

Biomechanics of Skeletal Muscle

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SUMMARY62

Skeletal muscle is a fascinating biological tissue able to transform chemical energy to mechanical energy. The focus of this chapter is on the mechanical behavior of skeletal muscle as it contributes to function and dysfunction of the musculoskeletal system. Although a basic understanding of the energy transformation from chemical to mechanical energy is essential to a full understanding of the behavior of muscle, it is beyond the scope of this book. The reader is urged to consult other sources for a discussion of the chemical and physiological interactions that produce and affect a muscle contraction [36,47,78].

Skeletal muscle has three basic performance parameters that describe its function:

- Movement production
- Force production
- Endurance

The production of movement and force is the mechanical outcome of skeletal muscle contraction. The factors that influence these parameters are the focus of this chapter. A brief description of the morphology of muscles and the physiological processes that produce

contraction needed to understand these mechanical parameters is also presented here. Specifically the purposes of this chapter are to

- Review briefly the structure of muscle and the mechanism of skeletal muscle contraction
- Examine the factors that influence a muscle's ability to produce a motion
- Examine the factors that influence a muscle's ability to produce force
- Consider how muscle architecture is specialized to optimize a muscle's ability to produce force or joint motion
- Demonstrate how an understanding of these factors can be used clinically to optimize a patient's performance
- Discuss the adaptations that muscle undergoes with prolonged changes in length and activity

STRUCTURE OF SKELETAL MUSCLE

The functional unit that produces motion at a joint consists of two discrete units, the muscle belly and the tendon that binds the muscle belly to the bone. The structure of the muscle belly itself is presented in the current chapter. The structure and mechanical properties of the tendon, composed of connective tissue, are presented in Chapter 6. The muscle belly consists of the muscle cells, or fibers, that produce the contraction and the connective tissue encasing the muscle fibers. Each is discussed below.

Structure of an Individual Muscle Fiber

A skeletal muscle fiber is a long cylindrical, multinucleated cell that is filled with smaller units of filaments (Fig. 4.1). These filamentous structures are roughly aligned parallel to the muscle fiber itself. The largest of the filaments is the myofibril, composed of subunits called *sarcomeres* that are arranged end to end the length of the myofibril. Each sarcomere also contains filaments, known as *myofilaments*. There are two types of myofilaments within each sarcomere. The thicker myofilaments are composed of myosin protein molecules, and the thinner myofilaments are composed of molecules of the protein actin. Sliding of the actin myofilament on the myosin chain is the basic mechanism of muscle contraction.

THE SLIDING FILAMENT THEORY OF MUSCLE CONTRACTION

The sarcomere, containing the contractile proteins actin and myosin, is the basic functional unit of muscle. Contraction of

a whole muscle is actually the sum of singular contraction events occurring within the individual sarcomeres. Therefore, it is necessary to understand the organization of the sarcomere. The thinner actin chains are more abundant than the myosin myofilaments in a sarcomere. The actin myofilaments are anchored at both ends of the sarcomere at the Z-line and project into the interior of the sarcomere where they surround a thicker myosin myofilament (Fig. 4.2). This arrangement of myosin myofilaments surrounded by actin myofilaments is repeated throughout the sarcomere, filling its interior and giving the muscle fiber its characteristic striations. The amount of these contractile proteins within the cells is strongly related to a muscle's contractile force [5,6,24].

Contraction results from the formation of cross-bridges between the myosin and actin myofilaments, causing the actin chains to "slide" on the myosin chain (Fig. 4.3). The tension of the contraction depends upon the number of cross-bridges formed between the actin and myosin myofilaments. The number of cross-bridges formed depends not only on the abundance of the actin and myosin molecules, but also on the frequency of the stimulus to form cross-bridges.

Contraction is initiated by an electrical stimulus from the associated motor neuron causing depolarization of the muscle fiber. When the fiber is depolarized, calcium is released into the cell and binds with the regulating protein troponin. The combination of calcium with troponin acts as a trigger, causing actin to bind with myosin, beginning the contraction. Cessation of the nerve's stimulus causes a reduction in calcium levels within the muscle fiber, inhibiting the cross-bridges between actin and myosin. The muscle relaxes [78]. If stimulation of the muscle fiber occurs at a sufficiently high frequency, new cross-bridges are formed before prior interactions are completely severed, causing a fusion of succeeding contractions. Ultimately a sustained, or

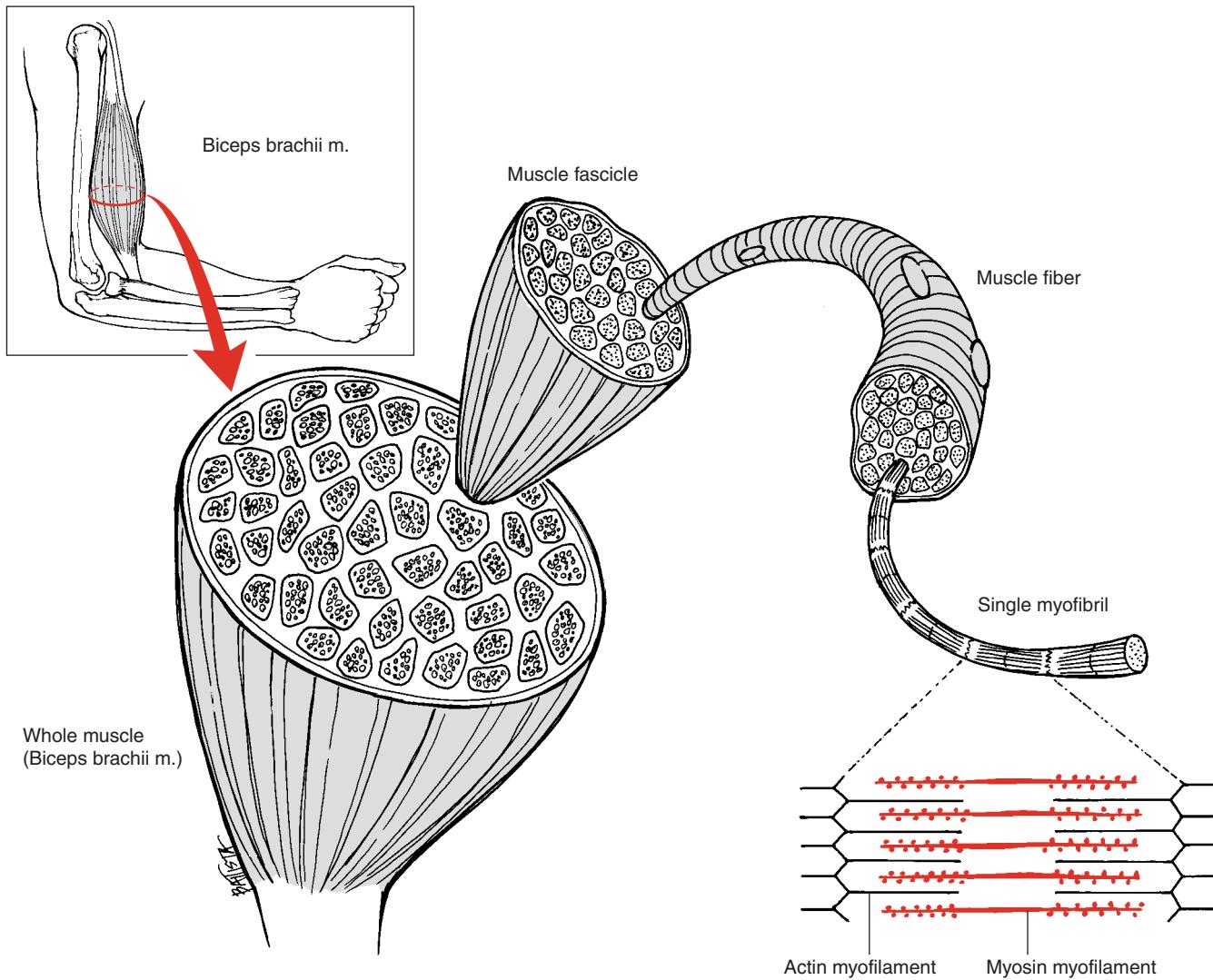


Figure 4.1: Organization of muscle. A progressively magnified view of a whole muscle demonstrates the organization of the filaments composing the muscle.

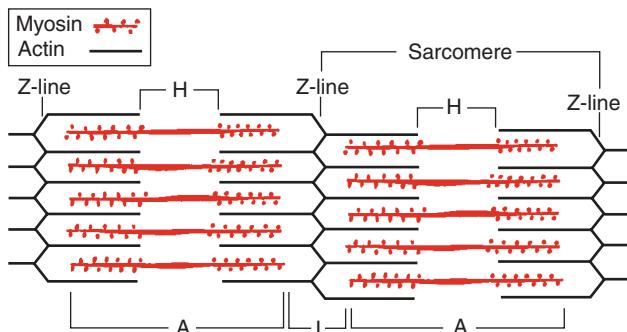


Figure 4.2: Organization of actin and myosin within a muscle fiber. The arrangement of the actin and myosin chains in two adjacent sarcomeres within a fiber produces the characteristic striations of skeletal muscle.

tetanic, contraction is produced. Modulation of the frequency and magnitude of the initial stimulus has an effect on the force of contraction of a whole muscle and is discussed later in this chapter.

The Connective Tissue System within the Muscle Belly

The muscle belly consists of the muscle cells, or fibers, and the connective tissue that binds the cells together (Fig. 4.4). The outermost layer of connective tissue that surrounds the entire muscle belly is known as the *epimysium*. The muscle belly is divided into smaller bundles or fascicles by additional connective tissue known as *perimysium*. Finally individual fibers within these larger sheaths are surrounded by more

