double-helical rod terminating in two globular heads, each of which has two light chains attached. (B) In solution, myosin molecules spontaneously aggregate to form filaments with heads at both ends. (C) In a thick filament, the two globular heads of a myosin molecule project to form a cross-bridge. The next bridge is separated by 14.3 nm and 60°. (D) The overlap of actin and myosin filaments in a relaxed myofibril and the various refractive bands that are created. (E) Contraction is produced by actin filaments sliding over the myosin filament, causing approximation of the Z-lines and narrowing of the H-region. (from Skeletal Muscles [McComas, 1996])

The electrical voltage which is detectable on the surface of muscles during a contraction can be explained by the fairly well established theory of motor units. The axon of each motoneuron that leaves the spinal cord usually innervates many muscle fibers. All the muscle fibers innervated by a single motoneuron along with its large axon and its terminal branches, are called a motor unit [Basmajian & De Luca, 1985] (Figure 2.2). In general, muscles that react rapidly and whose control is precise, have few muscle fibers (as few as 10 to 15) in each motor unit and have a large number of motoneurons directed to the muscle. On the other hand, slow acting muscles, which do not require a very fine degree of control may have as many as 300 to 800 muscle fibers in each motor unit [Guyton, 1971]. Usually muscle fibers of adjacent motor units overlap with small bundles of 10 to 15 fibers from one motor unit lying among similar bundles of the second motor unit. This configuration allows the separate motor
units to contract in support of each other rather than entirely as an individual segment [Guyton, 1971]. The force of contraction increases progressively as the number of contracting motor units increases. That is, as more and more motor units are activated simultaneously, the strength of contraction increases. In addition, successive muscle twitches overlap in time, which means that the first muscle twitch is not completely over by the time that the second one begins. Therefore, since the muscle is already in a partially contracted state when the second twitch begins, the degree of muscle shortening this time is slightly greater than that which occurs with a single muscle twitch. Consequently, at more rapid rates of contraction, the degree of summation of successive contractions becomes greater and greater until an optimum frequency is reached [Guyton, 1971].

**Figure 2** The anatomy of a motor unit. (A) Cross section of the spinal cord (B) Enlargement of anterior horn with motoneurons, one of which shown sending its axon through the ventral root to the muscle (below). (C) The muscle fibers supplied by the branches of the single axon are darkened. This population of fibers, together with the motor axon and motoneuron, constitute a single motor unit. (*from Skeletal Muscle [McComas, 1996]*)

Motor units differ from each other not only in size, but also in their recruitment order and firing rate. The most consistent observation of motor unit behavior reported in the literature concerns the order of recruitment as a function of motoneuron size. In a steady contraction, small motor units are recruited before the large ones [McComas, 1996]. It has been shown that the slower conduction velocities, and thus the smaller axons, are associated with the lower threshold motor units [Freund et al, 1973]. The order of motor unit recruitment is also a function of fiber type and size of muscle. Type I fibers, which are known as slow twitch fibers are recruited first followed by type II (fast twitch) fibers. In sudden
movements type II fibers may have the lowest threshold and be recruited first [McComas, 1996]. Small muscles recruit all their motor units below 50% maximum voluntary contraction (MVC) and larger muscles recruit motor units throughout the full range of voluntary force [Basmajian & De Luca, 1985]. However, the order of motor unit recruitment may change if the functional role of the same muscle changes, e.g. McComas [1996] cited Gielen and Denier Van Der Gon [1990] to state that “in the biceps the threshold of a motor unit depends on whether the muscle is being used to flex the elbow, supinate the forearm, or externally rotate humerus.” [McComas, 1996, page 208]

Firing rates of motor units are also muscle dependent. In general the smaller the muscle, the higher the firing rate. During sustained contractions, the firing rate of motor units decreases as a function of time and this phenomenon is more evident in fast-twitch fibers than in slow-twitch fibers [Basmajian & De Luca, 1985]. Table 1 summarizes the discussed muscle fiber characteristics.

Force can be generated by increasing the firing rate of the motor units as well as by recruitment of new motor units. It is a common belief that at the beginning of a contraction, recruitment of new motor units is the dominant factor with the smallest motor units being recruited first while the increase of firing rate plays a secondary role. However, for force levels ranging from 30% to 75% of MVC, the dominant factor is an increase in firing rate. Some recruitment of larger motor units also occurs but this factor plays a secondary role [Basmajian & De Luca, 1985].

