BIOMECHANICS AND MOTOR CONTROL OF HUMAN MOVEMENT

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Muscle Mechanics

7.0 INTRODUCTION

The most intriguing and challenging area of study in biomechanics is probably the muscle itself. It is the "living" part of the system. The neural control, metabolism, and biomechanical characteristics of muscle are subjects of continuing research. The purpose of this chapter is to report the state of knowledge with regard to the biophysical characteristics of individual motor units, connective tissue, and the total muscle itself. The characteristics of the individual units are described in detail, and it is shown how these characteristics influence the biomechanical function of the overall muscle.

7.0.1 The Motor Unit

The smallest subunit that can be controlled is called a *motor unit* because it is innervated separately by a motor axon. Neurologically the motor unit consists of a synaptic junction in the ventral root of the spinal cord, a motor axon, and a motor end plate in the muscle fibers. Under the control of the motor unit are as few as three muscle fibers or as many as 2000, depending on the fineness of the control required (Feinstein et al., 1955). Muscles of the fingers, face, and eyes have a small number of shorter fibers in a motor unit, while the large muscles of the leg have a large number of long fibers in their motor units. A muscle fiber is about 100 μm in diameter and consists of fibrils about 1 μm in diameter. Fibrils in turn consist of filaments about 100 Å in diameter. Electron micrographs of fibrils show the basic mechanical structure of the interacting actin and myosin filaments. In the schematic diagram of Figure 7.1 the darker and wider myosin protein bands are interlaced with the lighter and smaller actin protein bands. The space between them consists of a cross-

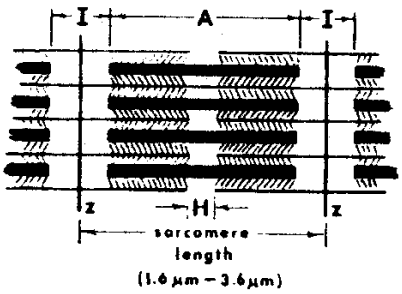


Figure 7.1 Basic structure of the muscle contractile element showing the Z lines and sarcomere length. Wider dark myosin filament interacts across cross bridges (crosshatched lines) with the narrower actin filament. Darker and lighter bands (A, H, and I) are shown.

bridge structure, and it is here that the tension is created and the shortening or lengthening takes place. The term *contractile element* is used to describe the part of the muscle that generates the tension, and it is this part that shortens and lengthens as positive or negative work is done. The basic length of the myofibril is the distance between the Z lines and is called the *sarcomere length*. It can vary from 1.5 μm at full shortening to 2.5 μm at resting length to about 4.0 μm at full lengthening.

The structure of the muscle is such that many filaments are in parallel and many sarcomere elements are in series to make up a single contractile element. Consider a motor unit of a cross-sectional area of 0.1 cm^2 and a resting length of 10 cm. The number of sarcomere contractile elements in series would be 10 cm/2.5 μm = 40,000 and the number of filaments (each with an area of 10^{-8} cm^2) in parallel would be $0.1/10^{-8} = 10^7$. Thus the number of contractile elements of sarcomere length packed into this motor unit would be 4×10^{11}.

The active contractile elements are contained within another fibrous structure of connective tissue called *fascia*. These tissue sheaths enclose the muscles, separating them into layers and groups and ultimately connecting them to the tendons at either end. The mechanical characteristics of connective tissue are important in the overall biomechanics of the muscle. Some of the connective tissue is in series with the contractile element, while some is in parallel. The effect of this connective tissue has been modeled as springs and viscous dampers, and is discussed in detail in Section 7.3.

7.0.2 Recruitment of Motor Units

Each muscle has a finite number of motor units, each of which is controlled by a separate nerve ending. Excitation of each unit is an all-or-nothing event. The electrical indication is a motor unit action potential; the mechanical result is a twitch of tension. An increase in tension can therefore be accomplished in two ways: by an increase in the stimulation rate for that motor unit or by the excitation (recruitment) of an additional motor unit. Figure 7.2 shows the EMG of a needle electrode in a muscle as the tension was gradually

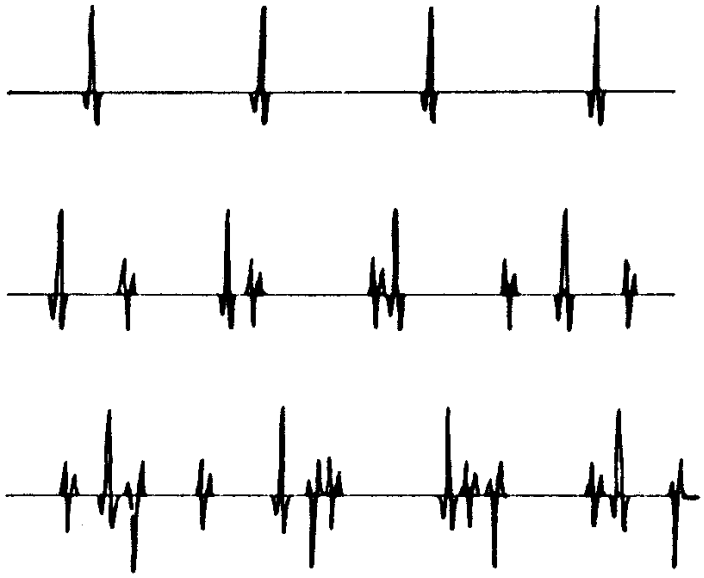


Figure 7.2 EMG from an indwelling electrode in a muscle as it begins to develop tension. The smallest motor unit is first recruited, and as its rate increases, a second, then a third motor unit are recruited. Each motor action potential has a characteristic shape at a given electrode, which depends on the size of the motor unit and the distance from the electrode to the fibers of the motor unit (see Section 8.1.3).

increased. The upper tracing shows one motor unit firing, the middle trace two motor units, and the lower trace three units. Initially muscle tension increases because the firing rate increases. At a certain tension the second motor unit was recruited, and further tension increases are then accomplished by increases in the rate of the second motor unit plus possible further increases in the rate of the first unit. As each unit has a maximum firing rate, it appears that this maximum rate is reached after the next unit is recruited. When the tension is reduced, the reverse process occurs. The firing rate of the recruited units decreases until the minimum rate for the last recruited unit is reached, at which point the unit drops out. Each unit usually drops out in reverse to the order in which it was recruited. A mathematical model describing this phenomenon was developed by Wani and Guha (1975) and Milner-Brown and Stein (1975).

7.0.3 Size Principle

Considerable research and controversy has taken place over the past two decades over how the motor units are recruited. Which ones are recruited first? Are they always recruited in the same order? It is now generally accepted that they are recruited according to the *size principle* (Henneman, 1974*a*), which states that the size of the newly recruited motor unit increases with the tension level at which it is recruited. This means that the smallest unit is recruited first and the largest unit last. In this manner low tension movements can be achieved in finely graded steps. Conversely, those movements requiring high forces but not needing fine control are accomplished by

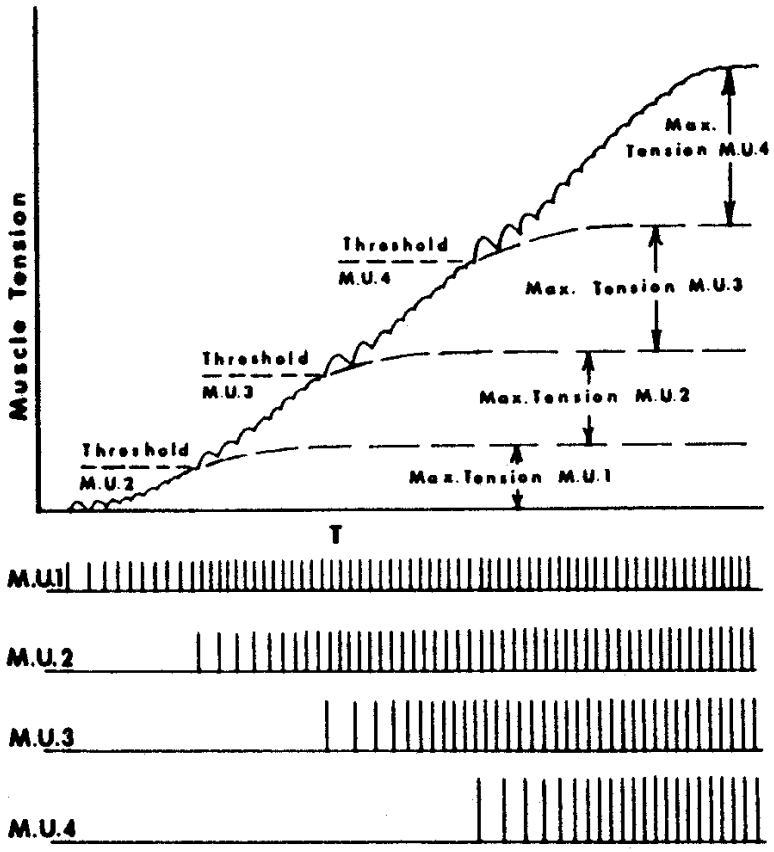


Figure 7.3 Size principle of recruitment of motor units. Smaller motor units are recruited first; successively larger units begin firing at increasing tension levels. In all cases the newly recruited unit fires at a base frequency, then increases to a maximum.