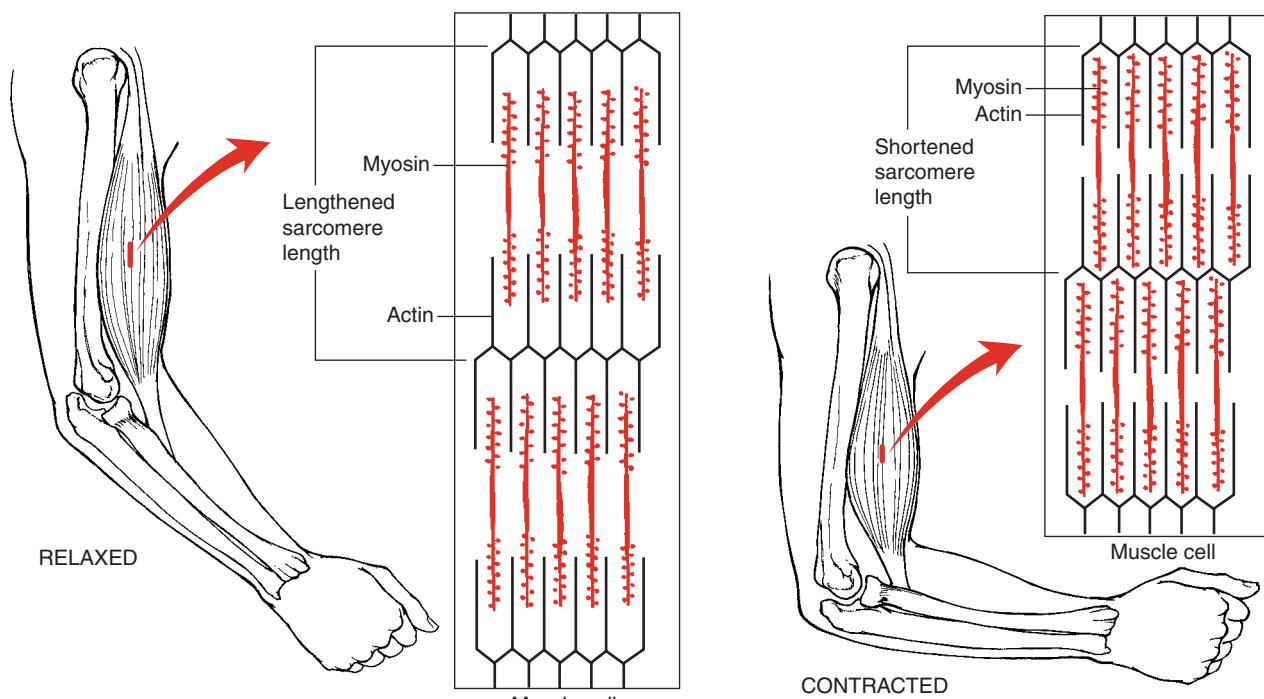


Figure 4.3: The sliding filament model. Contraction of skeletal muscle results from the sliding of the actin chains on the myosin chains.

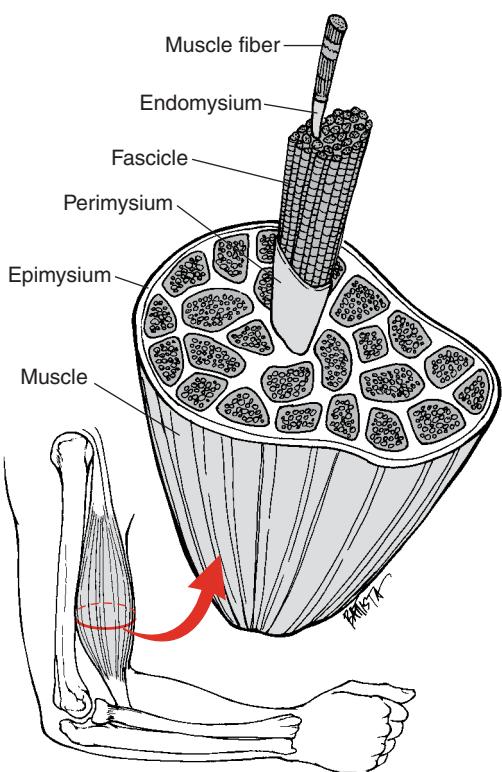


Figure 4.4: Organization of the connective tissue within muscle. The whole muscle belly is invested in an organized system of connective tissue. It consists of the epimysium surrounding the whole belly, the perimysium encasing smaller bundles of muscle fibers, and the endomysium that covers individual muscle fibers.

connective tissue, the endomysium. Thus the entire muscle belly is invested in a large network of connective tissue that then is bound to the connective tissue tendons at either end of the muscle. The amount of connective tissue within a muscle and the size of the connecting tendons vary widely from muscle to muscle. The amount of connective tissue found within an individual muscle influences the mechanical properties of that muscle and helps explain the varied mechanical responses of individual muscles. The contribution of the connective tissue to a muscle's behavior is discussed later in this chapter.

FACTORS THAT INFLUENCE A MUSCLE'S ABILITY TO PRODUCE A MOTION

An essential function of muscle is to produce joint movement. The passive range of motion (ROM) available at a joint depends on the shape of the articular surfaces as well as on the surrounding soft tissues. However the joint's active ROM depends on a muscle's ability to pull the limb through a joint's available ROM. Under normal conditions, active ROM is approximately equal to a joint's passive ROM. However there is a wide variation in the amount of passive motion available at joints throughout the body. The knee joint is capable of flexing through an arc of approximately 140°, but the metacarpophalangeal (MCP) joint of the thumb usually is capable of no more than about 90° of flexion. Joints that exhibit large ROMs require muscles capable of moving the

joint through the entire range. However such muscles are unnecessary at joints with smaller excursions. Thus muscles exhibit structural specializations that influence the magnitude of the excursion that is produced by a contraction. These specializations are

- The length of the fibers composing the muscle
- The length of the muscle's moment arm.

How each of these characteristics affects active motion of a joint is discussed below.

Effect of Fiber Length on Joint Excursion

Fiber length has a significant influence on the magnitude of the joint motion that results from a muscle contraction. The fundamental behavior of muscle is shortening, and it is this shortening that produces joint motion. The myofilaments in each sarcomere are 1 to 2 μm long; the myosin myofilaments are longer than the actin myofilaments [112,133]. Thus sarcomeres in humans are a few micrometers in length, varying from approximately 1.25 to 4.5 μm with muscle stretch and contraction [82–84,127]. Each sarcomere can shorten to approximately the length of its myosin molecules. Because

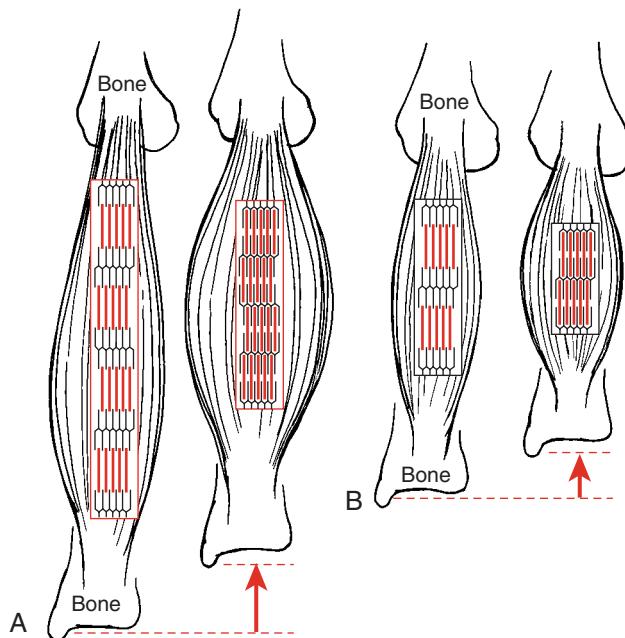


Figure 4.5: The relationship between fiber length and shortening capacity of the whole muscle. A muscle with more sarcomeres in series (**A**) can shorten more than a fiber with fewer sarcomeres in series (**B**).

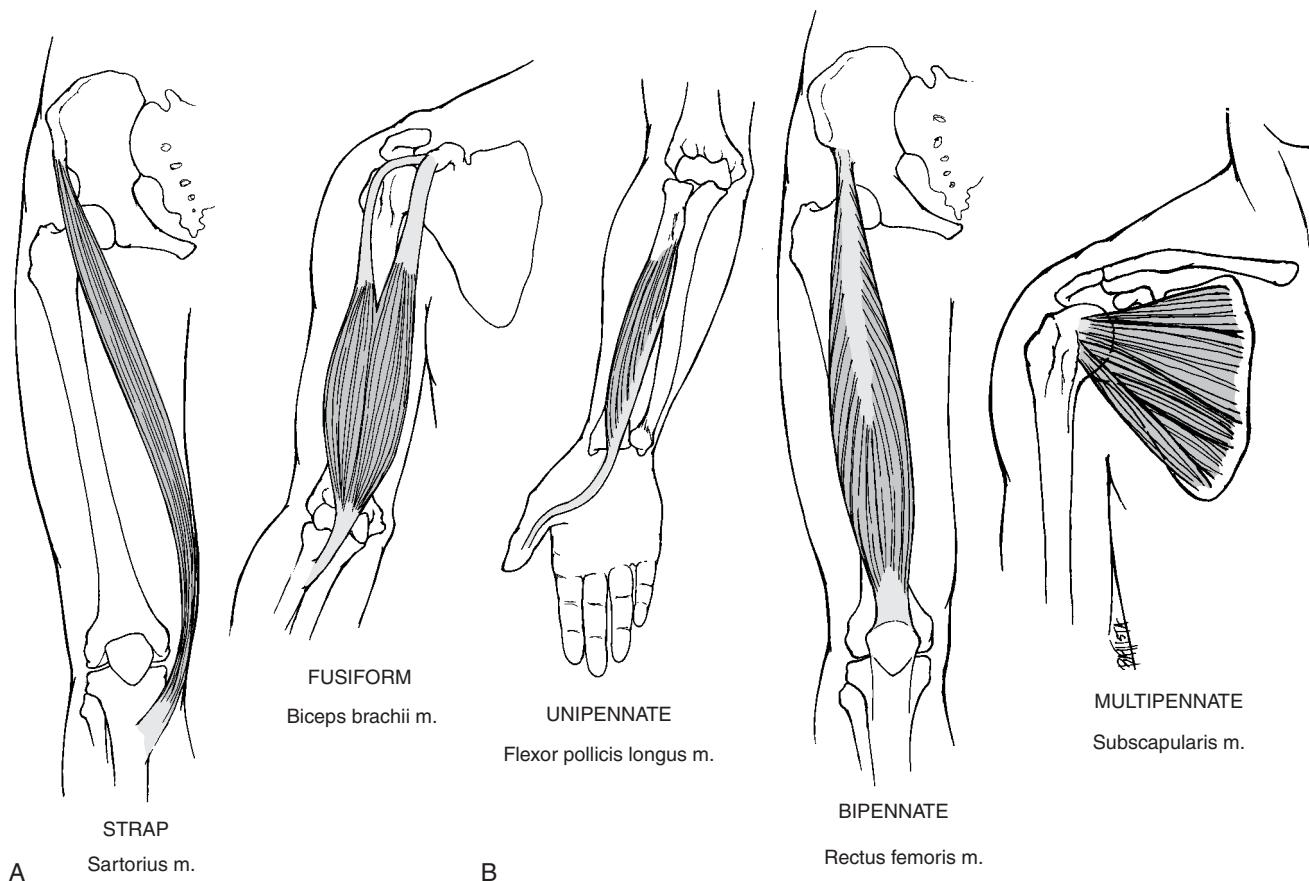


Figure 4.6: Muscle architecture. **A.** Muscles with parallel fibers include fusiform (biceps brachii) and strap (sartorius) muscles. **B.** Pennate muscles include unipennate (flexor pollicis longus), bipennate (rectus femoris), and multipennate (subscapularis).

the sarcomeres are arranged in series in a myofibril, the amount of shortening that a myofibril and, ultimately, a muscle fiber can produce is the sum of the shortening in all of the sarcomeres. Thus the total shortening of a muscle fiber depends upon the number of sarcomeres arranged in series within each myofibril. The more sarcomeres in a fiber, the longer the fiber is and the more it is able to shorten (Fig. 4.5). The amount a muscle fiber can shorten is proportional to its length [13,81,138]. A fiber can shorten roughly 50 to 60% of its length [39,138] although there is some evidence that fibers exhibit varied shortening capabilities [13].

