CHAPTER 4

Biomechanics of Skeletal Muscle

CHAPTER CONTENTS

STRUCTURE OF SKELETAL MUSCLE 46
Structure of an Individual Muscle Fiber 46
The Connective Tissue System within the Muscle Belly 48
FACTORS THAT INFLUENCE A MUSCLE'S ABILITY TO PRODUCE A MOTION 48
Effect of Fiber Length on Joint Excursion 48
Effect of Muscle Moment Arms on Joint Excursion 50
Joint Excursion as a Function of Both Fiber Length and the Anatomical Moment Arm of a Muscle 51
FACTORS THAT INFLUENCE A MUSCLE'S STRENGTH 52
Muscle Size and Its Effect on Force Production 52
Relationship between Force Production and Instantaneous Muscle Length (Stretch) 53
Relationship between a Muscle's Moment Arm and Its Force Production 56
Relationship between Force Production and Contraction Velocity 58
Relationship between Force Production and Level of Recruitment of Motor Units within the Muscle 60
Relationship between Force Production and Fiber Type 61
ADAPTATION OF MUSCLE TO ALTERED FUNCTION 62
Adaptation of Muscle to Prolonged Length Changes 62
Adaptations of Muscle to Sustained Changes in Activity Level 63
SUMMARY 64

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 [41,52,86].

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 are 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 person'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

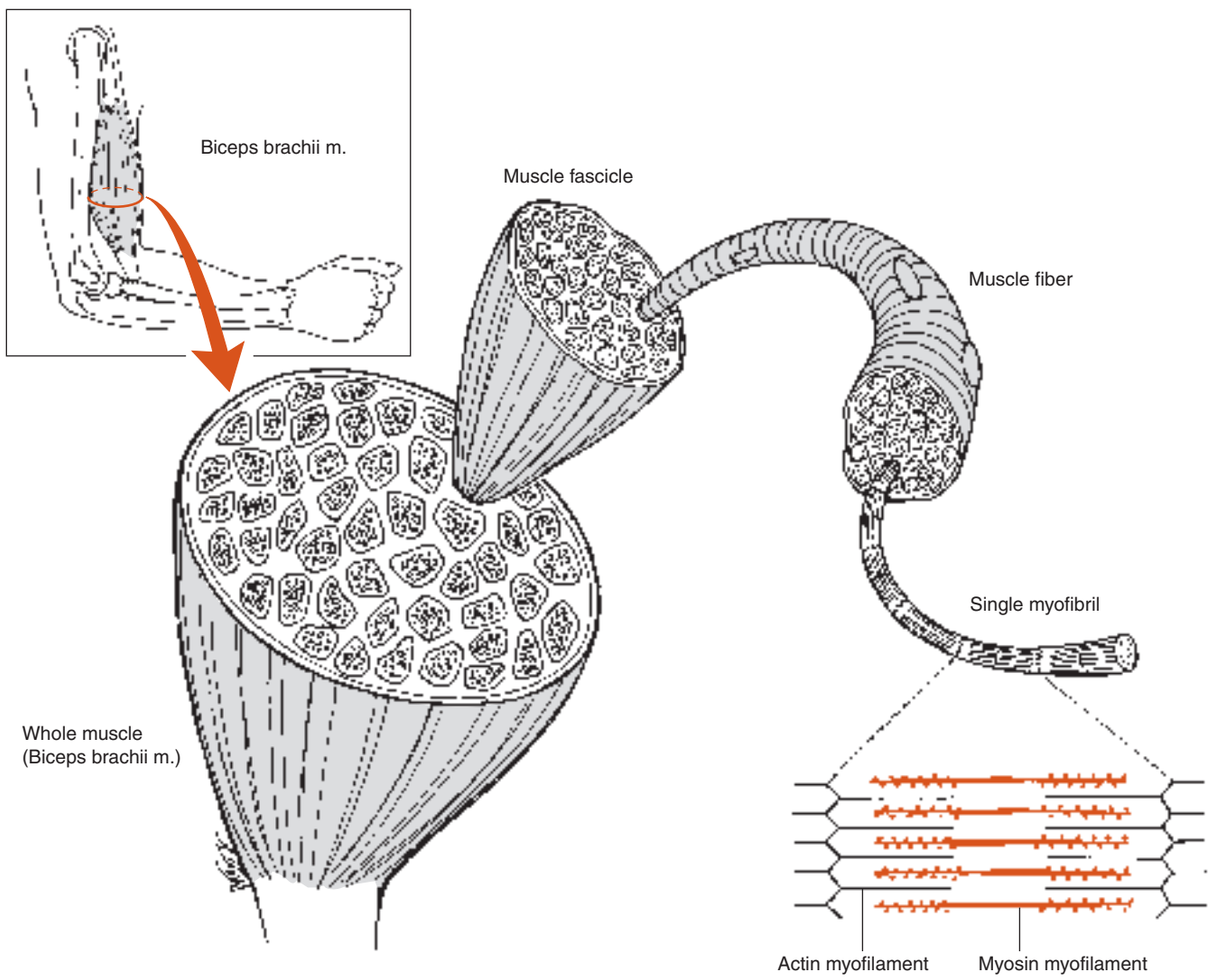

Figure 4.1: Organization of muscle. A progressively magnified view of a whole muscle demonstrates the organization of the filaments composing the muscle.

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.

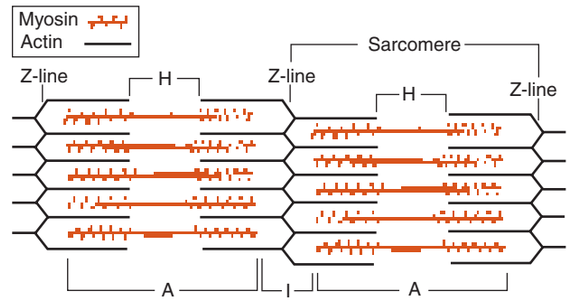

Figure 4.2: Organization of actin and myosin within a muscle fiber. The arrangement of the actin and myosin chains in two adjacent sarcomeres within a fiber produces the characteristic striations of skeletal muscle.

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 [6,7,27].

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 [86]. If

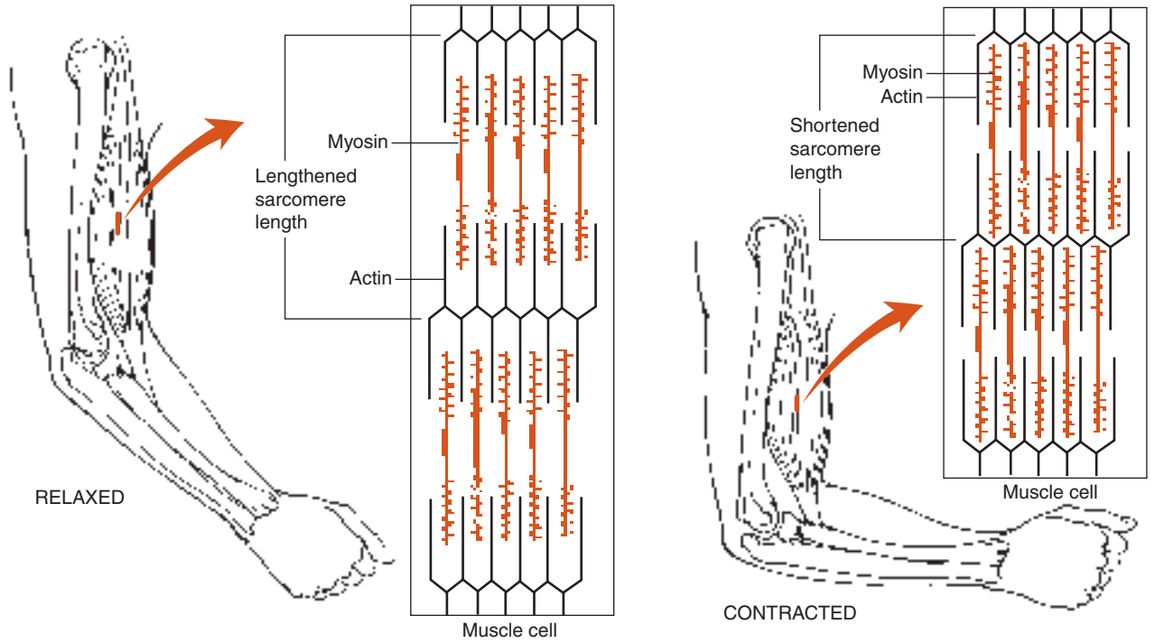

Figure 4.3: The sliding filament model. Contraction of skeletal muscle results from the sliding of the actin chains on the myosin chains.