recruiting the larger motor units. Figure 7.3 depicts a hypothetical tension curve resulting from successive recruitment of several motor units. The smallest motor unit (MU 1) is recruited first, usually at an initial frequency ranging from about 5–13 Hz. Tension increases as MU 1 fires more rapidly, until a certain tension is reached at which MU 2 is recruited. Here MU 2 starts firing at its initial low rate and further tension is achieved by the increased firing of both MU 1 and 2. At a certain tension MU 1 will reach its maximum firing rate (15–60 Hz) and will therefore be generating its maximum tension. This process of increasing tension, reaching new thresholds, and recruiting another larger motor unit continues until maximum voluntary contraction is reached (not shown in Figure 7.3). At that point all motor units will be firing at their maximum frequencies. Tension is reduced by the reverse process: successive reduction of firing rates and dropping out of the larger units first (DeLuca et al., 1982). During rapid ballistic movements the firing rates of individual motor units have been estimated to reach 120 Hz (Desmedt and Goodaux, 1978).

The motor unit action potential increases with the size of the motor unit with which it is associated (Milner-Brown and Stein, 1975). The reason for this appears to be twofold. The larger the motor unit, the larger the motoneuron that innervates it, and the greater the depolarization potentials seen at the

motor end plate. Second, the greater the mass of the motor unit, the greater the voltage changes seen at a given electrode. However, it is never possible from a given recording site to predict the size of a motor unit because its action potential also decreases with the distance between the recording site and the electrode. Thus a large motor unit located a distance from the electrode may produce a smaller action potential than that produced by a small motor unit directly beneath the electrode.

7.0.4 Types of Motor Units—Fast- and Slow-Twitch Classification

There have been many criteria and varying terminologies associated with the types of motor units present in any muscle (Henneman, 1974*b*). Biochemists have used metabolic or staining measures to categorize the fiber types. Biomechanics researchers have used force (twitch) measures (Milner-Brown et al., 1973*b*), and electrophysiologists have used EMG indicators (Wormolts and Engel, 1973; Milner-Brown and Stein, 1975). The smaller slow-twitch motor units have been called *tonic units*. Histochemically they are the smaller units (type I) and metabolically they have fibers rich in mitochondria, are highly capillarized, and therefore have a high capacity for aerobic metabolism. Mechanically they produce twitches with a low peak tension and a long time to peak (60–120 ms). The larger fast-twitch motor units are called *phasic units* (type II). They have less mitochondria, are poorly capillarized, and therefore rely on anaerobic metabolism. The twitches have larger peak tensions in a shorter time (10–50 ms). Figure 7.4 is a typical histochemical stain of fibers of

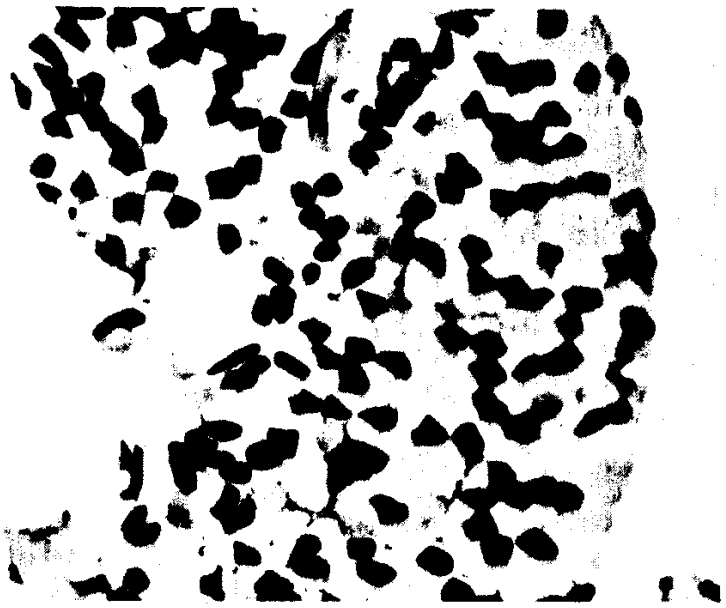


Figure 7.4 Histochemical stain showing dark slow-twitch fibers and light fast-twitch fibers. A myofibrillar ATPase stain, pH 4.3, was used to stain the vastus lateralis of a female volleyball player. (Reproduced by permission of Professor J. A. Thomson, University of Waterloo, Waterloo, Ont., Canada.)

a muscle that contains both slow- and fast-twitch fibers. An ATPase-type stain was used here, so slow-twitch fibers appear dark and fast-twitch fibers appear light. If an indwelling microelectrode were present in the area of these fibers, the muscle action potential from these darker slow-twitch fibers would be smaller than that from the lighter stained fast-twitch fibers.

In spite of the histochemical classification described, there is growing biophysical evidence (Milner-Brown et al., 1973*b*) that the motor units controlled by any motoneuron pool form a continuous spectrum of sizes and excitabilities.

7.0.5 The Muscle Twitch

Thus far we have said very little about the smallest increment of tension, that of the individual twitch itself. As was described in Section 7.0.4, each motor unit has its unique time course of tension. Although there are individual differences in each newly recruited motor unit, they all have the same characteristic shape. The time-course curve follows quite closely that of the impulse response of a critically damped second-order system (Milner-Brown et al., 1973*a*). The electrical stimulus of a motor unit, as indicated by this action potential, is of short duration and can be considered an impulse. The mechanical response to this impulse is the much longer duration twitch. The general expression for a second-order critically dampled impulse response is

$$F(t) = F_0 \frac{t}{T} e^{-t/T} \tag{7.1}$$

For the curve plotted in Figure 7.5 the twitch time T is the time for the tension to reach a maximum and F_0 is a constant for that given motor unit. T is the contraction time and is larger for the slow-twitch fibers than for the

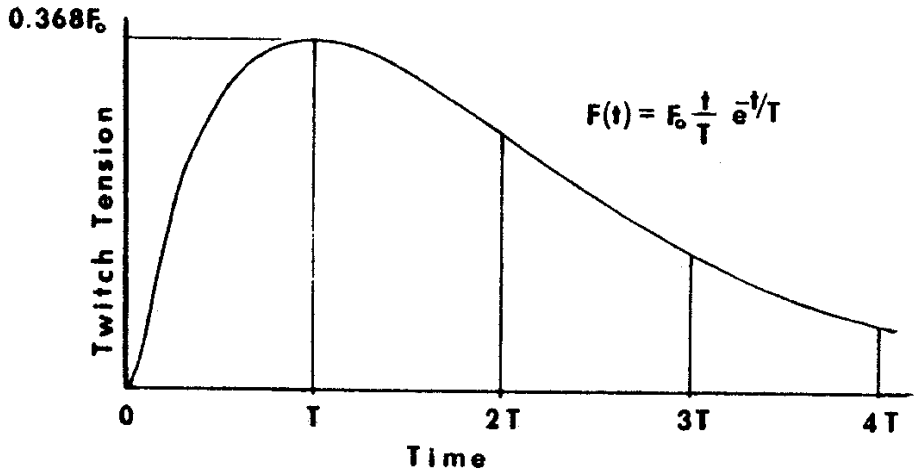


Figure 7.5 Time course of a muscle twitch, modeled as the impulse response of a second-order critically damped system. Contraction time T varies with each motor unit type, from about 20 to 100 ms. Effective tension lasts until about $4T$.

fast-twitch motor units, while F_0 increases for the larger fast-twitch units. Muscles tested by Buchthal and Schmalbruch (1970) using submaximal stimulations showed a wide range of contraction times. Muscles of the upper limbs generally had short T values compared with the leg muscles. Typical mean values of T and their range were

Triceps brachii	44.5 ms (16–68 ms)
Biceps brachii	52.0 ms (16–85 ms)
Tibialis anterior	58.0 ms (38–80 ms)
Soleus	74.0 ms (52–100 ms)
Medial gastrocnemius	79.0 ms (40–110 ms)

These same researchers found that T increased in all muscles as they were cooled; for example, the biceps brachialis had a contraction time that increased from 54 ms at 37°C to 124 ms at 23°C. It is evident that the cause of the delayed buildup of tension is the slower metabolic rates and increased muscle viscosity seen at lower temperatures.