Since fiber shortening is a function of fiber length, the amount a whole muscle can shorten is dictated by the length of its constituent fibers. An individual whole muscle is composed mostly of fibers of similar lengths [13]. However there is a wide variation in fiber lengths found in the human body, ranging from a few centimeters to approximately half a meter [78,130]. The length of the fibers within a muscle is a function of the architecture of that muscle rather than of the muscle's total length. The following describes how fiber length and muscle architecture are related.

ARCHITECTURE OF SKELETAL MUSCLE

Although all skeletal muscle is composed of muscle fibers, the arrangement of those fibers can vary significantly among muscles. This fiber arrangement has marked effects on a muscle's ability to produce movement and to generate force. Fiber arrangements have different names but fall into two major categories, **parallel** and **pennate** [37] (Fig. 4.6). In general, the fibers within a parallel fiber muscle are approximately parallel to the length of the whole muscle. These muscles can be classified as either **fusiform** or **strap** muscles. Fusiform muscles have tendons at both ends of the muscle so that the muscle fibers taper to insert into the tendons. Strap muscles have less prominent tendons, and therefore their fibers taper less at both ends of the whole muscle. Parallel fiber muscles are composed of relatively long fibers, although these fibers still are shorter than the whole muscle. Even the sartorius muscle, a classic strap muscle, contains fibers that are only about 90% of its total length.

In contrast, a pennate muscle has one or more tendons that extend most of the length of the whole muscle. Fibers run obliquely to insert into these tendons. Pennate muscles fall into subcategories according to the number of tendons penetrating the muscle. There are **unipennate**, **bipennate**, and **multipennate** muscles. A comparison of two muscles of similar total length, one with parallel fibers and the other with a pennate arrangement, helps to illustrate the effect of fiber arrangement on fiber length (Fig. 4.7). The muscle with parallel fibers has longer fibers than those found in the pennate muscle. Because the amount of shortening that a muscle can undergo depends on the length of its fibers, the muscle with parallel fibers is able to shorten more than the pennate muscle. If fiber length alone affected joint excursion, the muscle with parallel fibers would produce a larger joint excursion

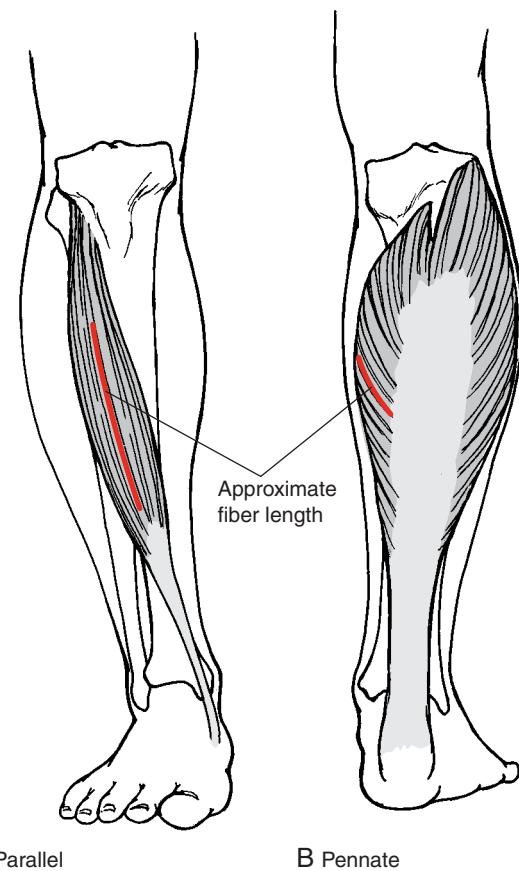


Figure 4.7: The relationship between muscle architecture and muscle fiber length. The fibers in a muscle with parallel fibers are typically longer than the fibers in a muscle of similar overall size but with pennate fibers.

than the muscle composed of pennate fibers [82]. However, a muscle's ability to move a limb through an excursion also depends on the length of the muscle's moment arm. Its effect is described below.

Effect of Muscle Moment Arms on Joint Excursion

Chapter 1 defines the moment arm of a muscle as the perpendicular distance between the muscle and the point of rotation. This moment arm depends on the location of the muscle's attachment on the bone and on the angle formed by the line of pull of the muscle and the limb to which the muscle attaches. This angle is known as the **angle of application** (Fig. 4.8). The location of an individual muscle's attachment on the bone is relatively constant across the population. Therefore, the distance along the bone between the muscle's attachment and the center of rotation of the joint can be estimated roughly by anyone with a knowledge of anatomy and can be measured precisely as well [51,73,87,135]. This distance is related to the true moment arm by the sine of the angle of application, θ , which can also be estimated or measured directly.

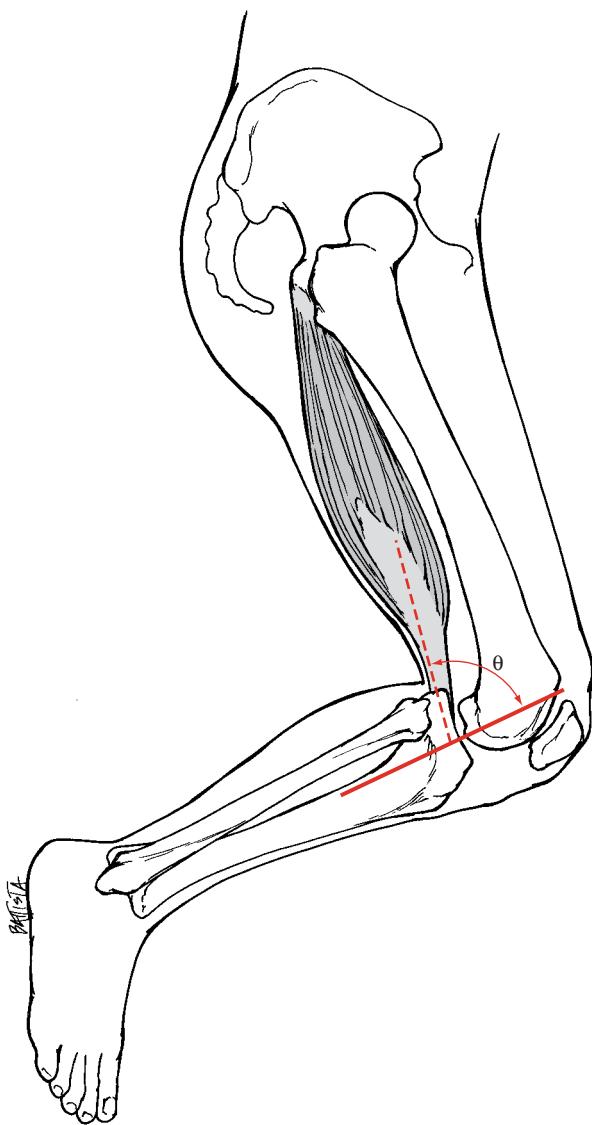


Figure 4.8: Angle of application. A muscle's angle of application is the angle formed between the line of pull of the muscle and the bone to which the muscle attaches.

A muscle's moment arm has a significant effect on the joint excursion produced by a contraction of the muscle. A muscle with a short moment arm produces a larger angular excursion than another muscle with a similar shortening capacity but with a longer moment arm. Principles of basic geometry help explain the relationship between muscle moment arms and angular excursion. Given two circles of different sizes, an angle, θ , defines an arc on each circle (Fig. 4.9). However, the arc of the larger circle is larger than the arc of the smaller circle. Thus the distance traveled on the larger circle to move through the angle θ is greater than that on the smaller circle. Similarly, a muscle with a long moment arm must shorten more to produce the same angular displacement as a muscle with a short moment arm [68,69].

Joint Excursion as a Function of Both Fiber Length and the Anatomical Moment Arm of a Muscle

The preceding discussion reveals that both a muscle's fiber length and its moment arm have direct effects on the amount of excursion a muscle contraction produces. These effects can be summarized by the following:

- Because muscle fibers possess a similar relative shortening capability, longer fibers produce more absolute shortening than shorter fibers.
- Because muscles with parallel fibers generally have longer fibers than pennate muscles, whole muscles composed of parallel fibers have a larger shortening capacity than whole muscles of similar length composed of pennate fibers.
- Muscles with shorter anatomical moment arms are capable of producing greater angular excursions of a joint than muscles of similar fiber length with larger anatomical moment arms.

It is interesting to see how these characteristics are combined in individual muscles. Muscles combine these seemingly opposing attributes in various ways, resulting in diverse functional capacities. It appears that some muscles, like the gluteus maximus, possess both long fibers and relatively short moment arms. Such muscles are capable of producing relatively large joint excursions [55]. Others, like the brachioradialis muscle at the elbow, combine relatively long muscle fibers with large moment arms [81]. The long fibers enhance the muscle's ability to produce a large excursion. However, the large moment arm decreases the muscle's ability to produce a large excursion. This apparent contradiction is explained in part by the recognition that the factors that influence production of movement, muscle architecture and anatomical moment arm, also influence force production capabilities in a muscle. Muscles must find ways to balance the competing demands of force production and joint excursion.

CLINICAL RELEVANCE: CONSIDERATIONS REGARDING TENDON TRANSFERS

Muscle fiber arrangement and muscle moment arms are inherent characteristics of a muscle and normally change very little with exercise or functional use. However, surgeons commonly transfer a muscle or muscles to replace the function of paralyzed muscles [13,14]. Successful restoration of function requires that the surgeon not only replace the nonfunctioning muscle with a functional muscle but also must ensure that the replacement muscle has an excursion-generating capacity similar to that of the original muscle. This may be accomplished by choosing a structurally similar muscle or by surgically manipulating the moment arm to increase or decrease the excursion capability [138].