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 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 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.

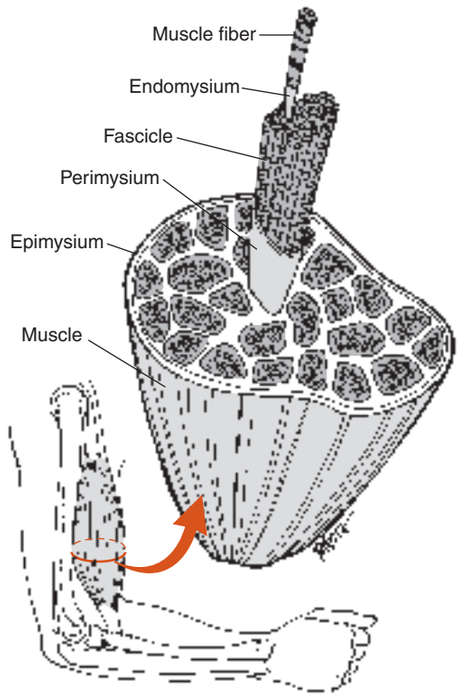

Figure 4.4: Organization of the connective tissue within muscle. The whole muscle belly is invested in an organized system of connective tissue. It consists of the epimysium surrounding the whole belly, the perimysium encasing smaller bundles of muscle fibers, and the endomysium that covers individual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

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 [125,149]. Thus sarcomeres in humans are a few micrometers in length, varying from approximately 1.25 to 4.5 μm with muscle contraction and stretch [90–92,143]. Each sarcomere can shorten to approximately the length of its myosin molecules. Because 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 [15,89,155]. A fiber can shorten roughly 50 to 60% of its length [44,155], although

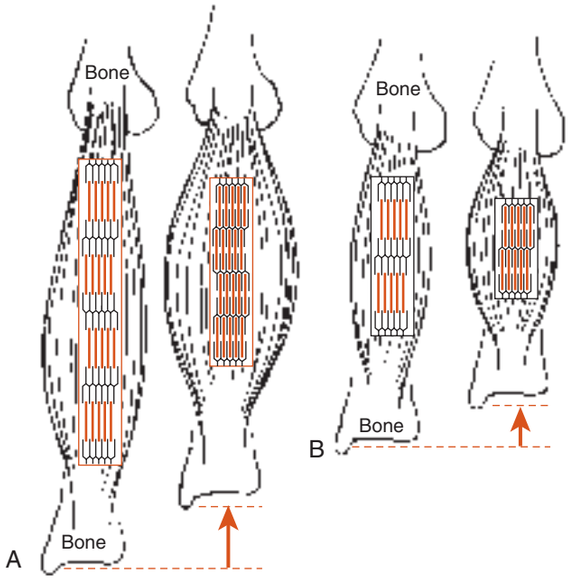

Figure 4.5: The relationship between fiber length and shortening capacity of the whole muscle. A muscle with more sarcomeres in series **(A)** can shorten more than a fiber with fewer sarcomeres in series **(B).**

there is some evidence that fibers exhibit varied shortening capabilities [15].

The absolute amount of shortening a fiber undergoes is a function of its fiber length. Similarly, 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 [15]. However there is a wide variation in fiber lengths found in the human body, ranging from a few centimeters to approximately half a meter [86,146]. 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** [42] (*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

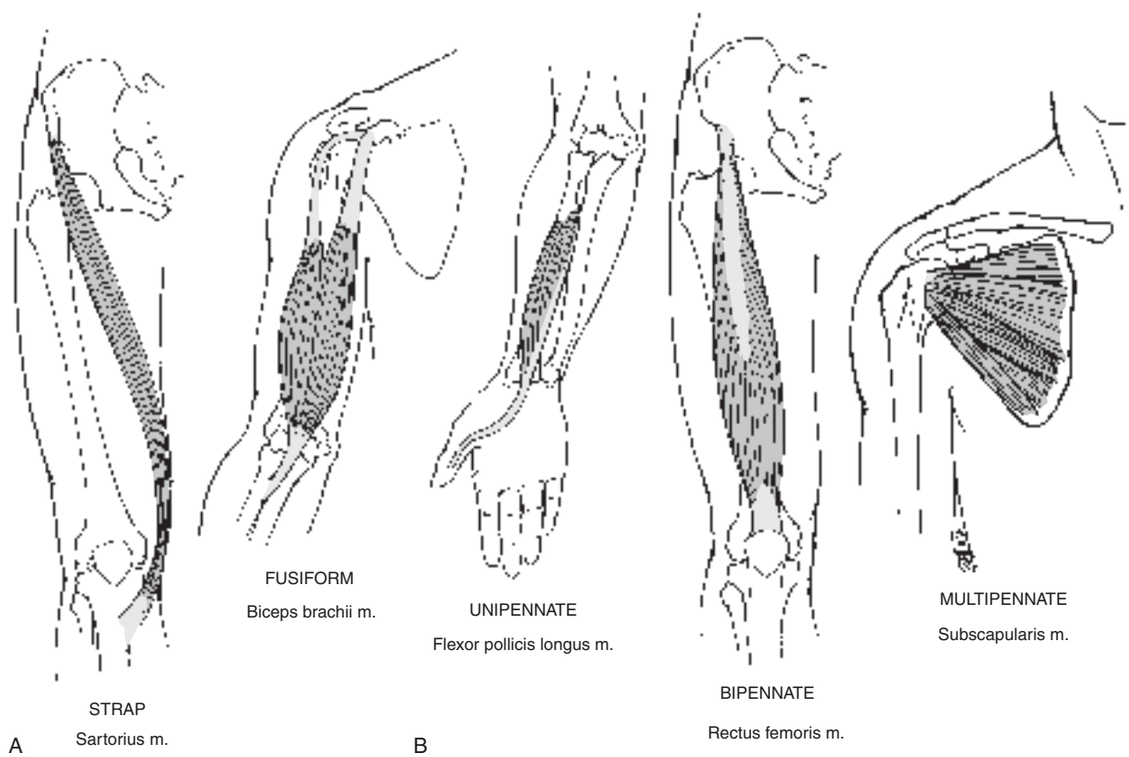

Figure 4.6: Muscle architecture. **A.** Muscles with parallel fibers include fusiform (biceps brachii) and strap (sartorius) muscles. **B.** Pennate muscles include unipennate (flexor pollicis longus), bipennate (rectus femoris), and multipennate (subscapularis).

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 than the muscle composed of pennate fibers [90]. 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.

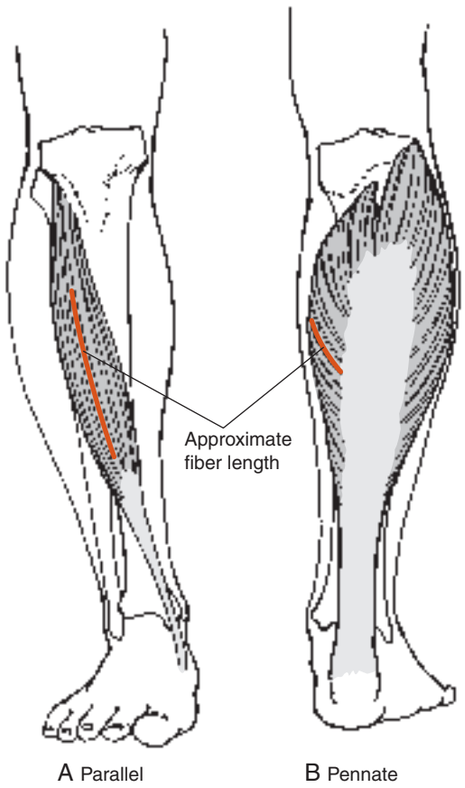

Figure 4.7: The relationship between muscle architecture and muscle fiber length. The fibers in a muscle with parallel fibers are typically longer than the fibers in a muscle of similar overall size but with pennate fibers.