Many other researchers have tested other muscles using different techniques. Milner-Brown et al. (1973*b*) looked at the first dorsal interossei during voluntary contractions and measured T to average 55 ms (±12 ms) while Desmedt and Godaux (1978) calculated a mean of 60 ms on the same muscle but used supramaximal stimulations. These latter researchers using the same technique reported T for the tibialis anterior as 84 ms and for the soleus as 100 ms. Bellemare et al. (1983), also using supramaximal stimulations, reported soleus T = 116 ms (±9 ms) and biceps T = 66 ms (±9 ms). Thus it appears that the contraction time varies considerably depending on the muscle, the person, and the experimental technique employed.

7.0.6 Shape of Graded Contractions

The shape of a voluntary tension curve depends to a certain extent on the shape of the individual muscle twitches. For example, if we elicit a maximum contraction in a certain muscle, the rate of increase of tension depends on the individual motor units and how they are recruited. Even if all motor units were turned on at the same instant and fired at their maximum rate, maximum tension could never be achieved in less than the average contraction time for that muscle. However, as described in Figure 7.2 and depicted in Figure 7.3, the recruitment of motor units does not take place all at once during a voluntary contraction. The smaller slow-twitch units are recruited first in accordance with the size principle, with the largest units not being recruited until tension has built up in the smaller units. Thus it can take several hundred milliseconds to reach maximum tension. During voluntary relaxation of the same muscle the drop in tension is governed by the shape of the trailing edge of the twitch curve. This delay in the drop of tension is even more pro-

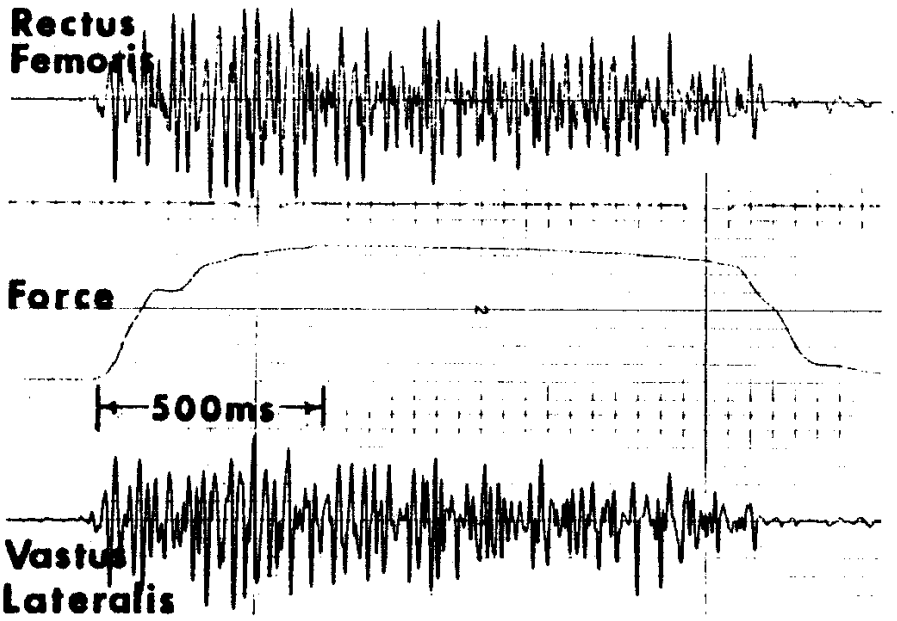


Figure 7.6 Tension buildup and decrease during a rapid maximum voluntary contraction and relaxation. The time to peak tension can be 200 ms or longer, mainly because of the recruitment according to the size principle and because of delay between each motor unit action potential and twitch tension. Note the presence of tension for about 150 ms after the cessation of EMG activity.

nounced than the rise. This delay, combined with the delay in dropping out of the motor units themselves according to the size principle, means that a muscle takes longer to turn off than to turn on. Typical turn-on times could be 200 ms and turn-off times 300 ms, as shown in Figure 7.6, where maximum rapid turn-on and turn-off are plotted showing the tension curve and the associated EMG.

7.1 FORCE–LENGTH CHARACTERISTICS OF MUSCLES

As indicated in Section 7.0.1, the muscle consists of an active element, called the *contractile element,* and passive connective tissue. The net force–length characteristics of a muscle are a combination of the force–length characteristics of both active and passive elements.

7.1.1 Force–Length Curve of the Contractile Element

The key to the shape of the force–length curve is the changes of the structure of the myofibril at the sarcomere level (Gordon et al., 1966). Figure 7.7 is a schematic representation of the myosin and actin cross-bridge arrangement. At resting length, about 2.5 μm, there are a maximum number of cross bridges between the filaments, and therefore a maximum tension is possible. As the muscle lengthens, the filaments are pulled apart, the number of cross

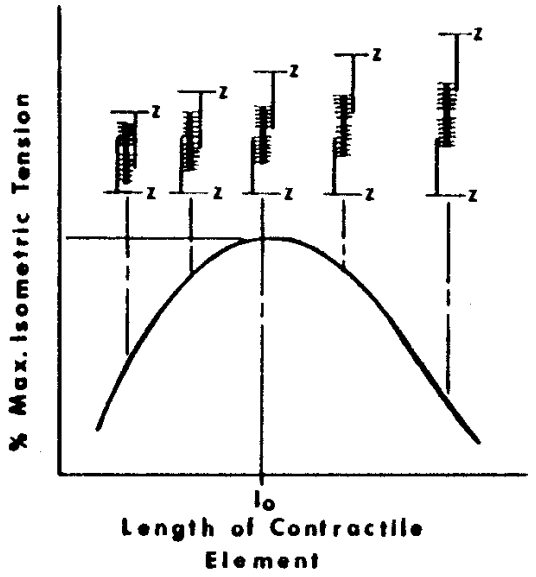


Figure 7.7 Tension produced by a muscle as it changes length about its resting length l_0. Drop of tension on either side of maximum can be explained by interactions of cross-bridge attachments in the contractile elements.

bridges reduce, and tension decreases. At full length, about 4.0 μm, there are no cross bridges and the tension reduces to zero. As the muscle shortens to less than resting length, there is an overlapping of the cross bridges and an interference takes place. This results in a reduction of tension that continues until a full overlap occurs, at about 1.5 μm. The tension doesn't drop to zero, but is drastically reduced by these interfering elements.

7.1.2 Influence of Parallel Connective Tissue

The connective tissue that surrounds the contractile element influences the force–length curve. It is called the *parallel elastic component,* and it acts much like an elastic band. When the muscle is at resting length or less, the parallel elastic component is in a slack state with no tension. As the muscle lengthens, the parallel element is no longer loose, so tension begins to build up, slowly at first and then more rapidly. Unlike most springs, which have a linear force–length relationship, the parallel element is quite nonlinear. In Figure 7.8 we see the force–length curve of this element F_p combined with that of the overall contractile component F_c. If we sum the forces from both elements, we see the overall muscle force–length characteristic F_t. The force–length curve typically presented is usually for a maximum contraction. The passive force F_p of the parallel element is always present, but the amount of active tension in the contractile element at any given length is under voluntary control. Thus the overall force–length characteristics are a function of the percentage of excitation, as seen in Figure 7.9.

The student can demonstrate the drop of tension at either end of the force–length curve by two simple experiments. The hamstrings, as a two-joint mus-

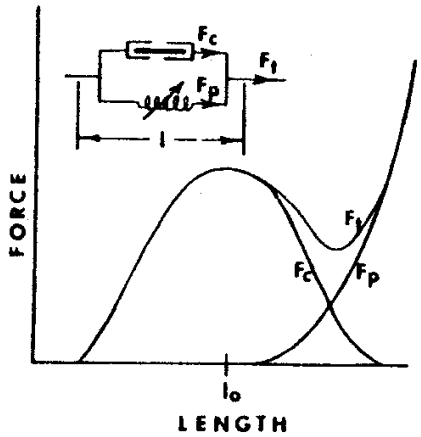


Figure 7.8 Contractile element producing maximum tension F_c along with the tension F_p from the parallel elastic element. Tendon tension is $F_t = F_c + F_p$.

cle, can be made to shorten as follows: the person stands on one leg, leaning backward with the swing hip fully extended, then contracts the hamstrings to flex the leg. He will feel the tension decrease drastically when the hamstring muscles shorten before the knee is completely flexed. The converse situation can be realized if the person attempts to extend the hip joint while the knee is fully extended.

7.1.3 Series Elastic Tissue

All connective tissue in series with the contractile component, including the tendon, is called the *series elastic element.* Under isometric contractions it will lengthen slightly as tension increases. However, during dynamic situations the series elastic element, in conjunction with viscous components, does influence the time course of the muscle tension.