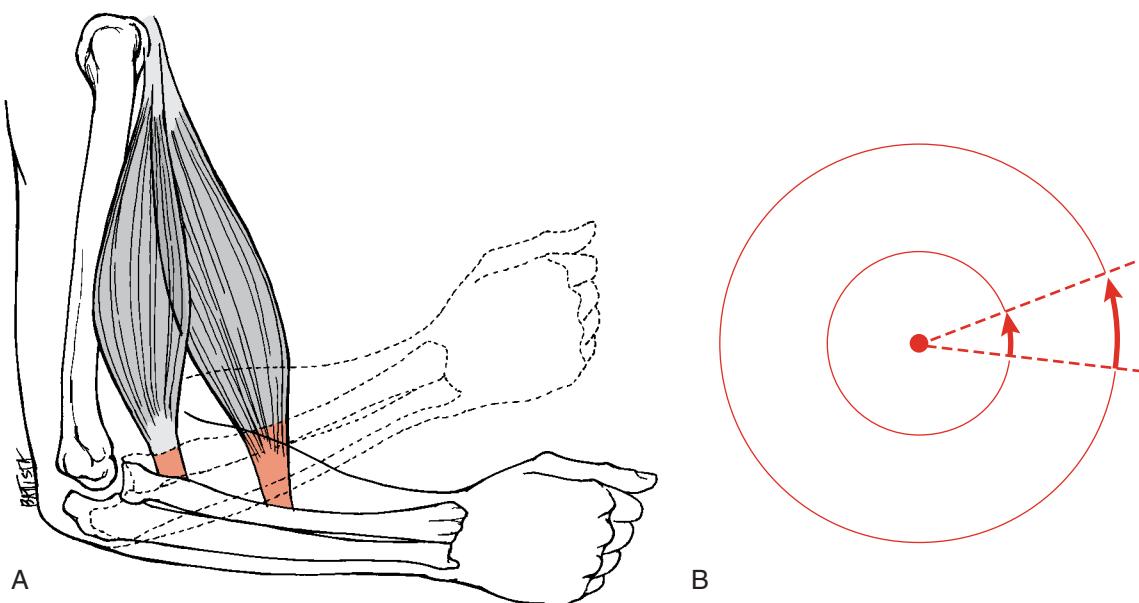


Figure 4.9: The relationship between a muscle's moment arm and excursion. The length of a muscle's moment arm affects the excursion that results from a contraction. **A.** Movement through an angle requires more shortening in a muscle with a long moment arm than in a muscle with a short moment arm. **B.** The arc subtended by an angle is larger in a large circle than in a small circle.

For example, the flexor carpi radialis muscle at the wrist is a good substitute for the extensor digitorum muscle of the fingers in the event of radial nerve palsy. The wrist flexor has long muscle fibers and, therefore, the capacity to extend the fingers through their full ROM. In contrast, the flexor carpi ulnaris, another muscle of the wrist, has very short fibers and lacks the capacity to move the fingers through their full excursion. Thus the functional outcome depends on the surgeon's understanding of muscle mechanics, including those factors that influence the production of motion.

FACTORS THAT INFLUENCE A MUSCLE'S STRENGTH

Strength is the most familiar characteristic of muscle performance. However, the term *strength* has many different interpretations. Understanding the factors affecting strength requires a clear understanding of how the term is used. The basic activity of muscle is to shorten, thus producing a tensile force. As noted in Chapter 1, a force also produces a moment, or a tendency to rotate, when the force is exerted at some distance from the point of rotation. The ability to generate a tensile force and the ability to create a moment are both used to describe a muscle's strength. Assessment of muscle strength *in vivo* is typically performed by determining the muscle's ability to produce a moment. Such assessments include determination of the amount of manual resistance an individual can sustain without joint rotation, the amount of weight a subject can lift, or the direct measurement of moments using a

device such as an isokinetic dynamometer. In contrast, *in vitro* studies often assess muscle strength by measuring a muscle's ability to generate a tensile force. Of course the muscle's tensile force of contraction and its resulting moment are related by the following:

$$\mathbf{M} = \mathbf{F} \times \mathbf{d} \quad (\text{Equation 4.1})$$

where **M** is the moment generated by the muscle's tensile force (**F**), and **d** is the muscle's moment arm. Therefore, muscle strength as assessed typically in the clinic by the measurement of the moment produced by a contraction is a function of an array of factors that influence both the tensile force of contraction, **F**, and the muscle's moment arm, **d** [49]. To obtain valid assessments of muscle strength and to optimize muscle function, the clinician must understand the factors that influence the output of the muscle. All of the following factors ultimately influence the moment produced by the muscle's contraction. Some affect the contractile force, and others influence the muscle's ability to generate a moment. The primary factors influencing the muscle's strength are

- Muscle size
- Muscle moment arm
- Stretch of the muscle
- Contraction velocity
- Fiber types composing the muscle
- Level of muscle fiber recruitment

Each of the factors listed above has a significant effect on the muscle's moment production. An understanding of each factor and its role in moment production allows the clinician

to use these factors to optimize a patient's performance and to understand the alteration in muscle performance with pathology. The effects of size, moment arm, and stretch are most apparent in isometric contractions. Consequently, the experiments demonstrating these effects usually employ isometric contractions. However, the reader must recognize that the effects are manifested in all types of contraction. Each factor is discussed below.

Muscle Size and Its Effect on Force Production

As noted earlier in this chapter, the force of contraction is a function of the number of cross-links made between the actin and myosin chains [1,34]. The more cross-links formed, the stronger the force of contraction. Therefore, the force of contraction depends upon the amount of actin and myosin available and thus on the number of fibers a muscle contains. In other words, the force of contraction is related to a muscle's size [59,113]. In fact, muscle size is the most important single factor determining the tensile force generated by a muscle's contraction [39]. Estimates of the maximal contractile force per unit of muscle range from approximately 20 to 135 N/cm² [13,20,108,138]. These data reveal a wide disparity in the estimates of the maximum tensile force that muscle can produce. Additional research is needed to determine if all skeletal muscle has the same potential maximum and what that maximum really is.

Although the estimates presented above vary widely, they do demonstrate that the maximum tensile force produced by an individual muscle is a function of its area. However, the overall size of a muscle may be a poor indication of the number of fibers contained in that muscle. The relationship between muscle size and force of contraction is complicated by the muscle's architecture. The **anatomical cross-sectional area** of the muscle is the cross-sectional area at the muscle's widest point and perpendicular to the length of the whole muscle. In a parallel fiber muscle this cross-sectional area cuts across most of the fibers of the muscle (Fig. 4.10). However, in a pennate muscle the anatomical cross-sectional area cuts across only a portion of the fibers composing the muscle. Thus the anatomical cross-sectional area underestimates the number of fibers contained in a pennate muscle and hence its force production capabilities.

The standard measure used to approximate the number of fibers of a whole muscle is its **physiological cross-sectional area (PCSA)**. The PCSA is the area of a slice that passes through all of the fibers of a muscle [13]. The PCSA is approximately equal to the anatomical cross-sectional area in a parallel fiber muscle. However, in a pennate muscle the PCSA is considerably larger than its anatomical cross-sectional area. The PCSAs of two muscles of similar overall size demonstrate the influence of muscle architecture on force production. Although their anatomical cross-sectional areas are very similar, the pennate muscle has a much larger

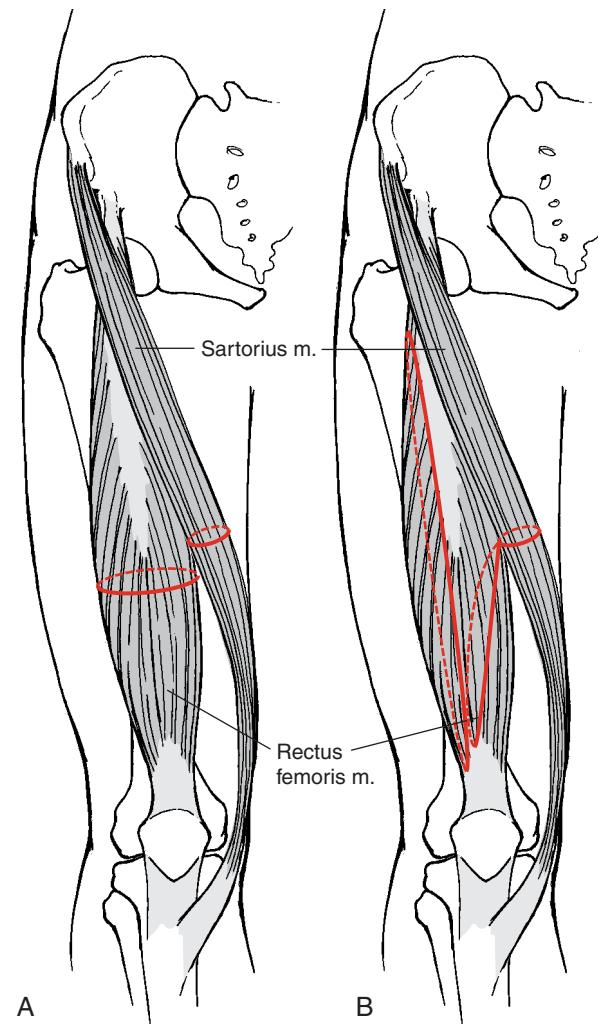


Figure 4.10: The relationship between muscle architecture and muscle size. **A.** The anatomical cross-sectional area of a muscle is the area of a slice through the widest part of the muscle perpendicular to the muscle's length. It is similar in a parallel fiber muscle and a pennate muscle of similar overall size. **B.** The physiological cross-sectional area of a muscle is the area of a slice that cuts across all of the fibers of the muscle. It is quite different for a parallel fiber muscle and a pennate muscle.

PCSA. Thus if all other factors are equal, the pennate muscle is capable of generating more contraction force than the muscle with parallel fibers [56,82,102].

The angle at which the fibers insert into the tendon also influences the total force that is applied to the limb by a pennate muscle. This angle is known as the *angle of pennation*. The tensile force generated by the whole muscle is the vector sum of the force components that are applied parallel to the muscle's tendon (Fig. 4.11). Therefore, as the angle of pennation increases, the tensile component of the contraction force decreases. Consequently, although pennation allows a muscle to increase the number of fibers it contains without increasing its overall size, the ultimate benefit to contraction force made by pennation also depends upon the angle

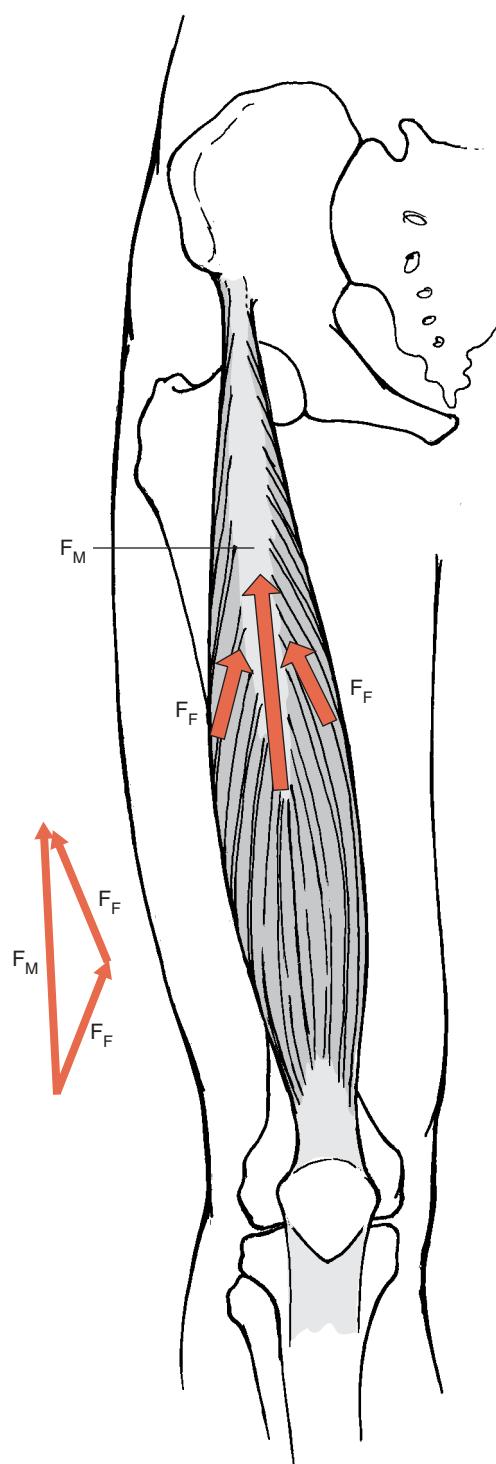


Figure 4.11: The pull of a pennate muscle. The overall tensile force (F_M) of a muscle is the vector sum of the force of contraction of the pennate fibers (F_F).

of pennation. In most muscles the pennation angle is 30° or less, and thus pennation typically increases the tensile force produced by contraction [78,130].