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Type I</th>
<th>Type II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diameter</td>
<td>Small</td>
<td>Large</td>
</tr>
<tr>
<td>Muscle Color</td>
<td>Red</td>
<td>White</td>
</tr>
<tr>
<td>Speed of Contraction</td>
<td>Slow</td>
<td>Fast</td>
</tr>
<tr>
<td>Rate of Fatigue</td>
<td>Slow</td>
<td>Fast</td>
</tr>
<tr>
<td>Motor Unit Size</td>
<td>Large</td>
<td>Small</td>
</tr>
<tr>
<td>Axon Conduction Velocity</td>
<td>Slow</td>
<td>Fast</td>
</tr>
<tr>
<td>Major Function</td>
<td>Tonic</td>
<td>Phasic</td>
</tr>
</tbody>
</table>

**Table 1** Summary of the muscle fiber characteristics (*from Norkin & Levangie, 1992*).
**Muscle Fatigue**

Sustained and strong contraction of a muscle leads to the well-known state of muscle fatigue. This is simply the inability of the contractile processes of the muscle fiber to continue to produce the same amount of work. In fact, the nerve continues to function properly, the nerve impulses pass normally through the neuromuscular junction onto the muscle fiber, and even normal action potentials spread over the muscle fibers, but the contraction becomes weaker and weaker because of depletion of energy supplies in the muscle fibers themselves. If a muscle becomes fatigued to an extreme extent, it is likely to become continually contracted, remaining contracted and rigid for many minutes even without an action potential as a stimulus. This is called *physiological contracture* of the muscle [Guyton, 1971]. At the physiological fatigue stage, a burst of activity can still be achieved as a result of encouragement or fear, its duration is however, highly variable and depends on the emotional state of the individual.

An assumed mechanism for peripheral fatigue is that the ATP is required to make the actin and myosin molecules separate during the process of muscle relaxation, otherwise the myosin and actin filaments will continue to be bound to each other. In extreme degrees of muscle fatigue, essentially all the ATP is depleted; this accounts for the rigid binding of the actin and myosin filaments in the contracted state of the muscle. The reasons for ATP depletion are many. A full discussion is beyond the scope of this study.

**Electromyography (EMG)**

As was explained in Section 2.1, an action potential propagating down the axon of a motor nerve is transmitted chemically to the muscle fibers in the motor unit. This action potential gives rise to a shortening of the muscle fibers which in turn produces force; the signal resulting from the action potential in each muscle fiber of the motor unit is called the motor unit action potential (MUAP). However, in order to sustain a muscle contraction the motor unit must be repeatedly activated; hence resulting in a train of motor unit action potentials (MUAPT). The MUAPTs are, as a rule, completely asynchronous and this, along with the low pass dynamics of the muscle fibers, results in a smooth force output [Basmajian & De Luca, 1985]. The action potentials propagating along the muscle fibers give rise to an electrical signal which can be detected by surface or intramuscular electrodes. The resulting EMG signal, represents the gross electrical activity of the active motor units in the vicinity of the electrodes.
In human muscle tissue, the amplitude of the action potentials is dependent upon the diameter of the muscle fiber, the distance between the active muscle fiber and the detection site, and the filtering properties of the electrode [Basmajian & De Luca, 1985]. The amplitude of the EMG signal increases as the radius of the muscle fiber increases, and it decreases in approximately inverse proportion to the distance between the active fiber and the detection site. The action potential duration is inversely related to the conduction velocity of the nerve branch and muscle fiber [Basmajian & De Luca, 1985]. The waveform, and therefore the frequency spectrum of the action potentials, will be affected by the tissue between the muscle fiber and the detection site.

**EMG Parameters**

Several parameters of the EMG signal, such as root mean square (RMS) of the amplitude, median power frequency (MDPF), mean power frequency (MPF), the ratio of the RMS value of high-frequency components to the RMS value of the low-frequency components (RP), and peak frequency (PF) have been considered by researchers through the years. Stulen and De Luca [1981] compared the MDPF, MPF and RP. They pointed out that RP is the most sensitive parameter to changes in the conduction velocity of the muscle fiber. However, it was discarded due to several drawbacks such as having a non-linear relationship with changes in the conduction velocity and being dependent on the initial value of the characteristic frequency chosen to divide the spectrum. Stulen and De Luca [1981] also showed that MDPF and MPF have an equal linear sensitivity to the conduction velocity, with the MDPF about 20% less sensitive to signal to noise ratio than the MPF and concluded that MDPF is the most reliable parameter of EMG. In another study Hary et al [1982], concluded that MPF is the preferred parameter because of its lower standard deviation compared to that of MDPF and therefore smaller threshold for detecting statistically significant changes. Balestra et al. [1988] also compared the performance of MDPF and MPF parameters in both simulated EMG signals and EMG signals recorded from muscle during sustained muscle contraction. Their results showed that in both simulated and voluntary EMG signals, the MPF had a standard deviation at least 35% smaller than that of the MDPF regardless of the spectral estimation algorithm used.