During isometric contractions the series elastic component is under tension and therefore is stretched a finite amount. Because the overall length of the

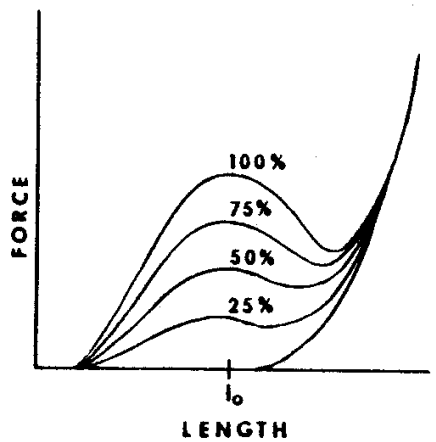


Figure 7.9 Tendon tension resulting from various levels of muscle activation. Parallel elastic element generates tension independent of the activation of the contractile element.

muscle is kept constant, the stretching of the series elastic element can only occur if there is an equal shortening of the contractile element itself. This is described as internal shortening. Figure 7.10 illustrates this point at several contraction levels. Although the external muscle length L is kept constant, the increased tension from the contractile element causes the series elastic element to lengthen by the same amount as the contractile element shortens internally. The amount of internal shortening from rest to maximum tension is only a few percent of the resting length in most muscles (van Ingen Schenau, 1984), but has been shown to be as high as 7% in others (Bahler, 1967). It is widely inferred that these series elastic elements store large amounts of energy as muscles are stretched prior to an explosive shortening in athletic movements. However, van Ingen Schenau (1984) has shown that the elastic capacity of these series elements is far too small to explain improved performances resulting from prestretch. He argues that some other mechanism at the cross bridges must be responsible.

Experiments to determine the force–length characteristics of the series element can only be done on isolated muscle and will require dynamic changes of force or length. A typical experimental setup is shown in Figure 7.11. The muscle is stimulated to a certain level of tension while held at a certain isometric length. The load (tension) is suddenly dropped to zero by releasing one end of the muscle. With the force suddenly removed, the series elastic element, which is considered to have no mass, suddenly shortens to its relaxed length. This sudden shortening can be recorded, and the experiment repeated for another force level until a force-shortening curve can be plotted. During the rapid shortening period (about 2 ms) it is assumed that the contractile element has not had time to change length, even though it is still generating tension. Two other precautions must be observed when conducting this experiment. First, the isometric length prior to the release must be such that there is no tension in the parallel elastic element. This will be so if the muscle

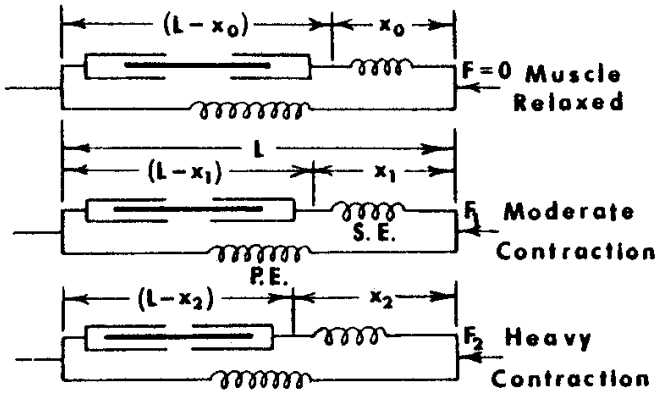


Figure 7.10 Introduction of the series elastic (S.E.) element. During isometric contractions the tendon tension reflects a lengthening of the series element and an internal shortening of the contractile element. During most human movement the presence of the series elastic element is not too significant, but during high-performance movements such as jumping it is responsible for storage of energy as a muscle lengthens immediately prior to rapid shortening.

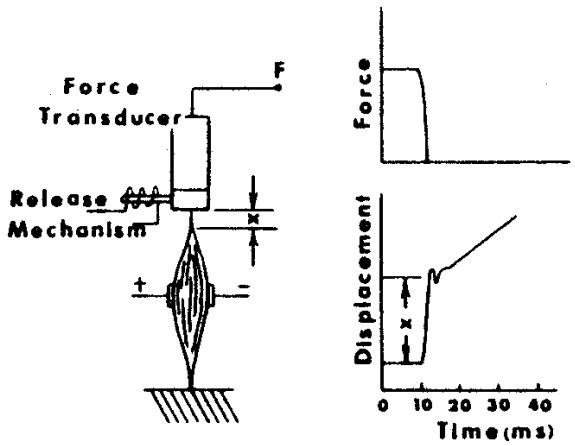


Figure 7.11 Experimental arrangement to determine the spring constant of the series elastic element. Muscle is stimulated; after the tension builds up in the muscle, the release mechanism activates, allowing an almost instantaneous shortening x, while the force change is measured on a force transducer.

is at resting length or shorter. Second, the system that records the sudden shortening of the series elastic element must have negligible mass and viscosity. Because of the high acceleration and velocity associated with the rapid shortening, the resisting force of the attached recording transducer should be negligible. If such conditions are not possible, a correction should be made to account for the mass or viscosity of the transducer.

7.1.4 In Vivo Force–Length Measures

Length–tension relationships of striated muscle have been well researched in mammalian and amphibian species, but in humans the studies have been limited to changes in joint moments during maximum, voluntary contraction (MVC) as the joint angle is altered. Two problems arise in such studies. First, it is usually impossible to generate an MVC for a single agonist without activating the remaining agonists. Thus the joint moment is the sum of the moments generated by several agonists, each of which is likely to be operating at a different point in its force–length curve. Also, the moment generated by any muscle is a product of its force and its moment arm length, both of which change as the joint angle changes. Thus any change in joint moment with joint angle cannot be attributed uniquely to the length–tension characteristics of the muscle.

One in vivo study that did yield some meaningful results was reported by Sale et al. (1982), who quantified the plantarflexor moment changes as the ankle angle was positioned over a range of 50° (30° plantarflexion to 20° dorsiflexion). The contribution of the gastrocnemius was reduced by having the knee flexed to 90°, thus causing it to be slack. Thus the contribution of the soleus was considered to be dominant. Also, the range of movement of the ankle was considered to have limited influence on the moment arm length of the soleus. Thus the moment–ankle angle curve was considered to reflect

the force–length characteristics of the soleus muscle. The results of the MVC, peak twitch tensions and a 10-Hz tetanic stimulation, were almost the same: the peak tension was seen at 15° dorsiflexion and dropped off linearly to near zero as the ankle plantarflexed to 30°. Such a curve agreed qualitatively with a muscle whose resting length was at 15° dorsiflexion and whose cross bridges reached a full overlap of 30° plantarflexion.

7.2 FORCE–VELOCITY CHARACTERISTICS

The previous section was concerned primarily with isometric contractions, and most physiological experiments are conducted under such conditions. However, movement cannot be accomplished without a change in muscle length. Alternate shortening and lengthening occurs regularly during any given movement, so it is important to see the effect of muscle velocity on muscle tension.

7.2.1 Concentric Contractions

The tension in a muscle decreases as it shortens under load. The characteristic curve that describes this effect is called a *force–velocity curve* and is shown in Figure 7.12. The usual curve is plotted for a maximum contraction. However, this condition is rarely seen except in athletic events, and then only for short bursts of time. In Figure 7.12 the curves for a 75%, 50%, and 25% contraction are shown as well. Isometric contractions lie along the zero-velocity axis of this graph and should be considered as nothing more than a special case within the whole range of possible velocities. It should be noted that this curve represents the characteristics at a certain muscle length. To incorporate length as a variable as well as velocity requires a three-dimensional plot, as is discussed in Section 7.2.3.

The decrease of tension as the shortening velocity increases has been attributed to two main causes. A major reason appears to be the loss in tension as the cross bridges in the contractile element break and then reform in a shortened condition. A second cause appears to be the fluid viscosity in both the contractile element and the connective tissue. Such viscosity results in friction that requires an internal force to overcome and therefore results in a reduced tendon force. Whichever the cause of loss of tension, it is clear that the total effect is similar to that of viscous friction in a mechanical system and can therefore be modeled as some form of fluid damper. The loss of tension related to a combination of the number of cross bridges breaking and reforming and passive viscosity makes the force–velocity curve somewhat complicated to describe. If all the viscosity were passive, the slope of the force–velocity curve would be independent of activation. Conversely, if all the viscosity were related to the number of active cross bridges, the slope would be proportional to the activation. Green (1969) has analyzed families of force–velocity curves to determine the relative contribution of each of these mechanisms.

A curve fit of the force–velocity curve was demonstrated by Fenn and March (1935) who used the equation

$$V = V_0 e^{P/B} - KP \tag{7.2}$$

where V = shortening velocity at any force
V_0 = shortening velocity of unloaded muscle
P = force
B, K = constants

A few years later Hill (1938) proposed a different mathematical relationship that bore some meaning with regard to the internal thermodynamics. Hill's curve was in the form of a hyperbola,

$$(P + a)(V + b) = (P_0 + a)b \tag{7.3}$$

where P_0 = maximum isometric tension
a = coefficient of shortening heat
$b = a \cdot V_0/P_0$
V_0 = maximum velocity (when $P = 0$)

More recently this hyperbolic form has been found to fit the empirical constant only during isotonic contractions near resting length.