Muscle architecture demonstrates how muscles exhibit specializations that enhance one performance characteristic

or another. Long fibers in a muscle promote the excursion-producing capacity of the muscle. However, spatial constraints of the human body prevent a muscle with long fibers from having a very large cross-sectional area and hence a large force-production capacity. On the other hand, muscles with a large PCSA can be fit into small areas by arranging the fibers in a pennate pattern. However, the short fibers limit the excursion capacity of the muscle. Thus fiber arrangement suggests that pennate muscles are specialized for force production but have limited ability to produce a large excursion. Conversely, a muscle with parallel fibers has an improved ability to produce an excursion but produces a smaller contractile force than a pennate muscle of the same overall size. Thus the intrinsic structural characteristics of a muscle help define the performance of the muscle by affecting both the force of contraction and the amount of the resulting joint excursion. These intrinsic factors respond to an increase or decrease in activity over time [24,56,107,129]. However, instantaneous changes in the muscle also result in large but temporary responses in a muscle's performance. These changes include stretching the muscle and altering its moment arm. These effects are described below.

Relationship between Force Production and Instantaneous Muscle Length (Stretch)

Since the strength of muscle contraction is a function of the number of cross-links made between the actin and myosin chains within the sarcomeres, alterations in the proximity of the actin and myosin chains also influence a muscle's force of contraction. The maximum number of cross-links between the actin and myosin myofilaments and hence the maximum contractile force in the sarcomere occurs when the full length of the actin strands at each end of the sarcomere are in contact with the myosin molecule [30,45,112] (Fig. 4.12). This length is operationally defined as the **resting length** of the muscle. The sarcomere can shorten slightly from this point,

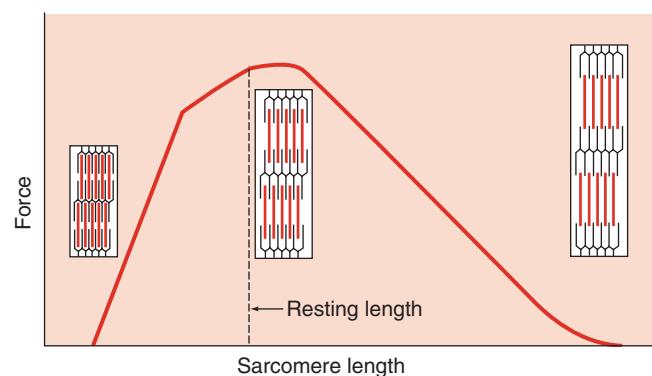


Figure 4.12: The length-tension curve of a sarcomere. The length-tension curve of a sarcomere demonstrates how the length of the sarcomere influences its force production.

maintaining the maximum cross-linking. However, increased shortening causes the actin strands from each end of the sarcomere to interfere with each other. This reduces the number of available sites for cross-bridge formation, and the force of contraction decreases. Similarly, when the sarcomere is stretched from its resting length, contact between the actin and myosin myofilaments decreases, and thus the number of cross-links that can be made again diminishes. Consequently, the force of contraction decreases.

Investigation of the effects of stretch on the whole muscle reveals that the muscle response to stretch is affected both by the behavior of the sarcomere described above and by the elastic properties of the noncontractile components of the muscle, including the epimysium, perimysium, endomysium, and tendons [38,40,48,109]. The classic studies of the length-tension relationships in muscle were performed by Blix in the late 19th century but have been repeated and expanded by others in the ensuing 100 years [38,40,80,82,109]. These studies, performed on whole muscle, consistently demonstrate that as a muscle is stretched in the absence of a contraction, there is some length at which the muscle begins to resist the stretch (Fig. 4.13). As the stretch of the muscle increases, the muscle exerts a larger pull against the stretch. This pull is attributed to the elastic recoil of the passive structures within the muscle, such as the investing connective tissue. These components are known as the **parallel elastic components**. The tendons at either end of the muscle also provide a force against the stretch. These are described as the **series elastic components**.

The combined effects of muscle contraction and stretch of the elastic components are represented mechanically by a

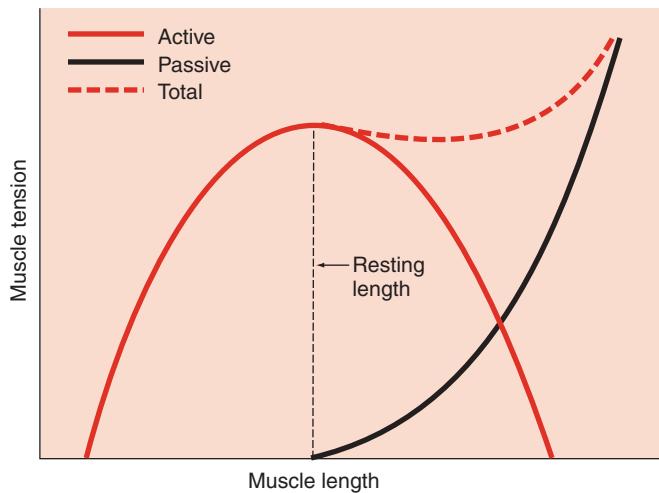


Figure 4.13: The length-tension curve of a whole muscle. The length-tension curve of a whole muscle demonstrates how muscle length affects the force production of the whole muscle. The contractile, or active, component; the passive component primarily due to the connective tissue; and the total muscle tension all are affected by the stretch of the muscle.

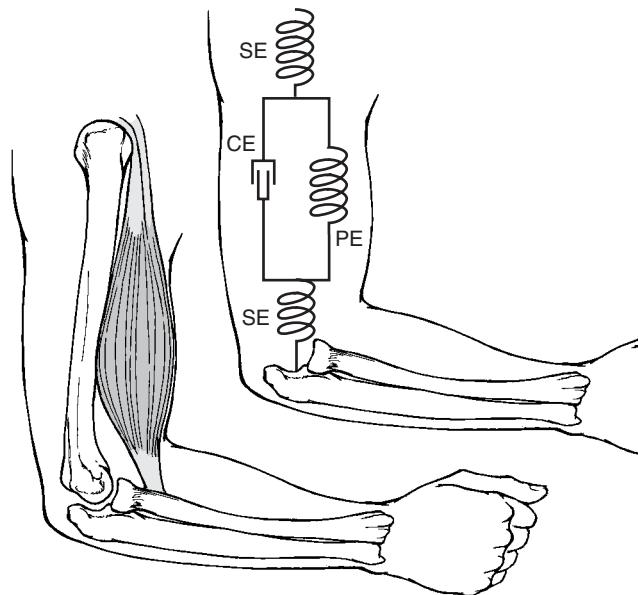


Figure 4.14: A mechanical model of the contractile and elastic components of a muscle. A muscle's contractile (actin and myosin) and elastic (connective tissue) components are often modeled mechanically as a combination of a contractile element with springs that are both in series and in parallel with the contractile component.

contractile element in series and in parallel with the series elastic components (Fig. 4.14). The response of both the contractile and elastic components together is examined by measuring the resistance to increasing stretch while simultaneously stimulating the muscle to induce a contraction. Such experiments reveal that when the muscle is very short, allowing no passive recoil force, stimulation produces a small contractile force. As the stretch increases and stimulations continue, the tension in the muscle increases. In the middle region of stretch, the muscle's force plateaus or even decreases, even with stimulation. This plateau occurs at approximately the resting length of the muscle. With additional stretch, the tension in the whole muscle begins to increase again and continues to increase with further stretch. By subtracting the results of the passive test from the results of the combined test, the contribution of the active, or contractile, component to muscle tension is determined. The active contribution to muscle tension in the whole muscle is similar to the length-tension relationship seen in the individual sarcomeres. These results demonstrate that while the contractile contribution to muscle tension peaks in the midrange of stretch, the passive components of the muscle make an increasing contribution to force after the midrange of stretch. Thus the overall tension of the muscle is greatest when the muscle is stretched maximally.

It is important to recognize that the experiments described above are performed on disarticulated muscles. Consequently, the extremes of shortening and lengthening tested

are nonphysiological. An intact human muscle functions somewhere in the central portion of the length-tension curve, although the precise shape of the length-tension curve varies across muscles [40,136]. The response to stretch depends on the architecture of the individual muscle as well as the ratio of contractile tissue to connective tissue in the muscle [40]. In addition, the exact amount of stretch and shortening sustained by a muscle depends on the individual muscle and the joint. Muscles that cross two or more joints undergo more shortening and lengthening than muscles that span only one joint. The force output of such multijointed muscles is influenced significantly by the length-tension relationship [50,111].

A muscle's length, and therefore its force of contraction, changes as the joint position changes. However, the length of the muscle is only one factor that changes as the joint position changes. The moment arm of the muscle also varies with joint position. The influence of a muscle's moment arm on muscle performance is described below.



A



B

Figure 4.15: The effects of muscle length on performance.
A. When the wrist is in flexion it is difficult to flex the fingers fully because the finger flexors are so shortened.
B. When the wrist is in extension, the fingers readily flex to make a fist since the finger flexors are lengthened.

CLINICAL RELEVANCE: THE LENGTH-TENSION RELATIONSHIP OF MUSCLES IN VIVO

Weakness is a common impairment in individuals participating in rehabilitation. Sometimes individuals are too weak to be able to move the limb much at all. By positioning the patient's limb so that the contracting muscles are functioning in the stretched position, the clinician enhances the muscle's ability to generate tension. For example, hyperextension of the shoulder increases elbow flexion strength by stretching the biceps brachii. Conversely, placing muscles in a very shortened position decreases their ability to generate force. Muscles of the wrist and fingers provide a vivid example of how the effectiveness of muscles changes when they are lengthened or shortened (Fig. 4.15). It is difficult to make a forceful fist when the wrist is flexed because the finger flexor muscles are so short they produce insufficient force. This phenomenon is known as active insufficiency. Inspection of the wrist position when the fist is clenched normally reveals that the wrist is extended, thereby stretching the muscles, increasing their contractile force, and avoiding active insufficiency.