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 between 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 [57,81,95,151]. This

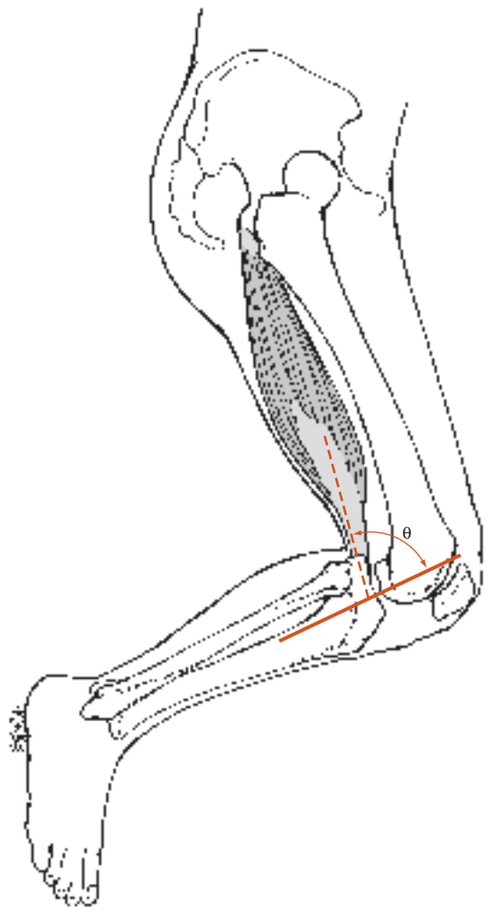

Figure 4.8: Angle of application. A muscle's angle of application is the angle formed between the line of pull of the muscle and the bone to which the muscle attaches.

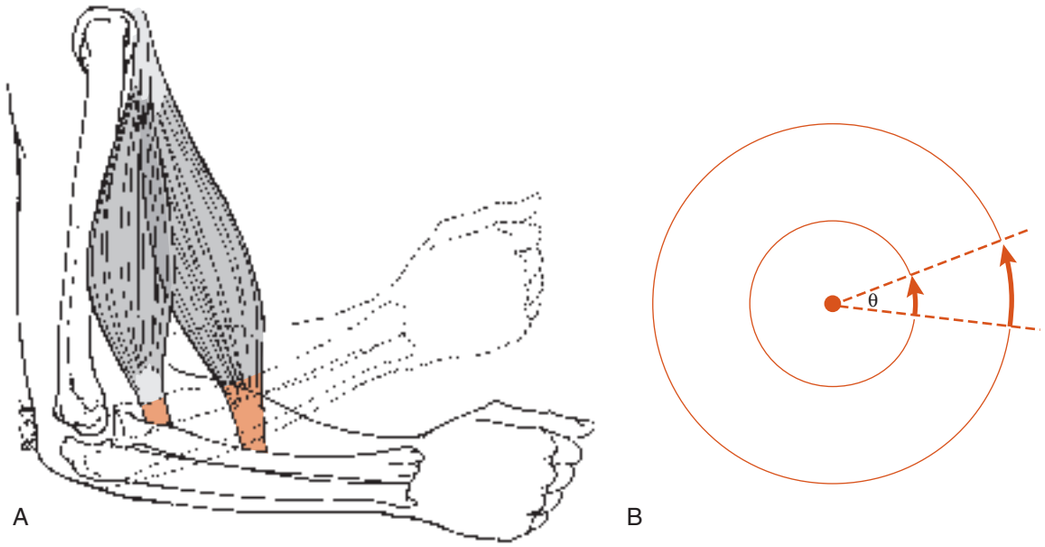

Figure 4.9: The relationship between a muscle's moment arm and excursion. The length of a muscle's moment arm affects the excursion that results from a contraction. **A.** Movement through an angle, θ, requires more shortening in a muscle with a long moment arm than in a muscle with a short moment arm.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

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.

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 [76,77].

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 [62]. Others, like the brachioradialis muscle at the elbow, combine relatively long muscle fibers with large moment arms [89]. 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. The ratio of a muscle's fiber length to its moment arm is a useful descriptor of a muscle's ability to produce an excursion and its torque-generating capability [99]. This ratio helps surgeons determine appropriate donor muscles to replace dysfunctional ones.

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 [15,16]. 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 [155].*

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:

$$\mathbf{M} = \mathbf{r} \times \mathbf{F} \qquad \text{(Equation 4.1)}$$

where **M** is the moment generated by the muscle's tensile force **(F)** applied at a distance **(r,** the muscle's moment arm) from the point of rotation (the joint axis). 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, **r** [54]. 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
- Level of muscle fiber recruitment
- Fiber types composing the muscle

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 person 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, which are contractions that produce no discernable joint motion. 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 [1,39]. 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 [67,126]. In fact, muscle size is the most important single factor determining the tensile force generated by a muscle's contraction [44,60]. Estimates of the maximal contractile force per unit of muscle range from approximately 20 to 135 N/cm^2 [15,22, 120,155]. 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 [15]. In a parallel fiber muscle the PCSA is approximately equal to the anatomical cross-sectional area. 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 PCSA. Thus if all other factors are equal, the pennate muscle is capable of generating more contraction force than the muscle with parallel fibers [64,90,114].

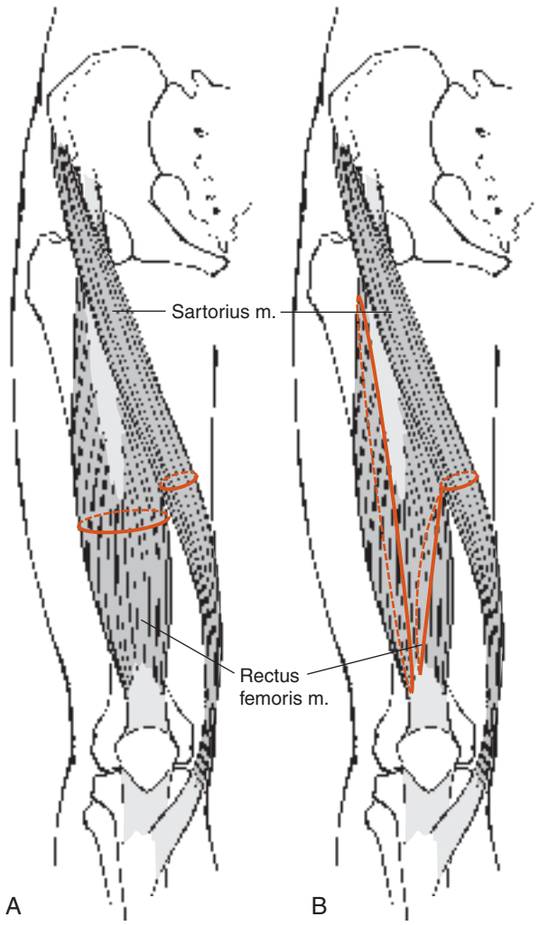

Figure 4.10: The relationship between muscle architecture and muscle size. **A.** The anatomical cross-sectional area of a muscle is the area of a slice through the widest part of the muscle perpendicular to the muscle's length. It is similar in a parallel fiber muscle and a pennate muscle of similar 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.

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. However, the larger the pennation angle is, the larger the PCSA is [2]. In most muscles the pennation angle is 30° or less, and thus pennation typically increases the tensile force produced by contraction [86,146]. Resistance training increases the fibers' angle of pennation (and the muscle's PCSA). This increase appears to result from increases, or hypertrophy, in the cross-sectional area of individual muscle fibers [2,13].