The maximum velocity of shortening is often expressed as a function of l_0, the resting fiber length of the contractile elements of the muscle (reported in Table 3.2). Animal experiments have shown the maximum shortening velocity to be about 6 l_0/s (Faulkner et al., 1980; Close, 1965). However, it appears that this must be low for some human activity. Woittiez (1984) has calculated the shortening velocity in soleus, for example, to be above 10 l_0/s (based on plantarflexor velocity in excess of 8 rad/s and a moment arm length of 5 cm).

7.2.2 Eccentric Contractions

The vast majority of research done on isolated muscle during in vivo experiments has involved concentric contractions. As a result, there is relatively little knowledge about the details of the force–velocity curve as the muscle lengthens. The curve certainly does not follow the detailed mathematical relationships that have been developed for concentric contractions.

This lack of information about eccentric contractions is unfortunate because normal human movement usually involves as much eccentric as concentric activity. If we neglect air and ground friction, level walking involves equal amounts of positive and negative work, and in downhill gait negative work dominates. Figure 7.12 shows the general shape of the force–velocity curve during eccentric contractions. It can be seen that this curve is an extension of the concentric curve.

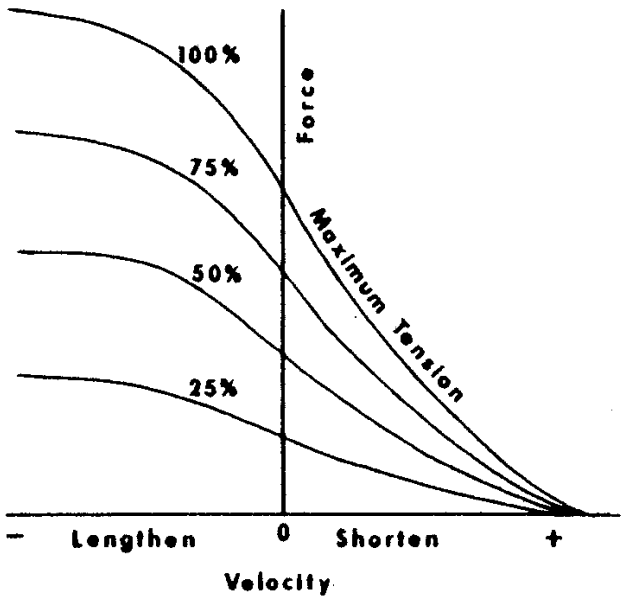


Figure 7.12 Force–velocity characteristics of skeletal muscle, showing a decrease of tension as the muscle shortens and an increase as it lengthens. All such characteristics must be taken as the muscle shortens or lengthens at a given length, and the length must be reported. A family of curves results if different levels of muscle activation are plotted; shown are those curves at 25, 50, 75, and 100% levels of activation.

Experimentally it is somewhat more difficult to conduct experiments involving eccentric work because an external device must be available to do the work on the human muscle. Such a requirement means that a motor is needed to provide an external force that will always exceed that of the muscle. Experiments on isolated muscle are safe to conduct, but in vivo experiments on humans are difficult because such a machine could cause lengthening even past the safe range of movement of a joint. The excessive force could tear the limb apart at the joint. Foolproof safety mechanisms would have to be installed to prevent such an occurrence.

The reasons given for the forces increasing as the velocity of lengthening increases are similar to those that account for the drop of tension during concentric contractions. First, within the contractile element it is understood that the force required to break the cross bridge protein links is greater than that required to hold it at its isometric length, and that this force increases as the rate of breaking increases. Second, the viscous friction of shortening is still very much present. However, because the direction of shortening has reversed, the tendon force must now be higher in order to overcome this internal damping friction. Independent of the exact cause of the force increase with velocity, it is conceptually safe to model the effect by a viscous damper.

7.2.3 Combination of Length and Velocity versus Force

In Section 7.1 and in this section it is evident that the tendon force is a function of both length and velocity. Therefore a proper representation of both

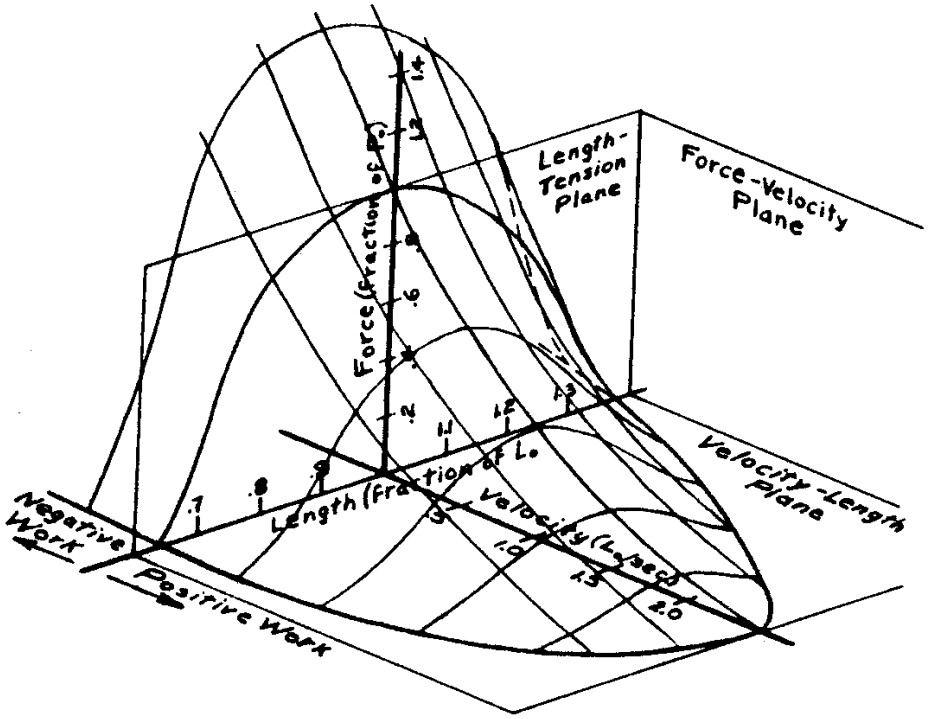


Figure 7.13 Three-dimensional plot showing the change in contractile element tension as a function of both velocity and length. Surface shown is for maximum muscle activation; a new "surface" will be needed to describe each level of activation. Influence of parallel elastic element is not shown.

these effects requires a three-dimensional plot like that shown in Figure 7.13. The resultant curve is actually a surface, which here is represented only for the maximum contraction condition. The more normal contractions are at a fraction of this maximum, so that surface plots would be required for each level of muscle activation, say at 75%, 50%, and 25%.

7.2.4 Combining Muscle Characteristics with Load Characteristics: Equilibrium

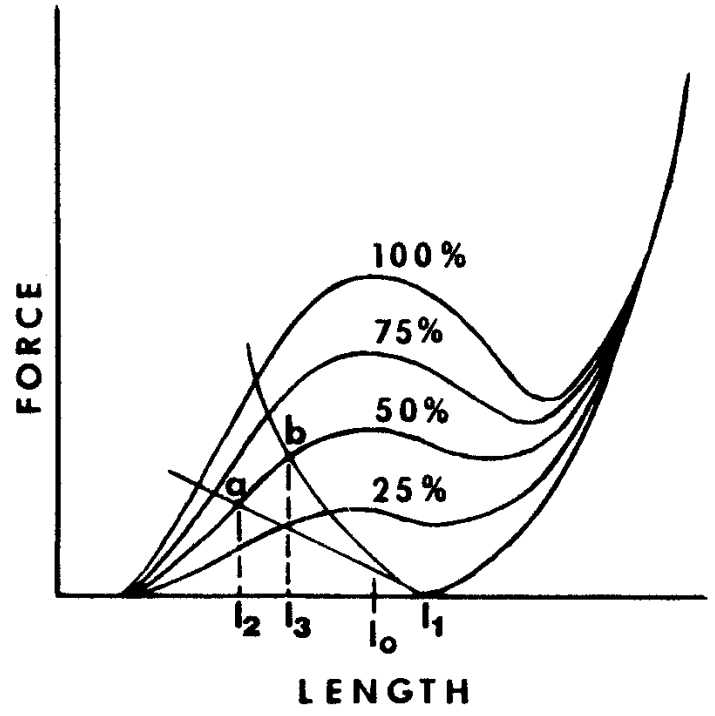
Muscles are the only motors in the human system, and when they are active, they must be in equilibrium with their load. The load can be static, such as holding a weight against gravity, or applying a static force against a fixed object (i.e., a wall, the floor), or in an isometric cocontraction where one muscle provides the load of the other. Or the load may be dynamic and the muscles are accelerating or decelerating an inertial load or overcoming the friction of a viscous load. In the vast majority of voluntary movement there is a mixture of static and dynamic load. The one condition that is satisfied during the entire movement is that of equilibrium: at any given point in time the operating point will be the intersection of the muscle and load characteristics. In a dynamic movement this operating or equilibrium point will be continuously changing. In Chapter 4 an equilibrium was recognized every time the equa-

tions of motion were written: Equation (4.3) and subsequent Examples 4.1, 4.2, 4.3, and 4.4. However, such equations recognized the net effect of all muscles acting at each joint and represented them as a torque motor without any concern about the internal characteristics of the muscles themselves. These are now examined.