**EMG and Fatigue**

The relationship of EMG parameters to muscle fatigue has been discussed for many years. It is commonly held that EMG behavior during the fatigue process is associated with a shift of its power spectrum toward lower frequencies and an increase in the RMS of the time signal. However, a difference in the trend of the power spectrum and the RMS shift in relation to the functional role of the
muscle has been found [Moussavi et al, 1996]. Motor unit recruitment, motor unit synchronization, and a change in the conduction velocity of muscle fibers are three explanations that have been proposed to account for changes observed during a sustained, isotonic, and isometric contraction. According to Basmajian and De Luca [1985] the motor unit recruitment is unlikely to be responsible for the spectral changes of the EMG signal because amplitude increase and frequency shift have been observed in the first dorsal interosseous muscle during 80% of MVC and it is believed that at this contraction level, there is no further recruitment of motor units. Synchronization, which is defined as the tendency for motor units to discharge at almost the same time, has often been cited as the cause of both frequency shift and amplitude increase. However, synchronization of motor units has been reported to be more evident at the end of endurance time of contraction, whereas the frequency shift is more pronounced at the beginning of a contraction [Basmajian & De Luca, 1985]. Conduction velocity along the muscle fibers has been reported to decrease during a sustained contraction. To be more specific, using the model of Lindstrom et al [1970], the power spectrum of the EMG signal may be expressed as:

\[ S(f) = (\frac{1}{\nu^2}).G(\frac{\nu}{\nu}) , \]

where \( \nu \) is the conduction velocity of muscle, and \( G(.) \) is a mathematical model of the power spectrum of the EMG signal as a function of frequency and is implicitly dependent on many anatomical and experimental factors. \( G(.) \) may be expressed as

\[ G(f) = \frac{F[\sigma_e^2(t)]]}{E(k_j)^2.k_a}.K , \]

where \( E(.) \) is the expectation operation, \( F[ ] \) is the Fourier transform, \( k_a \) is the average firing rate of a motor unit train, \( \sigma_e^2 \) is the mean squared value of the EMG signal, \( k_j \) is a random variable, and \( K \) is a constant [Shwedyk et al, 1977].

From the above equations, it is possible to draw the following observations concerning the behavior of the spectrum:

- A decrease in the firing rates of motor units contributes to a shift of the power spectrum toward lower frequencies.
- A decrease in conduction velocity causes the power spectrum to shift toward lower frequencies because the time-duration of the wave form increases as the conduction velocity decreases during sustained contraction and this would cause a simultaneous increase in the low-frequency components and a decrease in the high-frequency components.

There are a few hypotheses regarding the reason for the decrease in conduction velocity during a sustained contraction. They basically relate the decrease in conduction velocity to the changes of
intramuscular pH, and explain it with biochemical changes in the muscle fiber during a sustained contraction. However, the interaction between pH and conduction velocity is not firmly established [Basmajian & De Luca, 1985].

While the power spectrum shift of EMG with fatigue is a well established phenomenon, the RMS increase, however, is associated with ambiguity and controversy. Several investigators have studied the EMG signs of fatigue for trapezius muscle simultaneously with those of deltoid in an ab ducted or flexed arm position [Hansson et al, 1992; Gerdle et al, 1993]. While the results are similar, the interpretations are different and to some extent controversial. Since trapezius is considered to be a fatigue-resistant muscle with a predominance of type I muscle fibers, its less pronounced spectrum shift compared to that of deltoid is expected and has commonly been reported [Hansson et al, 1992; Oberg & Sandsjo, 1992; Gerdle et al, 1993]. However, Hansson et al. [1992] reported that the RMS value for trapezius showed a significant increase during an isometric fatigue process, the presented graphs showed that the deltoid’s RMS remained constant or decreased. Some investigators have explained this difference by invoking the fact that the fiber type composition of the two muscles is different [Hansson et al, 1992; Gerdle et al, 1993]. However, this explanation raises some controversial aspects about EMG behavior during fatigue. A simple fact, which has been ignored in previous research, is the functional role of the involved muscle, which may be a crucial determinant of the EMG signal characteristics. The hypothesis that the existence of such a relationship is an indicator of RMS behavior, has been studied in this research and is discussed in chapter 4.