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 [27,64,119,145]. 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

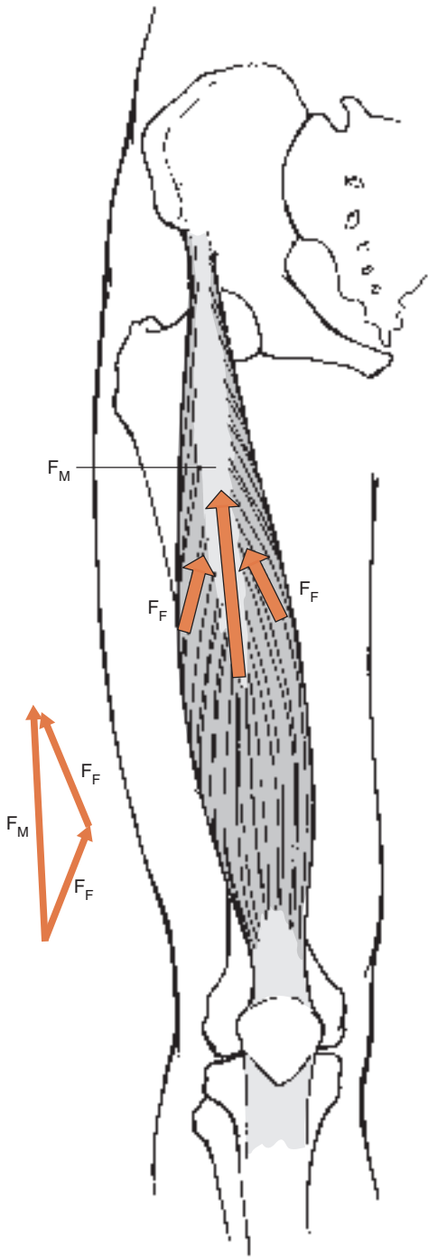

Figure 4.11: The pull of a pennate muscle. The overall tensile force (F_M) of a muscle is the vector sum of the force of contraction of the pennate fibers (F_F).

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 [34,50,125] (*Fig. 4.12*). This length is operationally defined as the **resting length** of the

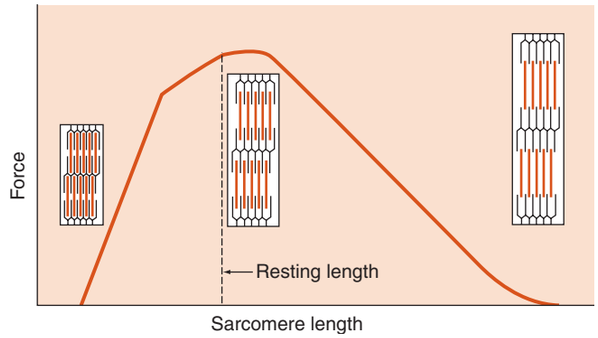

Figure 4.12: The length–tension curve of a sarcomere. The length–tension curve of a sarcomere demonstrates how the length of the sarcomere influences its force production.

muscle. The sarcomere can shorten slightly from this point, 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's 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 [43,45,53,121]. 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 [43,45, 88,90,121]. 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 contractile element in series and in parallel with the 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

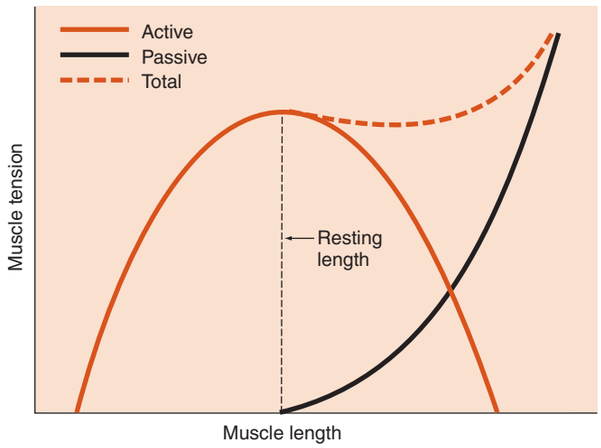

Figure 4.13: The length–tension curve of a whole muscle. The length–tension curve of a whole muscle demonstrates how muscle length affects the force production of the whole muscle. The contractile, or active, component; the passive component primarily due to the connective tissue; and the total muscle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

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 midregion 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 [45,152]. 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 [45]. 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 [56,123].

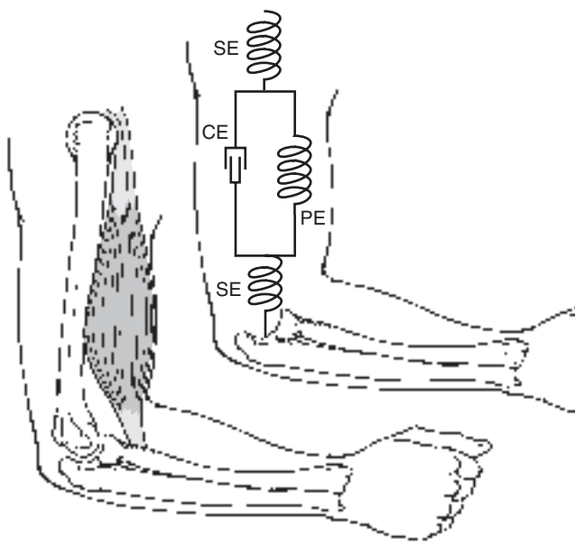

Figure 4.14: A mechanical model of the contractile and elastic components of a muscle. A muscle's contractile (actin and myosin) and elastic (connective tissue) components are often modeled mechanically as a combination of a contractile element (CE) with springs that represent the elastic elements that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

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.*

The classic length–tension relationship described so far has been studied by altering the length of a muscle passively and then assessing the strength of contraction at the new length. More recent studies have investigated the effects of

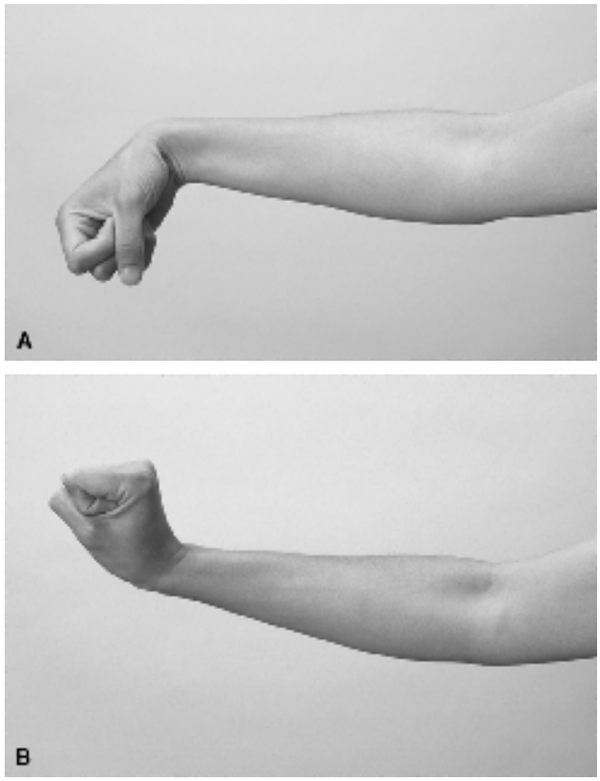

Figure 4.15: The effects of muscle length on performance. **A.** When the wrist is in flexion it is difficult to flex the fingers fully because the finger flexors are so shortened. **B.** When the wrist is in extension, the fingers readily flex to make a fist since the finger flexors are lengthened.

length changes on isometric strength while the muscle is actively contracting. These studies consistently demonstrate that the traditional length-tension relationships are amplified if the length changes occur during contraction. Specifically, if a contracting muscle is lengthened and then held at its lengthened position, the force generated at the lengthened position is greater than the strength measured at that same position with no preceding length change [55,128]. Similarly, shortening a muscle as it contracts produces more strength reduction than placing the relaxed muscle at the shortened position and then measuring its strength [124,128]. Many vigorous functional activities occur utilizing muscle contractions that consist of a lengthening then shortening contraction cycle [102]. Such a pattern of muscle activity appears to utilize the length–tension relationship to optimize a muscle's ability to generate force.

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.