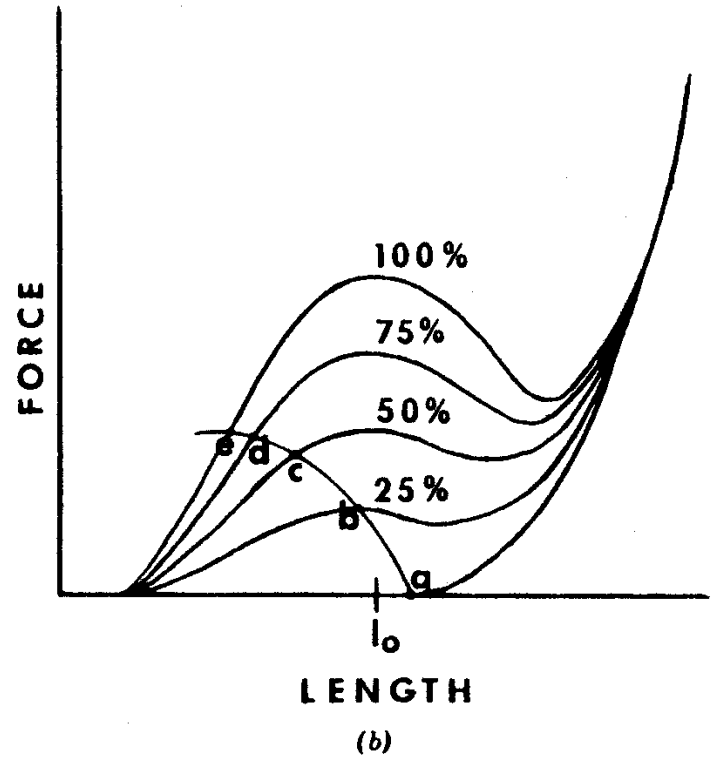
Consider a muscle that is contracting against a springlike load which can have a linear or a nonlinear force–length characteristic. Figure 7.14*a* plots the force–length characteristics of a muscle at four different levels of activation along with the linear and nonlinear characteristics of the spring loads. Assume that the muscle is at a length l_1 longer than the resting length, l_0 when the spring is at rest. The linear spring is compressed with a 50% muscle contraction, the equilibrium point *a* is reached, and the muscle will shorten from l_1 to l_2. When the nonlinear spring is compressed with a 50% contraction, the muscle shortens from l_1 to l_3 and the equilibrium point *b* is reached. If a gravitational load is lifted by the elbow flexors, the flexor muscles have the characteristics shown in Figure 7.14*b*. Starting with the forearm lowered to the side so that it is vertical, the initial equilibrium point is *a*. Then as the flexors contract and shorten, equilibrium points *b* through *e* are reached. Finally, as the muscles are activated 100%, the equilibrium point *e* is reached. During the transient conditions en route from *a* to *e* the equilibrium point will also move about on the force–velocity characteristics, tracing a three dimensional locus. The idea of dynamic equilibrium is now addressed.

We use the data from a typical walking stride to plot the time course of the contractions of the muscles about the ankle. Because the angular changes of the ankle are relatively small, we can consider that the muscle lengths are proportional to the ankle angle, and these length changes are also small. Thus we can plot the event on a two-dimensional force–velocity curve. The lengthening or shortening velocity can be considered proportional to the angular velocity and the tendon forces are considered proportional to the muscle moment. Thus using in vivo data, the traditional force–velocity curve becomes a moment–angular velocity curve. Figure 7.15 is the resultant plot for the period of time from heel contact to toe-off.

It can be seen from this time course of moment and angular velocity that this common movement is, in fact, quite complex. Contrary to what might be implied by force–velocity curves, a muscle does not operate along any simple curve, but actually goes through a complex combination of force and velocity changes. Initially the dorsiflexor muscles are on as the foot plantarflexes between the time of heel contact and flat foot (when the ground reaction force lies behind the ankle joint). Negative work is being done by the dorsiflexors as they lower the foot to the ground. After flat foot the plantarflexors dominate and create a moment that tends to slow down the leg as it rotates over the foot, which is now fixed on the floor. Again this is negative work. A few frames after heel-off positive work begins, as indicated by the simultaneous plantarflexor muscle moment and plantarflexor velocity. This period is the active push-off phase, when most of the "new" energy is put back into the body.



(*a*)



(*b*)

Figure 7.14 (*a*) Force–length characteristics of a muscle at four different levels of activation along with the characteristics of a linear and nonlinear spring. Intersection of spring (load) and muscle characteristics is the equilibrium point. (*b*) Equilibrium points of a load and elbow flexor muscle as the elbow is flexed against a gravitational load.

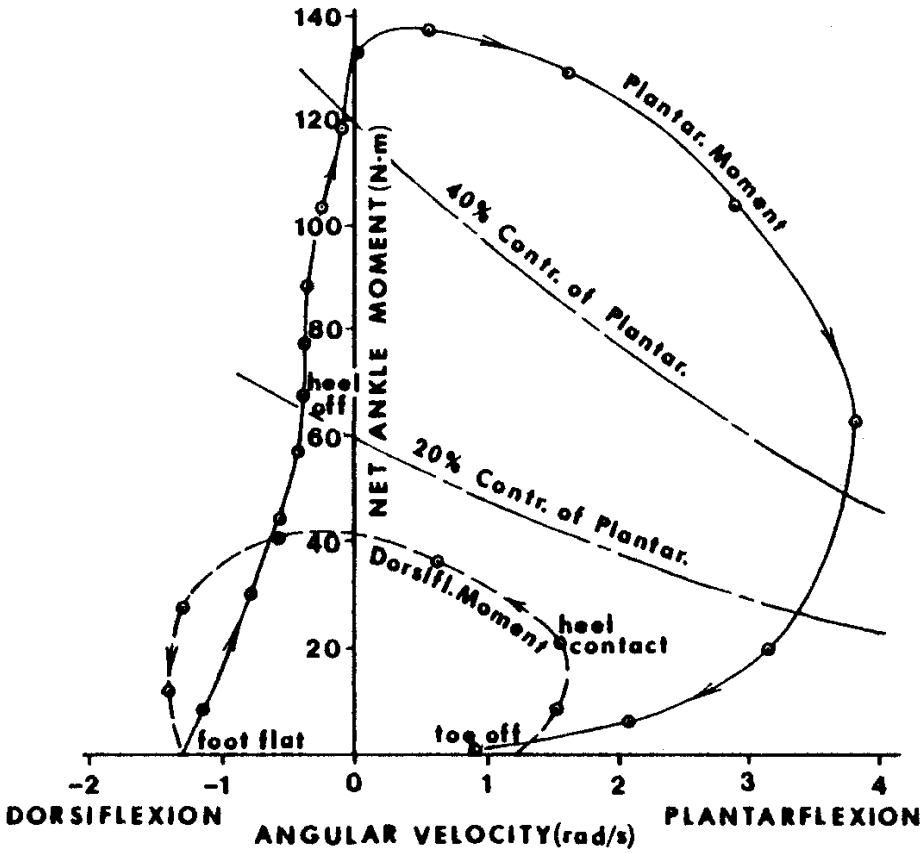


Figure 7.15 Time history of muscle moment–velocity during stance phase of walking. Dorsiflexor moment is shown as a dashed line; plantarflexor moment is a solid line. Stance begins with negative work, then alternating positive and negative, and finally a major positive work burst later in push-off, ending at toe-off.

7.3 MUSCLE MODELING

A variety of mechanical models of muscle have evolved to describe and predict tension, based on some input stimulation. Crowe (1970) and Gottlieb and Agarwal (1971) proposed a contractile component in conjunction with a linear series and a parallel elastic component plus a linear viscous damper. Glantz (1974) proposed nonlinear elastic components plus a linear viscous component. Winter (1976) has used a mass and a linear spring and damper system to simulate the second-order critically damped twitch. The purpose of this section is not to criticize or justify one model versus another, but rather to go over the principles behind the modeling and the components.

Figure 7.16 shows the force–displacement and force–velocity relationships of linear and nonlinear springs and dampers that have been proposed. The symbol for a viscous damper is a "piston in a cylinder," which can be considered full of a fluid of suitable viscosity represented by the constant K. The more common nonlinear models are exponential in form or to a power a, which is usually greater than 1. This is especially true of viscous friction, which often varies approximately as the velocity squared.