Relationship between a Muscle's Moment Arm and Its Force Production

As noted earlier, a muscle's ability to rotate a joint depends upon the muscle's force of contraction and on its moment arm, the perpendicular distance from the muscle force to the point of rotation [112]. The previous discussion reveals that muscle size and the stretch of the muscle have a significant impact on the force of contraction. However, the muscle's moment arm is critical in determining the moment generated by the muscle contraction. The larger the moment arm, the larger the moment created by the muscle contraction. The relationship between a muscle's moment arm and its angle of application is described earlier in the current chapter. The moment arm is determined by the sine of the angle of application and the distance between the muscle's attachment and the joint's axis of rotation (Fig. 4.16). The moment generated by a muscle contraction is maximum when the muscle's angle of application is 90°, since the sine of 90° equals 1. Additionally, a muscle with a large moment arm produces a larger moment than a muscle with a shorter moment arm if both muscles generate equal contractile forces (Fig. 4.17). The moment arms of some muscles such as the hamstrings change several centimeters through the full ROM of the joint, while others such as the flexor digitorum profundus demonstrate changes of a few millimeters [51,62,63,73,101,121,135]. Therefore, a muscle's ability to produce a moment varies with the joint position.

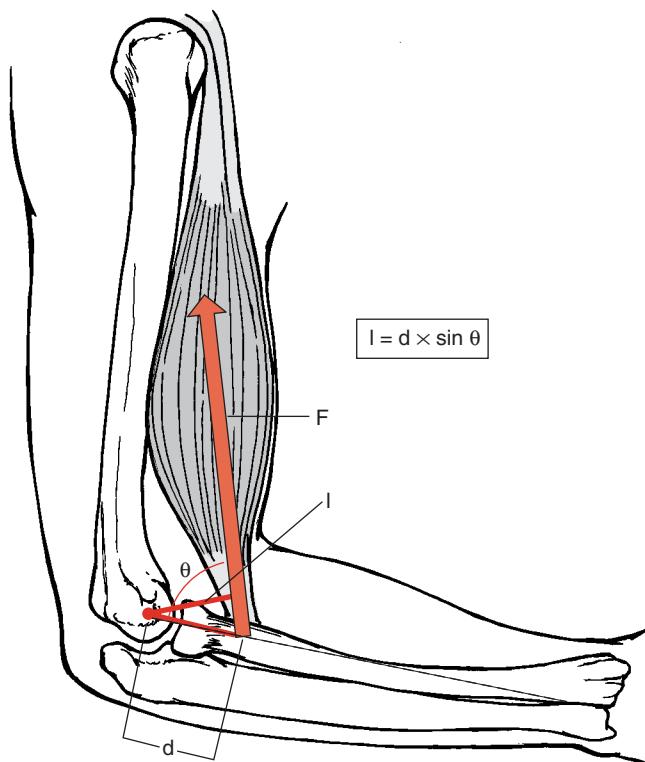


Figure 4.16: Moment arm of a muscle. A muscle's moment arm (I) is easily calculated using the muscle's angle of application (θ) and the distance (d) from the muscle attachment to the axis of rotation.

$$I = d \times \sin \theta$$

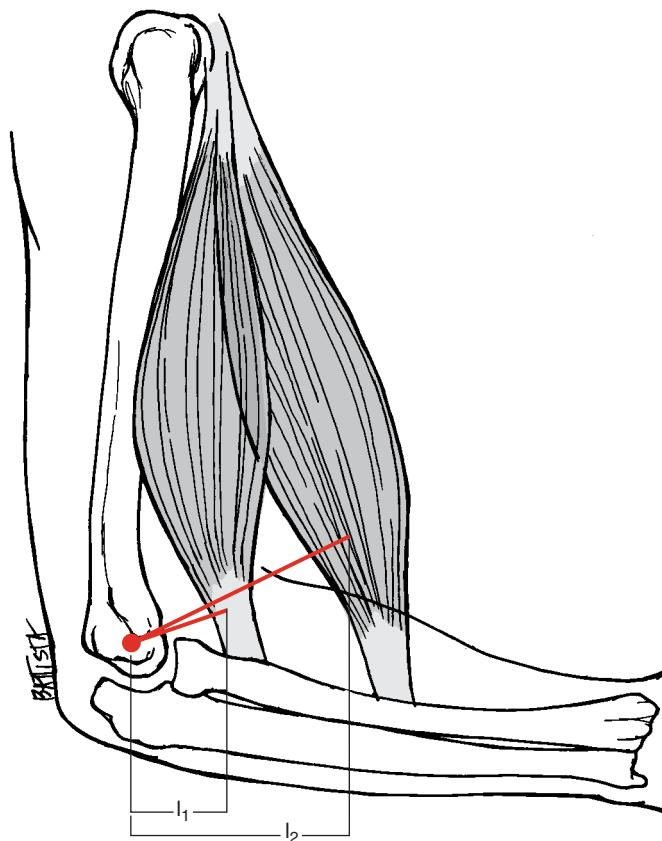


Figure 4.17: The effect of moment arm of a muscle on the muscle's performance. A muscle with a short moment arm (l_1) generates a smaller moment than a muscle with a longer moment arm (l_2) that generates the same contraction force.

INTERACTION BETWEEN A MUSCLE'S MOMENT ARM AND ITS LENGTH WITH CHANGING JOINT POSITIONS

It is easy to observe the positions that shorten or lengthen a muscle. For example, elbow flexion lengthens the elbow extensor muscles and shortens the elbow flexors. Although somewhat less obvious, a knowledge of anatomy allows the clinician to estimate the effects of joint position on a muscle's angle of application and thus on its moment arm. The angle of application of the biceps brachii is almost zero with the elbow extended and increases to over 90° with the elbow flexed maximally. In this case, the muscle's moment arm is at a minimum when the muscle's length is at a maximum. In contrast, the angle of application is greatest when the length is shortest. The optimal angle of application, 90°, occurs when the elbow is flexed to approximately 100° of elbow flexion [3,101]. Thus the muscle's ability to generate a large contractile force as a result of stretch is maximum in the very position in which the muscle's ability to produce a moment is smallest by virtue of its moment arm. Consequently, the biceps produces peak moments in the midrange of elbow flexion where neither the muscle's length nor angle of application is optimal. The relative contribution of moment

arm and muscle length to a muscle's ability to produce a moment varies among the muscles of the body and depends on the individual characteristics of each muscle and joint [55,74,79,91,100,132].

In a series of elegant experiments Lieber and colleagues assess the combined effects of muscle size, moment arm, and length on the ability of the primary wrist muscles, the flexor carpi ulnaris, flexor carpi radialis, extensor carpi ulnaris, and extensor carpi radialis longus and extensor carpi radialis brevis to produce a joint torque in the directions of wrist flexion, extension, and radial and ulnar deviation [80,86,87]. These investigations reveal that the influence of moment arms and muscle lengths differs markedly among these muscles of the wrist. The output from the wrist extensor muscles correlates well with the muscles' moment arms, suggesting that their output depends largely on the moment arms and is less influenced by muscle length. In contrast, the output of the wrist flexors is nearly maximum over a large portion of the wrist range, suggesting that both moment arm and muscle length have significant impact on the muscles' performance.

CLINICAL RELEVANCE: JOINT POSITION'S INFLUENCE ON MUSCLE STRENGTH

Joint position is likely to have a dramatic effect on the output from a muscle contraction, since joint position affects both the stretch and the moment arm of a muscle. The exact influence is revealed through careful testing and varies across muscles and joints. Similarly, only careful investigation provides an explanation for the precise nature of the relationship between joint position and muscle force. However, a valid clinical assessment of strength requires that the joint position at which strength is assessed be maintained for each subsequent test. The clinician must consider the effects of joint position on muscle output when measuring strength and also when designing intervention strategies to improve muscle function. Unless the effects of muscle moment arm and muscle length are held constant, changes in strength resulting from intervention cannot be distinguished from changes resulting from the mechanical change in the muscle.

The following scenario provides a helpful demonstration. In the initial visit to a patient treated at home, the clinician measures hip flexion strength while the patient is sitting in a wheelchair. Weakness is identified, and exercises are provided. On the next visit, 2 days later, the clinician finds the patient in bed and so measures hip flexion strength in bed with the hip extended. Hip flexion strength is greater at this measurement than in the previous measurement. The astute therapist recognizes that the apparent increase in strength may be attributable to the change in position, since muscle hypertrophy as a result of exercise is unlikely after only 2 days.

Relationship between Force Production and Contraction Velocity

The chapter to this point has examined the influence of muscle factors on force production only in isometric contractions, contractions with no visible change in muscle length. However in nonisometric contractions, the direction and speed of contraction influence the muscle's output. Speed of movement and its direction are described together by the vector quantity velocity. This section examines the effects of contraction velocity on muscle output. Both the direction and the magnitude of the velocity are important influences and are discussed individually below.

EFFECTS OF THE MAGNITUDE OF THE CONTRACTION VELOCITY ON FORCE PRODUCTION IN MUSCLE

Contractile velocity of a muscle is determined usually by the macroscopic change in length per unit time. Thus an **isometric contraction** has zero contraction velocity. It is important to recognize that on the microscopic level there is a change in length of the muscle even in an isometric contraction. In

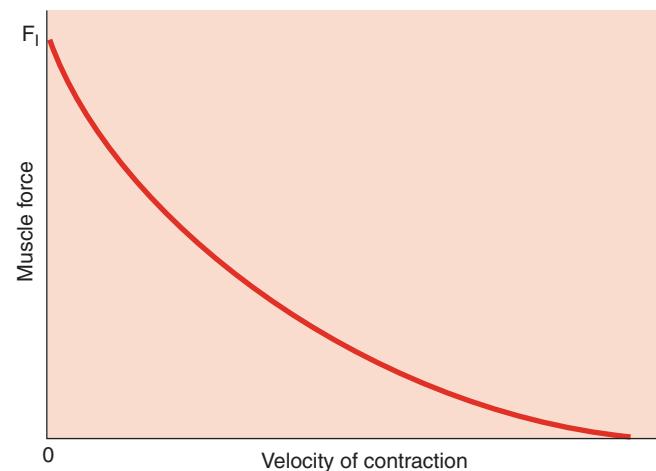


Figure 4.18: The relationship between contractile force and the velocity of contraction in isometric and concentric contractions. A plot of contractile force and the velocity of contraction from isometric (F_I) to concentric contractions shows that the strength of the contraction decreases with increasing contractile velocity.

contrast, a **concentric contraction** is defined as a contraction in which there is visible shortening of the muscle. Thus a concentric contraction has a positive contraction velocity.