Clinical Relevance

STRETCH-SHORTENING CYCLE OF MUSCLE CONTRACTION IN SPORTS: *The strength enhancement that comes from lengthening a contracting muscle prior to using it to produce motion is visible in countless activities, particularly in sports. For example the wind-up that precedes a throw or the backswing of a golf swing serves to stretch the muscles that will throw the ball or swing the golf club. The shoulder medial rotators are stretched prior to the forward motion of the throw, and the shoulder abductors and lateral rotators of the left arm are stretched prior to the forward motion of the golf swing for a right-handed golfer. Similarly the start of a running sprint event is characterized by a brief stretch of the plantar flexors, knee extensors and hip extensors before these same muscles shorten to push the runner down the track. The stretch of all of these muscles occurs as they are contracting and consequently amplifies even more the strength gains resulting from the stretch itself. (See the jumping activity in Chapter 4 laboratory.)*

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 [125]. 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 muscle's moment arm is maximum when the muscle's angle of application is 90°, since the sine of 90° equals 1. 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 very little change (*Fig. 4.18*) [57,70,71,81, 113,135,151]. Therefore, a muscle's ability to produce a moment varies with the joint position.

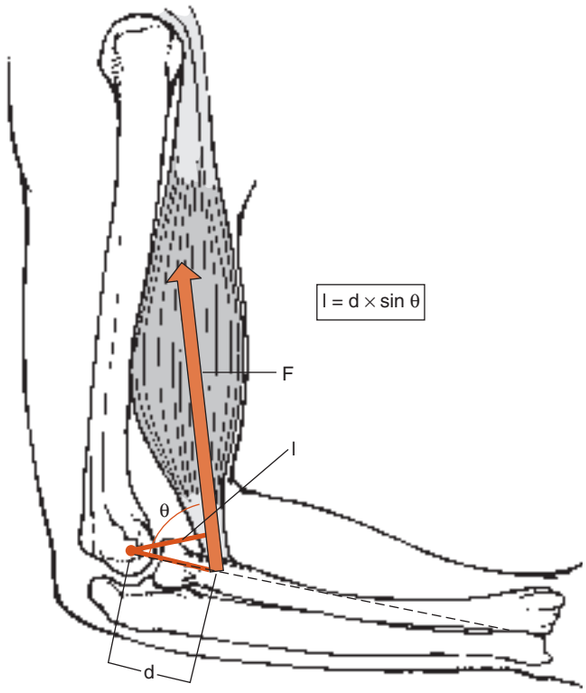

Figure 4.16: Moment arm of a muscle. A muscle's moment arm (l) is easily calculated using the muscle's angle of application (θ) and the distance (d) from the muscle attachment to the axis of rotation.

$$l = d \times \sin \theta$$

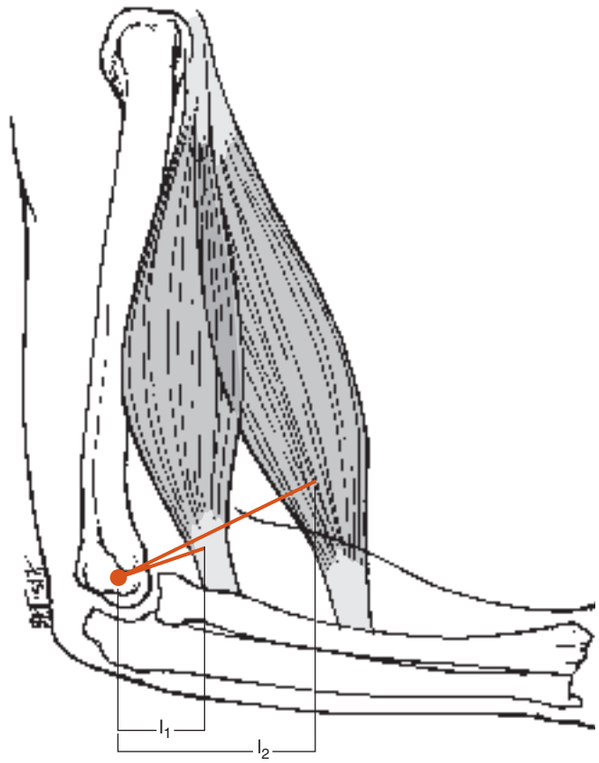

Figure 4.17: The effect of moment arm of a muscle on the muscle's performance. A muscle with a short moment arm (l_1) generates a smaller moment than a muscle with a longer moment arm (l_2) that generates the same contraction force.

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 [4,113]. 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 [62,82, 87,100,112,148].

In a series of elegant experiments Lieber and colleagues assessed 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 [88,94,95]. 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 their 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.

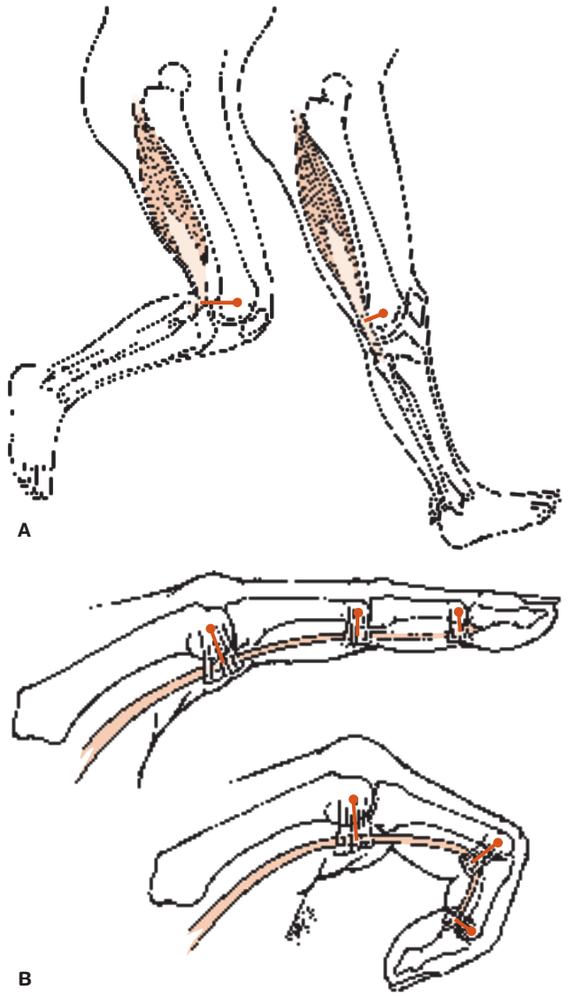

Figure 4.18: Changes in muscle moment arms. **A.** The hamstrings' moment arm at the knee is small with the knee extended and much larger with the knee flexed. **B.** The moment arm of a tendon of the flexor digitorum profundus at the finger changes little with the fingers extended or flexed.

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. Research demonstrates that the hip flexors are strongest with the hip close to extension where the muscles are in a lengthened position (Chapter 39). It is noteworthy to recognize that in this position the angle of application is relatively small as well, suggesting that muscle length is a larger influence on hip flexion strength than is angle of application.

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 contrast, a **concentric contraction,** also known as a **shortening contraction,** is defined as a contraction in which there is visible shortening of the muscle [37]. Thus a concentric contraction has a positive contraction velocity.

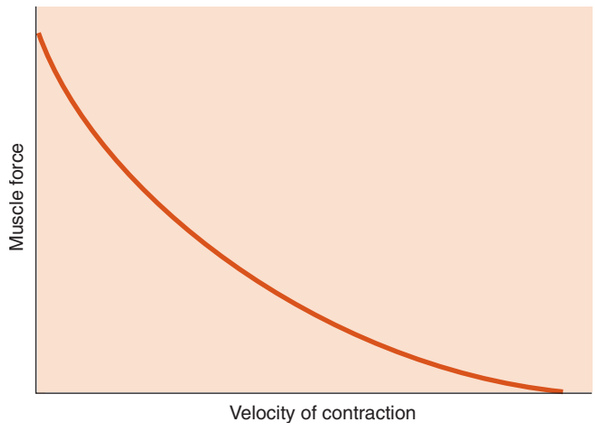

Figure 4.19: The relationship between contractile force and the velocity of contraction in isometric and concentric contractions. A plot of contractile force and the velocity of contraction from isometric (F_I) to concentric contractions shows that the strength of the contraction decreases with increasing contractile velocity.