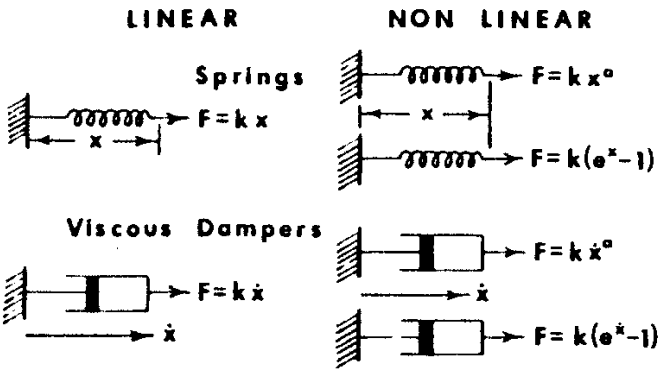


Figure 7.16 Schematic diagram of linear and nonlinear spring and viscous damper elements used to represent passive viscoelastic characteristics of muscle.

The total model of the passive components can take many configurations, as shown in Figure 7.17*a*. The parallel elastic component can be considered to be in parallel with the damper or a series of combinations of the damper and the series elastic component. For linear components it does not make the slightest difference which configuration is used, because they can be made equivalent. Fung (1971) has shown that

$$k_1 = k_3 + k_4, \qquad \frac{k_1 k_2}{k_1 + k_2} = k_3, \qquad \frac{b_1}{k_1 + k_2} = \frac{b_2}{k_4} \tag{7.4}$$

This means that if either model is known, the other equivalent model can replace it, and it will have the same dynamic characteristics.

The total model requires the active contractile component to be represented by some form of force generator. The time course of the tension from the contractile component is sometimes referred to as its *active state,* and

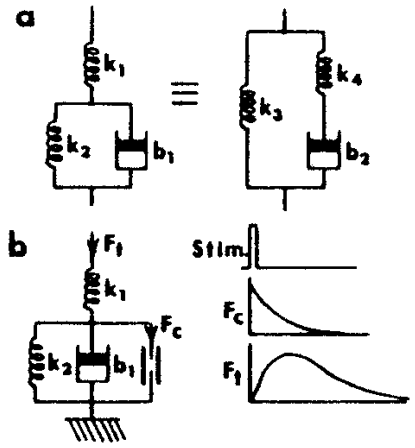


Figure 7.17 (*a*) Two equivalent series/parallel arrangements of linear elements of a muscle model. (*b*) Model showing contractile element acting on viscous elastic elements. Twitch tension of the tendon F_t results only if we assume an exponential activation tension from the contractile element F_c.

this is quite often assumed to be an exponential response to a stimulus. Figure 7.17*b* shows the contractile component combined with the passive components along with the time course of the active state F_c and the resultant tendon tension F_t. More complex models have been developed, one of which is presented in the next section.

7.3.1 Example Model—EMG Driven

Any realistic model of muscle must have a valid input to represent the motoneuron drive. It would be desirable to record the output of the motoneuron pool (summation of all recruited motor units) to any given muscle. Unfortunately this is impossible to do experimentally, so the best compromise is to record the EMG from the muscle. Students not familiar with the recording and interpretation of the EMG should read Chapter 8 before reviewing this example of muscle modeling.

Surface EMG has been shown to be more reliable than the EMG recorded from indwelling electrodes (Komi and Buskirk, 1970), and the number of motor units in the pick-up zone for surface electrodes is considerably larger than that for indwelling electrodes. These advantages combined with the ease of application make the surface EMG a valid signal to represent the average motor unit activity of most superficial muscles.

When each motor unit contracts, it results in a characteristic action potential and also the generation of an impulse of force by the activated muscle fibers. There is controversy regarding the shape of the tension waveform generated at the cross bridges versus the twitch waveform seen at the tendon. Some researchers assume the internal force waveform (sometimes called *active state*) to be a first-order exponential (Hatze, 1978; Gottlieb and Agarwal, 1971). However, research on muscles (Hill, 1953) showed that the time course of the biochemical thermal events was impulsive in nature. Using frog muscle at 0°C, the rate of heat production reaches a maximum 30 ms after stimulation. If we recall that reaction rates increase 2–3 times every 10°C, we could predict an increase of 15–40 in the reaction rates at muscle temperatures of 37°C. Therefore the rate of heat production (indicating the release of chemical energy at the cross bridges) would be over in less than 2 ms. Thus the associated mechanical energy release must also be assumed to be a short-duration impulse. Fortunately larger motor units also generate larger action potentials. Thus the absolute value of the EMG is a series of short-duration impulses which can be considered to represent the contractile element's impulsive force. Thus a full-wave rectified EMG followed by a nonlinearity (to model any nonlinearity between the EMG and muscle force) is used to represent the relationship between the EMG and the contractile element force.

Figure 7.18 shows the complete model, which incorporates the contractile element, a linear damper B to model any viscous or velocity-dependent elements (in the contractile element and connective tissue), a linear spring K to

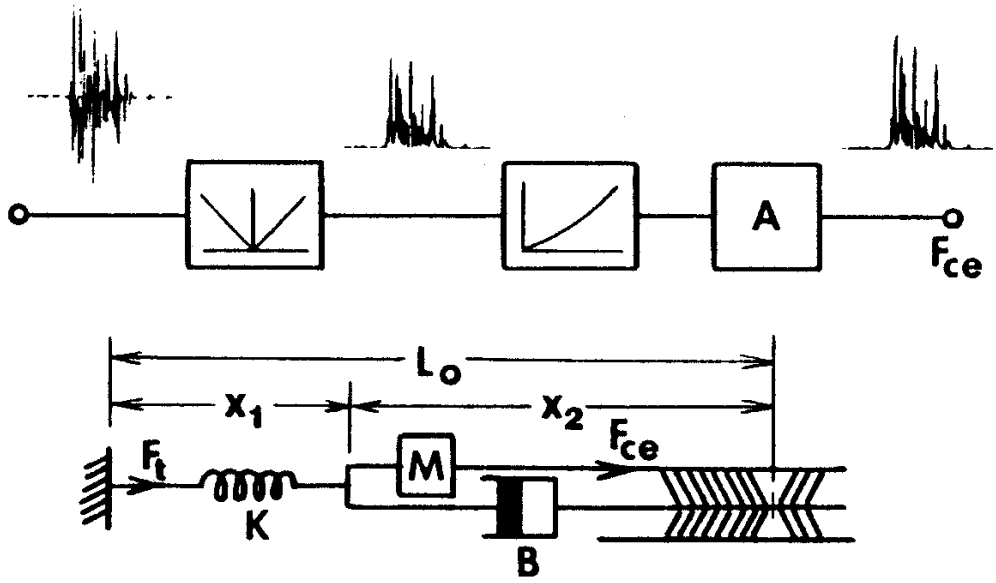


Figure 7.18 Biomechanical model of a muscle during an isometric contraction of varying tension. F_{ce} is modeled as an impulsive contractile element force acting on an equivalent mass M, a linear damping element B, and a series elastic element K. F_{ce} as input to the model is assumed to be the same shape as the full-wave rectified EMG with an empirically curve-fitted nonlinearity. See text for full details and justification for the assumptions.

represent the series elastic elements (in the tendon, fascia, and cross bridges), and a mass element M to represent the effective mass of muscle tissue that must be accelerated as the force impulse wave travels from the motor end plate region toward both tendons. For an isometric contraction the length L_0 of the muscle remains constant, but x_2 shortens, causing a lengthening of x_1 in the muscle's series elastic element. The impulsive contractile element force F_{ce}, acts on the mass to accelerate it, but to do so the mass acts on the damper and spring elements. Thus the equations of motion are

$$x_1 + x_2 = L_0 = \text{constant}$$

$$F_{ce} = M\ddot{x}_1 + B\dot{x}_1 + Kx_1 \qquad (7.5a)$$

The tendon force F_t is seen only if the series elastic spring increases its length beyond resting length or,

$$F_t = Kx_1 \qquad (7.5b)$$

In analog or digital form this second-order differential equation can be solved for x_1 (Figure 7.19) to predict the tendon force for any given EMG. The final part of the model is to determine K, B, and M for the muscle. Fortunately we do not have to measure them separately. Rather, we can make use of the fact that the twitch waveform is quite close to that of a critically damped second-order system (Milner-Brown et al., 1973*a*). The duration of