The relationship between contractile force and speed of contraction in isometric and shortening contractions has been studied for most of the 20th century and is well understood [32,33,60,67,110,125,131]. A plot of a muscle's force of contraction over contractile velocity for isometric and concentric contractions reveals that contractile force is maximum when contraction velocity is zero (isometric contraction) and decreases as contraction velocity increases (Fig. 4.18). Thus an isometric contraction produces more force than a concentric contraction of similar magnitude. Similarly, a rapid shortening contraction produces less force than a slow shortening contraction.

CLINICAL RELEVANCE: EXAMINING MUSCLE STRENGTH IN THE CLINIC

Both isometric and concentric contractions are used in the clinic to assess strength. For example, one form of the standardized manual muscle testing procedures examines the force of an isometric contraction at the end of range, while another form measures the force of a concentric contraction through the full ROM to grade a muscle's force [53]. Similarly, clinicians use isokinetic dynamometers to measure both isometric and concentric strength. Each of these tests is valid, and there is a correlation among maximum force at various contraction velocities [66,106]. However, it is important for clinicians to recognize that the absolute force produced depends on the testing mode. If all other factors of muscle performance are constant, the isometric contractions produce greater forces than the concentric forces. Judgments regarding the adequacy of an individual's strength must consider the effects of contraction velocity.

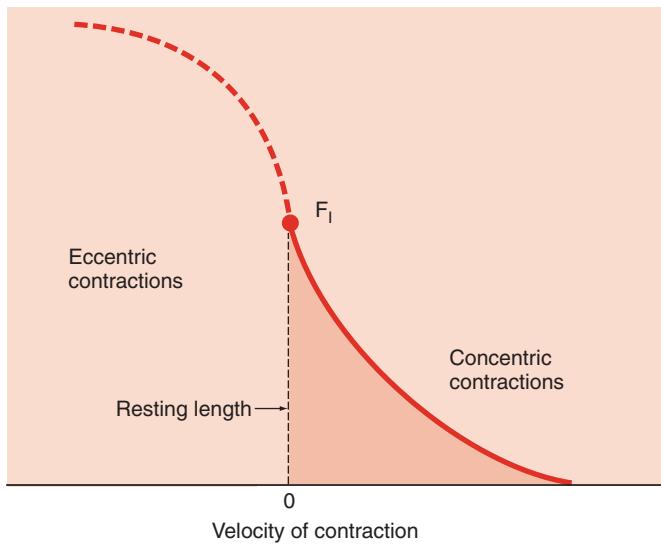


Figure 4.19: The relationship between contractile force and the velocity of contraction in isometric, concentric, and eccentric contractions. A plot of contractile force and the velocity of contraction from eccentric to concentric contractions shows that an eccentric contraction is stronger than either isometric (F_i) or concentric contractions.

EFFECTS OF THE DIRECTION OF CONTRACTION ON FORCE PRODUCTION IN MUSCLE

As noted earlier, both the magnitude and direction of the contraction influence a muscle's performance. A contraction that occurs as a muscle visibly lengthens is called an **eccentric contraction**. Eccentric contractile strength is less well understood than isometric and concentric strength, at least in part because it is difficult to study lengthening contractions over a large spectrum of speeds in intact muscles. Despite this limitation, many studies have been completed and provide important information regarding the comparative contractile force of eccentric contractions. A plot of muscle tension over the whole spectrum of contraction velocities reveals that eccentric contractions produce more force than either isometric or concentric contractions [25,32,41,52,54,70,72,105, 114,118,124] (Fig. 4.19). Maximum eccentric strength is estimated to be between 1.5 and 2.0 times maximum concentric strength [114,128]. The plot of muscle force as a function of contraction velocity also reveals that the effect of the magnitude of contraction velocity on force production plateaus in an eccentric contraction [25,32,83].

CLINICAL RELEVANCE: POST-EXERCISE MUSCLE SORENESS
Studies indicate that delayed-onset muscle soreness (DOMS) typically is associated with exercise involving eccentric exercise [10,35]. Although this phenomenon has not been thoroughly explained, one possible explanation is that a muscle generates greater forces in maximal eccentric contractions than in maximal concentric contractions. Thus the DOMS

may be the result of excessive mechanical loading of the muscle rather than an intrinsic difference in physiology of the eccentric contraction.

It is important to note that the length-tension relationship in muscle demonstrated earlier in the current chapter persists regardless of the direction or speed of the contraction. As a result, the shape of the plots of muscle force through the ROM are similar, regardless of velocity [67,71] (Fig. 4.20).

Relationship between Force Production and Level of Recruitment of Motor Units within the Muscle

Earlier in the current chapter it is reported that the strength of the cross-links between actin and myosin is influenced by the frequency of stimulation by the motor nerve. Examination of the function of a whole muscle reveals a similar relationship. A whole muscle is composed of smaller units called *motor units*. A **motor unit** consists of the individual muscle fibers innervated by a single motor nerve cell, or motoneuron. The force of contraction of a whole muscle is modulated by the frequency of stimulation by the motor nerve and by the number of motor units active. A single stimulus of low intensity from the motor nerve produces depolarization of the muscle and a **twitch** contraction of one or more motor units. As the frequency of the stimulus increases, the twitch is repeated. As in the single fiber, if the stimulus is repeated

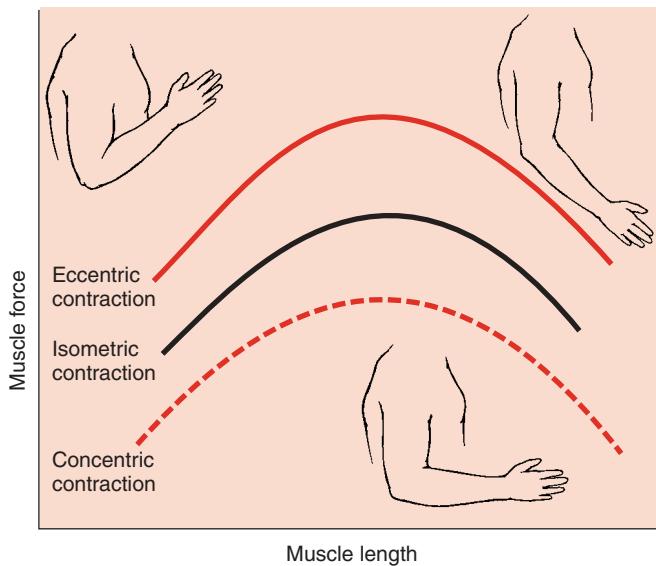


Figure 4.20: Comparison of eccentric, isometric, and concentric muscle strengths with changing muscle length. A comparison of eccentric, isometric, and concentric muscle strengths through an ROM reveals that the force of an eccentric contraction is greater than the force of an isometric contraction, which is greater than the force of a concentric contraction, regardless of the length of the muscle.

before the muscle relaxes, the twitches begin to fuse, and a sustained, or **tetanic**, contraction is elicited. As the intensity of the stimulus increases, more motor units are stimulated, and the force of contraction increases. Thus a muscle is able to produce maximal or submaximal contractions by modifying the characteristics of the stimulus from the nerve.

The amount of activity of a muscle is measured by its electromyogram (EMG). The EMG is the electrical activity induced by depolarization of the muscle fibers. In an isometric contraction, there is a strong relationship between the electrical activity of the muscle, its EMG, and the force of contraction. As isometric force increases, the EMG also increases [22,26,27,70,116,120,122,126]. This relationship is logical, since the force of contraction is a function of the number of cross-links formed between the actin and myosin chains and thus a function of the number of muscle fibers contracting. The EMG reflects the number of active fibers as well as their firing frequency [7,9,11,120]. However, the relationship of the muscle's EMG and its force of contraction is more complicated when the muscle is free to change length and the joint is free to move.

This chapter demonstrates that the size and stretch of the muscle, the muscle's moment arm, and the velocity of contraction all contribute to the force produced by contraction. The EMG merely serves to indicate the electrical activity in a muscle. Thus a larger muscle produces a larger EMG pattern during a maximal contraction than a smaller muscle performing a maximal contraction, since there are more motor units firing in the larger muscle. However, within the same muscle, a maximal eccentric contraction elicits an EMG pattern similar to that produced during a maximal concentric contraction, even though the force of contraction is greater in the eccentric contraction [70,114]. In the case of maximal contractions, the muscle recruits approximately the same number of motor units regardless of the output. The output of the muscle varies primarily because of the mechanical effects of contraction velocity.

CLINICAL RELEVANCE: ASSESSMENT OF PEAK STRENGTH

The basic premise of strength assessment is that the test subject is producing a maximal contraction; that is, the subject is maximally recruiting available motor units. The validity and reliability of muscle testing depends upon the tester's ability to motivate the individual to produce a maximal contraction. A classic study of the reliability of manual muscle testing reveals that an important factor explaining the lack of reliability is that some testers failed to elicit a maximal contraction, erroneously grading a submaximal contraction [57]. Encouraging a subject to produce a maximal effort requires both psychological and mechanical skills that are developed with knowledge and practice but are essential to valid and reliable measures of strength.

In a submaximal contraction, the muscle recruits enough motor units to produce the necessary muscle force. A muscle

that is lengthened or positioned with a large moment arm is said to be at a **mechanical advantage**. It can produce the same moment with less recruitment and consequently a smaller EMG than when the muscle is at a mechanical disadvantage, positioned at a shortened length, or with a small moment arm [58,97]. When a muscle is at a mechanical advantage or when it is stronger, it needs fewer motor units to generate a moment; when the muscle is at a mechanical disadvantage or is weaker, it must recruit more motor units to generate the same moment [8,26,96].

This literature review demonstrates that EMG reflects the relative activity of a muscle rather than providing a direct measure of the force of that muscle's contraction. The literature is filled with studies of the EMG activity of muscles during function. These studies are used to explain the role of muscles during activity. Reference to such articles is made frequently throughout this textbook. However, caution is needed when interpreting these studies, since EMG reflects only the relative activity of a muscle. Muscle size and mechanical advantage affect the recorded electrical activity. There are also several technical factors that influence the magnitude of EMG produced during muscle contraction. These include the type and size of the recording electrodes and the signal-processing procedures. These issues are beyond the scope of this book, but they serve as a warning to the clinician that interpretation of EMG and comparisons across studies must proceed with caution. To improve the generalizability of EMG data, analysis of the electrical activity of a muscle typically involves some form of **normalization** of the data. A common normalizing procedure is to compare the activity of a muscle to the EMG produced by a maximal voluntary contraction (MVC). The basic premise in this approach is that an MVC requires maximal recruitment of a muscle's motor units, which then produces a maximum electrical signal. This maximal activity is used as the basis for comparing the muscle's level of activity in other activities. Processing of the electrical signal also affects the interpretation of the signal [7]. A discussion of the issues involved in the analysis of EMG data is beyond the scope of this book. However, the reader is urged to use EMG data cautiously when analyzing the roles of individual muscles.