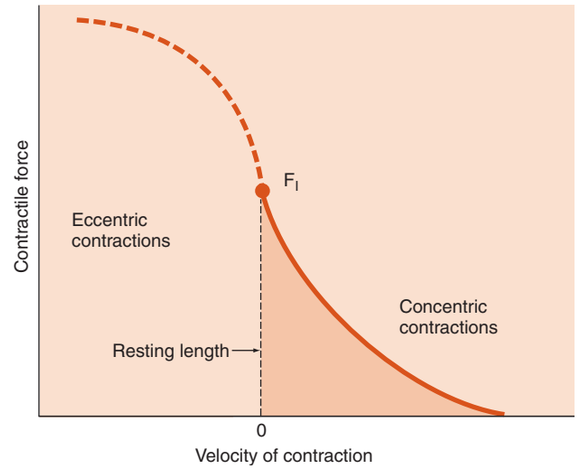

Figure 4.20: The relationship between contractile force and the velocity of contraction in isometric, concentric, and eccentric contractions. A plot of contractile force and the velocity of contraction from eccentric to concentric contractions shows that an eccentric contraction is stronger than either isometric (F_1) or concentric contractions.

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 [36,38,68,75,122,141,147]. 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. 4.19*). 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 [59]. 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 [74,118]. 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.*

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 [28,36,46,58,61,78,80,117,127,132,140,154] (*Fig. 4.20*). Maximum eccentric strength is estimated to be between 1.5 and 2.0 times maximum concentric strength [127,144]. 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 [28,36,91].

Clinical Relevance

POST-EXERCISE MUSCLE SORENESS: *Studies indicate that delayed-onset muscle soreness (DOMS) typically is associated with exercise using resisted eccentric exercise [11,40]. Although this phenomenon has not been thoroughly explained, one possible explanation is that a muscle*

(continued)

(Continued)
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 [75,79] (*Fig. 4.21*).

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 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.

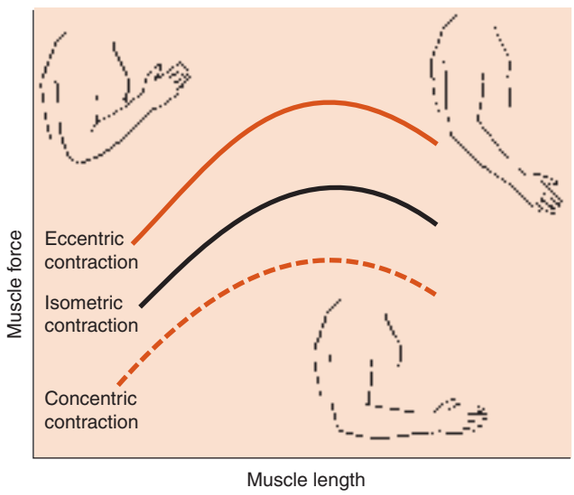

Figure 4.21: Comparison of eccentric, isometric, and concentric muscle strengths with changing muscle length. A comparison of eccentric, isometric, and concentric muscle strengths through an ROM reveals that the force of an eccentric contraction is greater than the force of an isometric contraction, which is greater than the force of a concentric contraction, regardless of the length of the muscle.

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 [24,30,31,78,130,136,137,142]. 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 [8,10,12,26,134]. 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 [78,127]. In the case of maximal contractions, the muscle recruits approximately the same number of motor units regardless of the output. The magnitude of the force output from concentric and eccentric contractions 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 [65]. 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.*

Maximal contractions are assumed to activate all of the motor units of the muscle. In young healthy adults this appears to be the case; that is they can typically activate 98–100% of the available motor units [103]. In contrast individuals who have pain or who are chronically inactive may be unable to fully activate the muscle, even though they are attempting to perform a maximal voluntary contraction (MVC) and appear to have an intact neuromuscular system [63,106,138]. These individuals exhibit **activation failure** in which, despite their best efforts and in the presence of intact muscles and nerves, they are unable to recruit all of the available motor units of the muscle. It is important that the clinician be able to determine if muscle weakness is the result of morphological changes in the muscles or nerves or activation failure.

Clinical Relevance

ACTIVATION FAILURE IN INDIVIDUALS WITH OSTEOARTHRITIS: *Individuals with either hip or knee osteoarthritis exhibit activation failure in the involved joints [63,138]. This failure is also described as* ***arthrogenic inhibition,*** *suggesting, that joint pain inhibits full muscle activation. Yet similar activation failure is found in individuals 1 year following total knee replacements when pain is no longer a complaint. Traditional exercises appear to have little effect on the activation failure, but more dynamic functional exercises have produced improved recruitment in patients with knee osteoarthritis [63]. Neuromuscular electrical stimulation also reduces activation failure. Identifying activation failure as a cause of muscle weakness may alter the intervention strategies used to improve 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 [66,109]. 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 [9,30,108].

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 [8]. 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 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 [153]. 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 [14]. 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 [110]. 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 [105,107].

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

The velocity of contraction also differs among fiber types [3,14]. 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 [14]. 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 [1,133]. However, as already noted, human muscles contain a mixture of fiber types [32,33,104,107]. 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 [25]. 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 noncontractile 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 [48,49,139, 150,153]. 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 [153].

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 [86,96].

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 [48,139,153]. 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 [77]. 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 [86].

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 [86,125,139].

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 [69]. 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 [23,116]. 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 [18,72,83,119,145]. 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 [25,27,61,97,101,115]. In addition, animal studies reveal that protein synthesis is consistent with a transition from type IIb fibers to type I fibers [6,86].

In contrast, decreased activity produces a decrease in CSA and loss of strength [47,85,115]. 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 [9]! 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 [5,6,96].

Although the preceding discussion demonstrates general patterns of muscle response to changes in activity level, the response is actually quite muscle dependent [85,86]. 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 [85]. Animal studies show similar differences among muscle groups [19,86]. Other mechanical factors such as stretch also affect a muscle's response to reduced activity [96].

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.*

Astronauts and cosmonauts experience a unique case of disuse atrophy resulting from their time spent in a microgravity environment. As with bed rest, microgravity induces atrophy of both Type I and Type II with evidence of a transition toward more Type II fibers [29]. Motor unit recruitment also appears altered. The resulting muscle weakness and decreased muscular endurance present significant challenges for protracted space travel and particularly for re-entry to earth's gravitational field.

Clinical Relevance

EXERCISING IN SPACE: *The International Space Lab is designed to allow prolonged stays in space and may serve as an intermediate stop for travelers to farther locations*

(continued)

(Continued)
such as Mars. However, unless these space travelers can exercise sufficiently to prevent the loss of muscle function that currently accompanies space travel, travel to outer space will remain limited to a select few individuals who can tolerate these changes. Exercise and rehabilitation experts who devise exercise equipment and regimens for microgravity use will find a very interested audience at the National Aeronautics and Space Agency (NASA) and other international space agencies.

AGING AS ANOTHER MODEL OF ALTERED ACTIVITY

Loss of strength is a well-established finding in aging adults [17,83,93,98,119,131]. This loss of strength is attributed to a decreased percentage of, and greater atrophy in, type II fibers [73,84,129]. As in the other adaptations of muscle described above, changes in muscle with age vary across muscles [20]. 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 [21,23,51]. Similarly, increasing strength in elders improves functional ability [35,111]. 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.