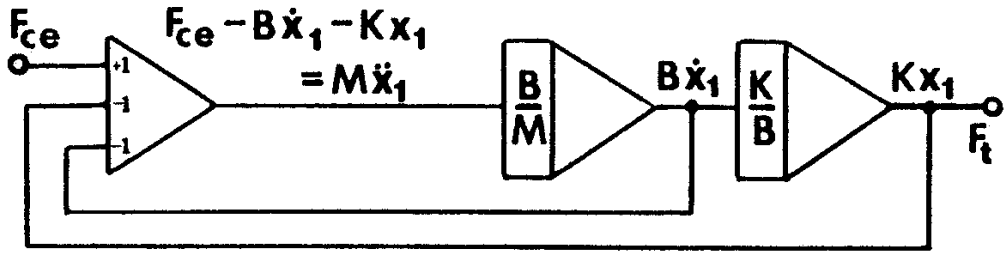
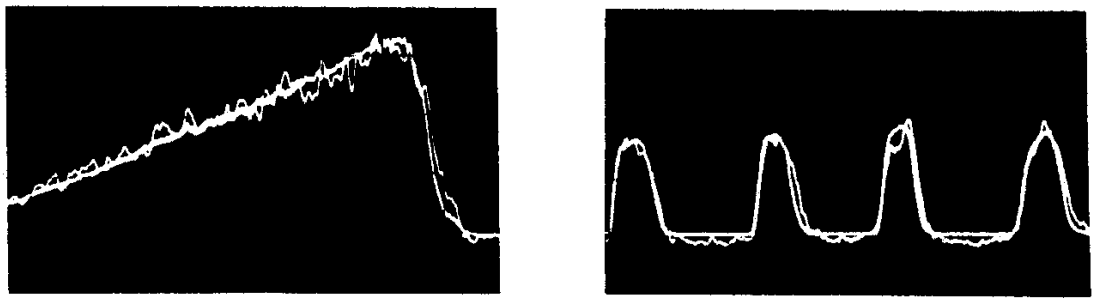


Figure 7.19 Analog or digital solution to solve for tendon force F_t for any given F_{ce} and for any muscle. B/M and K/B ratios are known if the twitch time T for that muscle is known. See text for complete details.

the twitches is sufficiently long, such that, in comparison, the motor unit action potential can be considered as an impulse. For a mass–spring–damper system that is critically damped the twitch time T (see Section 7.0.5) allows us to calculate the correct ratios, $T = 2M/B$ or $B/M = 2/T$. Also, $B = 2\sqrt{MK}$. Therefore $K/B = B/4M = 1/2T$. Therefore the differential equation as modeled in Figure 7.19 can be solved by knowing the values for B/M and K/B, which are automatically known if we know T for any given muscle. The only remaining parameter that is needed to curve-fit the model is a "gain" A to model the relationship between the EMG (in microvolts) and the contractile element tension F_{ce} (in newtons).

Figure 7.20 shows the results of comparisons between the predicted muscle tension F_t and that measured experimentally. The biceps muscle EMG was recorded along with elbow flexor tension from a force transducer attached firmly to the wrist via a cuff. The smoother curve is the transducer output, while the noisier signal is the predicted F_t. The difference between measured and predicted curves is minimal and can be partially attributed to several measurement and assumption errors. First, we are not measuring the tendon tension directly, but via a transducer attached to the wrist. The recorded tension will be further damped by the cuff/wrist tissue and in the synovial fluid and soft tissue in the elbow joint capsule. Second, certain unstated assumptions are inferred in this model. Other agonists (brachioradialis, brachialis)



(a) (b)

Figure 7.20 Oscilloscope traces of predicted F_t superimposed on that measured by a force transducer during isometric contractions of the biceps muscle. (a) During a ramp increase of tension. Noisier trace is F_t, smoother trace is the force transducer record. (b) Same model during rapid short bursts of force.

are assumed to have exactly the same activation profile as the biceps, and any antagonists (triceps) are assumed to be silent.

7.4 REFERENCES

Bahler, A.S. "Series Elastic Component of Mammalian Muscle," *Am. J. Physiol.* **213**:1560–1564, 1967.

Bellemare, F., J.J. Woods, R. Johansson, and B. Bigland-Ritchie. "Motor Unit Discharge Rates in Maximal Voluntary Contractions in Three Human Muscles," *J. Neurophysiol.* **50**:1380–1392, 1983.

Buchthal, F., and H. Schmalbruch. "Contraction Times and Fibre Types in Intact Human Muscle," *Acta Physiol. Scand.* **79**:435–452, 1970.

Close, R. "The Relation between Intrinsic Speed of Shortening and Duration of the Active State of Muscles," *J. Physiol.* **180**:542–559, 1965.

Crowe, A. "A Mechanical Model of Muscle and Its Application to the Intrafusal Fibres of Mammalian Muscle Spindle," *J. Biomech.* **3**:583–592, 1970.

DeLuca, C.J., R.S. LeFever, M.P. McCue, and A.P. Xenakis. "Control Scheme Governing Concurrently Active Motor Units during Voluntary Contractions," *J. Physiol.* **329**:129–142, 1982.

Desmedt, J.E., and E. Godaux. "Ballistic Contractions in Fast and Slow Human Muscles: Discharge Patterns of Single Motor Units," *J. Physiol.* **285**:185–196, 1978.

Faulkner, J.A., J.H. Niemeyer, L.C. Maxwell, and T.P. White. "Contractile Properties of Transplanted Extensor Digitorum Longus Muscle of Cats," *Am. J. Physiol.* **238**:120–126, 1980.

Feinstein, B., B. Lindegard, E. Nyman, and G. Wohlfart. "Morphological Studies of Motor Units in Normal Human Muscles," *Acta Anat.* **23**:127–142, 1955.

Fenn, W.O., and B.S. Marsh. "Muscular Force at Different Speeds of Shortening," *J. Physiol. London* **85**:277–297, 1935.

Fung, Y.C. "Comparison of Different Models of the Heart Muscle," *J. Biomech.* **4**:289–295, 1971.

Glantz, S.A. "A Constitutive Equation for the Passive Properties of Muscle," *J. Biomech.* **7**:137–145, 1974.

Gordon, A.M., A.F. Huxley, and F.J. Julian. "The Variation Is Isometric Tension with Sarcomere Length in Vertebrate Muscle Fibres," *J. Physiol.* **184**:170, 1966.

Gottlieb, G.L., and G.C. Agarwal. "Dynamic Relationship between Isometric Muscle Tension and the Electromyogram in Man," *J. Appl. Physiol.* **30**:345–351, 1971.

Green, D.G. "A Note on Modelling in Physiological Regulators," *Med. Biol. Eng.* **7**:41–47, 1969.

Hatze, H. "A General Myocybernetic Control Model of Skeletal Muscle," *Biol. Cybern.* **28**:143–157, 1978.

Henneman, E. "Organization of the Spinal Cord," in *Medical Physiology,* vol. 1, 13th ed., V.B. Mountcastle, Ed. (C.V. Mosby, St. Louis, MO, 1974*a*).

Henneman, E. "Peripheral Mechanism Involved in the Control of Muscle," in *Medical Physiology,* vol. 1, 13th ed., V.B. Montcastle, Ed. (C.V. Mosby, St. Louis, MO, 1974*b*).

Hill, A.V. "The Heat of Shortening and Dynamic Constants of Muscle," *Proc. R. Soc. B.* **126**:136–195, 1938.

Hill, A.V. "Chemical Change and Mechanical Response in Stimulated Muscle," *Proc. R. Soc. B.* **141**:314–320, 1953.

Komi, P.V., and E. R. Buskirk. "Reproducibility of Electromyographic Measures with Inserted Wire Electrodes and Surface Electrodes," *Electromyography* **10**:357–367, 1970.

Milner-Brown, H. S., R. B. Stein, and R. Yemm. "The Contractile Properties of Human Motor Units during Voluntary Isometric Contractions," *J. Physiol.* **228**:285–306, 1973*a*.

Milner-Brown, H. S., R. B. Stein, and R. Yemm. "The Orderly Recruitment of Human Motor Units during Voluntary Isometric Contractions," *J. Physiol.* **230**:359–370, 1973*b*.

Milner-Brown, H. S., and R. B. Stein. "The Relation between the Surface Electromyogram and Muscular Force," *J. Physiol.* **246**:549–569, 1975.

Sale, D., J. Quinlan, E. Marsh, A. J. McComas, and A.Y. Belanger. Influence of Joint Position on Ankle Plantarflexion in Humans, *J. Appl. Physiol.* **52**:1636–1642, 1982.

van Ingen Schenau, G. J. "An Alternate View of the Concept of Utilization of Elastic Energy in Human Movement," *Human Movement Sci.* **3**:301–336, 1984.

Wani, A. M., and S. K. Guha. "A Model for Gradation of Tension Recruitment and Rate Coding," *Med. Biol. Eng.* **13**:870–875, 1975.

Winter, D. A. "Biomechanical Model Related EMG to Changing Isometric Tension," in *Dig. 11th Int. Conf. Med. Biol. Eng.*, pp. 362–363, 1976.

Woittiez, R. G. "A Quantitative Study of Muscle Architecture and Muscle Function," Ph.D. dissertation, Free University, (Amsterdam), The Netherlands, 1984.

Wormolts, J. R., and W. K. Engel. "Correlation of Motor Unit Behaviour with Histochemical-Myofiber Type in Humans by Open-Biopsy Electromyography," in *New Developments in Electromyography and Clinical Neurophysiology,* vol. 1, J. E. Desmedt, Ed. (Karger, Basel, Switzerland, 1973).