Relationship between Force Production and Fiber Type

The last characteristic of muscle influencing the force of contraction to be discussed in this chapter is the type of fibers composing an individual muscle. Different types of muscle fibers possess different contractile properties. Therefore, their distribution within a muscle influences the contractile performance of a whole muscle. However, because human muscles are composed of a mix of fiber types, fiber type has less influence on the force-producing capacity of a muscle than do the factors discussed to this point.

There are a variety of ways to categorize voluntary muscle fibers based on such characteristics as their metabolic

TABLE 4.1 Basic Performance Characteristics of Types I, IIa, and IIb Muscle Fibers

	I	IIa	IIb
Contraction velocity	Slow	Moderately fast	Fast
Contractile force	Low	Variable	High
Fatigability	Fatigue resistant	Somewhat fatigue resistant	Rapidly fatiguing

processes, their histochemical composition, and their phenotype. Although each method examines different properties, each identifies groups ranging from fatigue-resistant fibers with slow contractile properties to rapidly fatiguing cells with faster contractile velocities [137]. A common cataloging system based on metabolic properties classifies most human muscle fibers as type I, type IIa, or type IIb fibers. Characteristics of these three fiber types are listed in Table 4.1. For the purposes of the current discussion, a closer examination of the mechanical properties of these fibers is indicated. In general, the contractile force of a type IIb fiber is greater than that of a type I fiber [12]. Thus muscles composed of more type IIb fibers are likely to generate larger contractile forces than a comparable muscle consisting of mostly type I fibers [98]. Type I fibers are innervated by small-diameter axons of the motor nerve. They are recruited first in a muscle contraction. Type IIb fibers are innervated by large axons and are recruited only after type I and type IIa fibers. Type IIb fibers are recruited as the resistance increases [94,95].

The velocity of contraction also differs among fiber types [2,12]. Consequently, the force–velocity relationship also varies among the fiber types. Data from human muscles suggest that type IIb fibers exert larger forces at higher velocities, while type I fibers have slower maximal contractile velocities as well as lower peak forces [12]. Thus muscles with a preponderance of type II fibers have a higher rate of force production and a higher contractile force than muscles with more type I fibers [1].

Postural muscles typically are composed largely of type I fibers, while muscles whose functions demand large bursts of force consist of more type II fibers [1,119]. However, as already noted, human muscles contain a mixture of fiber types [28,29,93,95]. Therefore, the contractile properties of whole muscles reflect the combined effects of the fibers types. Consequently, the other factors influencing force production such as muscle size and mechanical advantage appear to have a larger influence on contractile force [23]. However, muscle fibers demonstrate different responses to changes in activity and thus play a significant role in muscle adaptation. The adaptability of muscle is discussed briefly below.

ADAPTATION OF MUSCLE TO ALTERED FUNCTION

Muscle is perhaps the most mutable of biological tissues. A discussion of the mechanical properties of muscle cannot be complete without a brief discussion of the changes in these

mechanical properties resulting from changes in the demands placed on muscle. The following provides a brief discussion of the changes in muscle that occur in response to sustained changes in

- Muscle length
- Activity level

Understanding the effects of sustained changes in muscle length or activity level is complicated by the recognition that these factors are often combined in investigations. Studies assessing the effects of length changes often use immobilization to apply the length change. Consequently, the muscles respond to both the altered length and decreased activity. As a result, a complete understanding of the influence of these factors on muscle function continues to elude investigators. The following briefly reviews the current state of knowledge of muscles' adaptation to altered function.

Adaptation of Muscle to Prolonged Length Changes

The relationship between stretch of a muscle and its force of contraction is presented in detail elsewhere in this chapter. This relationship is a function of both the contractile and non-contractile components of muscle. However, it also is important to ponder the effect of prolonged length change on the length–tension relationship. Since muscles are organized in groups of opposing muscles, when one muscle is held on stretch, another muscle is held in a shortened position. Therefore, it is important to consider a muscle's response to both prolonged lengthening and prolonged shortening. The vast majority of studies examining alterations in muscle resulting from prolonged length changes use immobilization procedures to provide the length change. Therefore, the reader must exert caution when attempting to generalize these results to other cases such as postural abnormalities that do not involve immobilization.

CHANGES IN MUSCLE WITH PROLONGED LENGTHENING

In general, prolonged stretch of a muscle induces protein synthesis and the production of additional sarcomeres [43,44,123,134,137]. The muscle hypertrophies, and as a result, peak contractile force is increased with prolonged stretch. The addition of sarcomeres in series increases the overall length of the muscle fibers. This remodeling appears important in allowing the muscle to maintain its length–tension relationship.

There also is evidence of changes in the metabolic characteristics of muscle cells subjected to prolonged stretch. Some muscles exhibit changes in mRNA consistent with a transition from type II to type I fibers [137].

Although hypertrophy is the typical muscle response to prolonged stretch, studies report more varied responses among individual muscles. Changes in muscle mass, peak strength, and even gene expression with prolonged stretch vary across muscles and appear to depend upon the muscle's fiber type composition and its function [78,88].

CHANGES IN MUSCLE HELD IN A SHORTENED POSITION FOR A PROLONGED PERIOD

Investigation into the effects of prolonged shortening also demonstrates a complex response. Prolonged shortening produced by immobilization appears to accelerate atrophy, and muscles demonstrate a loss of sarcomeres [43,123,137]. Some muscles immobilized in a shortened position also show evidence of a transition toward type II fibers. Yet a study examining the effects of shortening without immobilization reports an increase in sarcomeres [69]. Results of this study suggest that tendon excursion may be a stronger factor than the shortening itself in determining the muscle's remodeling. In addition, like prolonged stretch, prolonged shortening yields different responses in different muscles [78].

Clearly, complete understanding of the factors inducing muscle adaptation requires further investigation. The studies reported here demonstrate that the adaptability of muscle to prolonged length changes is complex and depends on many factors besides the specific change in length. Yet these studies do consistently demonstrate changes that seem directed, at least in part, at maintaining a safe and functional length-tension relationship in each muscle [78,112,123].

CLINICAL RELEVANCE: PROLONGED LENGTH CHANGES IN MUSCLE AS THE RESULT OF POSTURAL ABNORMALITIES

Postural abnormalities reportedly produce prolonged lengthening of some muscles and prolonged shortening of other muscles [61]. This has led to the belief that abnormal posture produces changes in muscle strength. Studies have attempted to identify such changes in strength and changes in the length tension relationships of muscles that appear to be affected by postural abnormalities [21,104]. However, these studies fail to demonstrate a clear change in strength attributable to length changes. Yet clinicians continue to treat abnormal postural alignment with strengthening and stretching exercises. Although current studies neither prove nor disprove the existence of clinically measurable changes in muscle as the result of prolonged length changes, they emphasize the need for clinicians to use caution in assuming relationships between postural alignment and muscular strength.

Adaptations of Muscle to Sustained Changes in Activity Level

Muscle's basic response to changes in activity level is well known: increased activity results in hypertrophy and increased force production, and decreased activity leads to atrophy and decreased force production. Of course the exact response is far more complicated than this. The response depends on the nature of the activity change and on the nature of the muscle whose activity is altered.

Resistance exercise leads to muscle hypertrophy and increased strength in both men and women of virtually all ages [16,64,75,107,129]. Strengthening exercises in humans produce an increase in the cross-sectional area (CSA) of both type I and type II fibers, although there is evidence that there is a greater increase in the CSA of type II fibers [23,24,54,89,92,103]. In addition, animal studies reveal that protein synthesis is consistent with a transition from type IIb fibers to type I fibers [5,78].

In contrast, decreased activity produces a decrease in CSA and loss of strength [42,77,103]. One study reports a 13% decrease in some lower extremity strength in 10 healthy subjects who underwent only 10 days of non-weight-bearing activity [8]! Disuse atrophy is apparent in both type I and type II fibers. In addition, there is evidence supporting a transition from type I fibers to type II fibers [4,5,88].

Although the preceding discussion demonstrates general patterns of muscle response to changes in activity level, the response is actually quite muscle dependent [77,78]. One study reports a 26% loss in plantarflexion strength with no significant loss in dorsiflexion strength in healthy individuals following 5 weeks of bed rest [77]. Animal studies show similar differences among muscle groups [17,78]. Other mechanical factors such as stretch also affect a muscle's response to reduced activity [88].

CLINICAL RELEVANCE: DISUSE ATROPHY IN PATIENTS

Patients who have spent prolonged periods in bed are likely to demonstrate significant loss of strength resulting directly from the inactivity and unrelated to other simultaneous impairments or comorbidities. However, the effects of inactivity may be manifested differently in the various muscle groups of the body. The clinician must be aware of the likely loss of strength and also must consider the possible loss of muscular endurance that may result from a transition from type I to type II muscle fibers. In addition, the clinician also must screen carefully for these changes to identify those muscle groups that are most affected by disuse.

AGING AS ANOTHER MODEL OF ALTERED ACTIVITY

Loss of strength is a well-established finding in aging adults [15,75,85,90,107,117]. This loss of strength is attributed to a decreased percentage of, and greater atrophy in, type II fibers

[65,76,115]. As in the other adaptations of muscle described above, changes in muscle with age vary across muscles [18]. Some muscle groups appear to be more susceptible to age-related change; others seem impervious to such changes. Again these data reveal that the clinician must assess strength in the aging individual. However, the clinician must also take care to identify those muscle groups that are weakened and those that are relatively unaffected, to target the intervention specifically for optimal results.

CLINICAL RELEVANCE: DECREASED STRENGTH WITH AGING

Decreased functional ability is a frequent finding with aging. Although many factors contribute to diminished function with age, investigations demonstrate a relationship between diminished functional ability and decreased strength [19,21,46]. Similarly, increasing strength in elders improves functional ability [31,99]. One of the challenges in rehabilitation is to identify successful strategies to prevent or reduce strength loss and preserve functional ability in the aging population.

SUMMARY

This chapter reviews the basic mechanisms of muscle shortening and discusses in detail the individual factors that influence a muscle's ability to produce motion and to generate force. The primary factors influencing a muscle's ability to produce joint motion are the length of the muscle fibers within the muscle and the length of the muscle's moment arm. Muscle strength, including its tensile force of contraction and its resulting moment, is a function of muscle size, muscle moment arm length, stretch of the muscle, contraction velocity, fiber types within the muscle, and amount of muscle fiber recruitment. Each factor is described and examples are provided to demonstrate how an understanding of the factor can be used in the clinic to explain or optimize performance. The discussion also demonstrates that often as one factor is enhancing a performance characteristic another factor may be detracting from that performance. The final output of a muscle is the result of all of the factors influencing performance. Thus to understand the basis for a patient's performance, the clinician must be able to recognize how the individual factors influencing muscle performance change as joint position and motion change.

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