References

1. Aagaard P, Andersen JL: Correlation between contractile strength and myosin heavy chain isoform composition in human skeletal muscle. Med Sci Sports Exerc 1998; 30: 1217–1222.
2. Aagaard P, Andersen JL, Dyhre-Poulsen P, et al.: A mechanism for increased contractile strength of human pennate muscle in response to strength training: changes in muscle architecture. J Physiol 2001; 534: 613–623.
3. Adam C, Eckstein F, Milz S, Putz R: The distribution of cartilage thickness within the joints of the lower limb of elderly individuals. J Anat 1998; 193: 203–214.
4. An KN, Kaufman KR, Chao EYS: Physiological considerations of muscle force through the elbow joint. J Biomech 1989; 22: 1249–1256.
5. Andersen JL, Gruschy-Knudsen T, Sabdri C, et al.: Bed rest increases the amount of mismatched fibers in human skeletal muscle. J Appl Physiol 1999; 86: 455–460.
6. Baldwin KM: Effects of altered loading states on muscle plasticity: what have we learned from rodents? Med Sci Sports Exerc 1996; 28: S101–S106.
7. Baldwin KM, Valdez V, Herrick RE, et al.: Biochemical properties of overloaded fast-twitch skeletal muscle. J Appl Physiol 1982; 52: 467–472.
8. Basmajian JV, DeLuca CJ: Muscles Alive. Their Function Revealed by Electromyography. Baltimore: Williams & Wilkins, 1985.
9. Berg HE, Tesch PA: Changes in muscle function in response to 10 days of lower limb unloading in humans. Acta Physiol Scand 1996; 157: 63–70.
10. Bergstrom RM: The relation between the number of impulses and the integrated electrical activity in electromyogram. Acta Physiol Scand 1959; 45: 97–101.
11. Berry CB, Moritani T, Tolson H: Electrical activity and soreness in muscles after exercise. Am J Phys Med Rehabil 1990; 69: 60–66.
12. Bigland B, Lippold OCJ: The relation between force, velocity and integrated electrical activity in human muscles. J Physiol 1954; 123: 214–224.
13. Blazevich AJ, Gill ND, Deans N, Zhou S: Lack of human muscle architectural adaptation after short-term strength training. Muscle Nerve 2007; 35: 78–86.
14. Bottinelli R, Pellegrino MA, Canepari M, et al.: Specific contributions of various muscle fibre types to human muscle performance: an in vitro study. J Electromyogr Kinesiol 1999; 9: 87–95.
15. Brand PW, Beach RB, Thompson DE: Relative tension and potential excursion of muscles in the forearm and hand. J Hand Surg [Am]. 1999; 6: 209–219.
16. Brand PW, Hollister A: Clinical Mechanics of the Hand. St. Louis, MO: Mosby-Year Book, 1993.

17. Brown DA, Miller WC: Normative data for strength and flexibility of women throughout life. Eur J Appl Physiol 1998; 78: 77–82.
18. Brown M: Exercising and elderly person. Phys Ther Pract 1992; 1: 34–42.
19. Brown M, Hasser EM: Weight-bearing effects on skeletal muscle during and after simulated bed rest. Arch Phys Med Rehabil 1995; 76: 541–546.
20. Brown M, Hasser EM: Complexity of age-related change in skeletal muscle. J Gerontol 1996; 51A: B117–B123.
21. Brown M, Sinacore D, Host H: The relationship of strength to function in the older adult. J Gerontol 1995; 50A: 55–59.
22. Buchanan TS: Evidence that maximum muscle stress is not a constant: differences in specific tension in elbow flexors and extensors. Med Eng Phys 1995; 17: 529–536.
23. Buchner DM, Beresford SAA, Larson EB, et al.: Effects of physical activity on health status in older adults II: intervention studies. Annu Rev Public Health 1992; 13: 469–488.
24. Clancy EA, Hogan N: Relating agonist-antagonist electromyograms to joint torque during isometric, quasi-isotonic, nonfatiguing contractions. IEEE Trans Biomed Eng 1997; 44: 1024–1028.
25. Clarkson PM, Kroll W, Melchionda AM: Isokinetic strength, endurance, and fiber type composition in elite American paddlers. Eur J Appl Physiol 1982; 48: 67–76.
26. Coburn JW, Housh TJ, Malek MH, et al.: Mechanomyographic and electromyographic responses to eccentric muscle contractions. Muscle Nerve 2006; 33: 664–671.
27. Cress NM, Conley KE, Balding SL, et al.: Functional training: muscle structure, function and performance in older women. J Orthop Sports Phys Ther 1996; 24: 4–10.
28. Cress NM, Peters KS, Chandler JM: Eccentric and concentric force-velocity relationships of the quadriceps femoris muscle. J Orthop Sports Phys Ther 1992; 16: 82–86.
29. di Prampero PE, Narici MV: Muscles in microgravity: from fibres to human motion. J Biomech 2003; 36: 403–412.
30. Dolan P, Adams MA: The relationship between EMG activity and extensor moment generation in the erector spinae muscles during bending and lifting activities. J Biomech 1993; 26: 513–522.
31. Dolan P, Kingman I, DeLooze MP, et al: An EMG technique for measuring spinal loading during asymmetric lifting. Clin Biomech 2001; 16 Suppl: S17–S24.
32. Edgerton VR, Smith JL, Simpson DR: Muscle fibre type populations of human leg muscles. Histochem J 1975; 7: 259–266.
33. Elder GCB, Bradbury K, Roberts R: Variability of fiber type distributions within human muscles. J Appl Physiol 1982; 53: 1473–1480.
34. Elftman H: Biomechanics of muscle, with particular application to studies of gait. J Bone Joint Surg [AM] 1966; 48: 363–377.
35. Evans WJ: Exercise training guidelines for the elderly. Med Sci Sports Exerc 1999; 31: 12–17.
36. Evetovich TK, Housh TJ, Johnson GO, et al.: Gender comparisons of the mechanomyographic responses to maximal concentric and eccentric isokinetic muscle actions. Med Sci Sports Exerc 1998; 30: 1697–1702.
37. Faulkner JA: Terminology for contractions of muscles during shortening, while isometric, and during lengthening. J Appl Physiol 2003; 95: 455–459.
38. Fenn WO, Marsh BS: Muscular force at different speed of shortening. Proc R Soc B [Lond] 1998; 277–297.
39. Fitts RH, McDonald KS, Schluter JM: The determinants of skeletal muscle force and power: their adaptability with changes in activity pattern. J Biomech 1991; 24: 111–122.
40. Fitzgerald GK, Rothstein JM, Mayhew TP, Lamb RL: Exercise-induced muscle soreness after concentric and eccentric isokinetic contractions. Phys Ther 1991; 71: 505–513.
41. Foss ML, Keteyian SJ: Fox's physiological basis of exercise and sport. Boston: WCB/McGraw-Hill, 1998.
42. Fukunaga T, Kawakami Y, Kuno S, et al.: Muscle architecture and function in humans. J Biomech 1997; 30: 457–463.
43. Gandevia SC, McKenzie DK: Activation of human muscles at short muscle lengths during maximal static efforts. J Physiol [Lond] 1988; 407: 599–613.
44. Gans C: Fiber architecture and muscle function. Exerc Sports Sci Rev 1982; 10: 160–207.
45. Gareis H, Solomonow M, Baratta R, et al.: The isometric length-force models of nine different skeletal muscles. J Biomech 1992; 25: 903–916.
46. Ghena DR, Kurth AL, Thomas M, Mayhew J: Torque characteristics of the quadriceps and hamstring muscles during concentric and eccentric loading. J Orthop Sports Phys Ther 1991; 14: 149–154.
47. Gogia PP, Schneider VS, LeBlanc AD, et al.: Bed rest effect on extremity muscle torque in healthy men. Arch Phys Med Rehabil 1988; 69: 1030–1032.
48. Goldspink G: The influence of immobilization and stretch in protein turnover of rat skeletal muscle. J Physiol 1977; 264: 267–282.
49. Goldspink G: Changes in muscle mass and phenotype and the expression of autocrine and systemic growth factors by muscle in response to stretch and overload. J Anat 1999; 194: 323–334.
50. Gordon AM, Huxley AF, Julian FJ: The variation in isometric tension with sarcomere length in vertebrate muscle fibres. J Physiol 1966; 184: 170–192.
51. Graafmans WC, Ooms ME, Hofstee HMA, et al.: Falls in the elderly: a prospective study of the risk factors and risk profiles. Am J Epidemiol 1996; 143: 1129–1136.
52. Harms-Ringdahl K: Muscle strength. Edinburgh: Churchill Livingstone, 1993.
53. Hawkins D, Bey M: Muscle and tendon force-length properties and their interactions in vivo. J Biomech 1997; 30: 63–70.
54. Hawkins DA, Hull ML: A computer simulation of muscle-tendon mechanics. Comput Biol Med 1991; 21: 369–382.
55. Herzog W, Leonard TR: The role of passive structures in force enhancement of skeletal muscles following active stretch. J Biomech 2005; 38: 409–415.
56. Herzog W, Leonard TR, Renaud JM, et al.: Force-length properties and functional demands of cat gastrocnemius, soleus, and plantaris muscles. J Biomech 1992; 25: 1329–1335.
57. Herzog W, Read LJ: Lines of action and moment arms of the major force-carrying structures crossing the human knee joint. J Anat 1993; 182: 213–230.
58. Higbie EJ, Cureton KJ, Warren GLI, Prior BM: Effects of concentric and eccentric training on muscle strength, cross-sectional area, and neural activation. J Appl Physiol 1996; 81: 2173–2181.
59. Hislop HJ, Montgomery J: Daniel's and Worthingham's Muscle Testing: Techniques of Manual Examination. Philadelphia: WB Saunders, 1995.

60. Holzbaur KR, Delp SL, Gold GE, Murray WM: Moment-generating capacity of upper limb muscles in healthy adults. J Biomech 2007; 40(4): 742–749.
61. Hortobagyi T, Hill JP, Houmard JA, et al.: Adaptive responses to muscle lengthening and shortening in humans. J Appl Physiol 1996; 80: 765–772.
62. Hoy MG, Zajac FE: A musculoskeletal model of the human lower extremity: the effect of muscle, tendon, and moment arm on the moment-angle relationship of musculotendon actuators at the hip, knee, and ankle. J Biomech 1990; 23: 157–169.
63. Hurley MV, Newham DJ: The influence of arthrogenous muscle inhibition on quadriceps rehabilitation of patients with early, unilateral osteoarthritic knees. Br J Rheumatol 1993; 32: 127–131.
64. Ichinose Y: Relationship between muscle fiber pennation and force generation capability in Olympic athletes. Int J Sports Med 1998; 19: 541–546.
65. Iddings DM, Smith LK, Spencer WA: Muscle testing: Part 2. Reliability in clinical use. Phys Ther Rev 1960; 41: 249–256.
66. Inman VT, Ralston HJ, Saunders JBDM, et al.: Relation of human electromyogram to muscular tension. Electroenceph Clin Neurophysiol 1952; 4: 187–194.
67. Kanehisa H, Ikegawa S, Fukunaga T: Force-velocity relationships and fatigability of strength and endurance-trained subjects. Int J Sports Med. 1997; 18: 106–112.
68. Katz B: The relation between force and speed in muscular contraction. J Physiol 1939; 96: 45–64.
69. Kendall FP, McCreary EK, Provance PG: Muscle Testing and Function. Baltimore: Williams & Wilkins, 1993.
70. Ketchum LD, Thompson D, Pocock G, Wallingford D: A clinical study of forces generated by the intrinsic muscles of the index finger and the extrinsic flexor and extensor muscles of the hand. J Hand Surg [Am]. 1978; 3: 571–578.
71. Klein P, Mattys S, Rooze M: Moment arm length variations of selected muscles acting on talocrural and subtalar joints during movement: an in vitro study. J Biomech 1996; 29: 21–30.
72. Klinge K, Magnusson SP, Simonsen EB, et al.: The effect of strength and flexibility training on skeletal muscle electromyographic activity, stiffness, and viscoelastic stress relaxation response. Am J Sports Med. 1997; 25: 710–716.
73. Klitgaard H, Mantoni M, Schiaffino S, et al.: Function, morphology and protein expression of ageing skeletal muscle: a cross-sectional study of elderly men with different training backgrounds. Acta Physiol Scand. 1990; 140: 41–54.
74. Knapik JJ: Isokinetic, isometric and isotonic strength relationships. Arch Phys Med Rehabil 1983; 64: 77–80.
75. Knapik JJ, Wright JE, Mawdsley RH, Braun J: Isometric, isotonic, and isokinetic torque variations in four muscle groups through a range of joint motion. Phys Ther 1983; 63: 938–947.
76. Koh TJ, Herzog W: Increasing the moment arm of the tibialis anterior induces structural and functional adaptation: implications for tendon transfer. J Biomech 1998; 31: 593–599.
77. Koh TJ, Herzog W: Excursion is important in regulating sarcomere number in the growing rabbit tibialis anterior. J Physiol [Lond] 1998; 508: 267–280.
78. Komi PV: Relationship between muscle tension, EMG, and velocity of contraction under concentric and eccentric work. In: New Developments in Electromyography and Clinical Neurophysiology. Desmedt JE, ed. Basel: Karger, 1973; 596–606.
79. Komi PV: Measurement of the force-velocity relationship in human muscle under concentric and eccentric contractions. Biomechanics III 1979; 224–229.
80. Krylow AM, Sandercock TG: Dynamic force responses of muscle involving eccentric contraction. J Biomech 1997; 30: 27–33.
81. Kuechle DK, Newman SR, Itoi E, et al.: Shoulder muscle moment arms during horizontal flexion and elevation. J Shoulder Elbow Surg 1997; 6: 429–439.
82. Kulig K, Andrews JG, Hay JG: Human strength curves. Exerc Sports Sci Rev 1984; 12: 417–466.
83. Laforest S, St-Peirre DMM, Cyr J, Gayton D: Effects of age and regular exercise on muscle strength and endurance. Eur J Appl Physiol 1990; 60: 104–111.
84. Larsson L, Grimby G, Karlsson J: Muscle strength and speed of movement in relation to age and muscle morphology. J Appl Physiol 1979; 46: 451–456.
85. LeBlanc A, Gogia P, Schneider VS, et al.: Calf muscle area and strength changes after 5 weeks of horizontal bed rest. Am J Sports Med 1988; 16: 624–629.
86. Lieber RL: Skeletal Muscle Structure and Function: Implications for Rehabilitation and Sports Medicine. Baltimore: Williams & Wilkins, 1992.
87. Lieber RL, Boakes JL: Sarcomere length and joint kinematics during torque production in frog hindlimb. Am J Phys 1988; 254: C759–C768.
88. Lieber RL, Friden J: Musculoskeletal balance of the human wrist elucidated using intraoperative laser diffraction. J Electromyogr Kinesiol 1998; 8: 93–100.
89. Lieber RL, Jacobson MD, Fazeli BM, et al.: Architecture of selected muscles of the arm and forearm: anatomy and implications for tendon transfer. J Hand Surg [Am]. 1992; 17: 787–798.
90. Lieber RL, Ljung B, Friden J: Intraoperative sarcomere length measurements reveal differential design of human wrist extensor muscles. J Exp Biol 1997; 200: 19–25.
91. Lieber RL, Ljung B, Friden J: Sarcomere length in wrist extensor muscles, changes may provide insights into the etiology of chronic lateral epicondylitis. Acta Orthop Scand 1997; 68: 249–254.
92. Lieber RL, Loren GJ, Friden J: In vivo measurement of human wrist extensor muscle sarcomere length changes. J Neurophysiol. 1994; 71: 874–881.
93. Lindle RS, Metter EJ, Lynch NA, et al.: Age and gender comparisons of muscle strength in 654 women and men aged 20–93 yr. J Appl Physiol 1997; 83: 1581–1587.
94. Loren GJ, Lieber RL: Tendon biomechanical properties enhance human wrist muscle specialization. J Biomech 1995; 28: 791–799.
95. Loren GJ, Shoemaker SD, Burkholder TJ, et al.: Human wrist motors: biomechanical design and application to tendon transfers. J Biomech 1996; 29: 331–342.
96. Loughna PT: Disuse and passive stretch cause rapid alterations in expression of developmental and adult contractile protein genes in skeletal muscle. Development 1990; 109: 217–223.
97. MacDougall JD, Elder GCB, Sale DG, et al.: Effects of strength training and immobilization on human muscle fibers. J Appl Physiol 1980; 43: 25–34.
98. Maclennan WJ, Hall MPR, Timithy JI, Robinson M: Is weakness in old age due to muscle wasting? Age Ageing 1980; 9: 188–192.

99. Maganaris CN, Baltzopoulos V, Tsaopoulos D: Muscle fibre length-to-moment arm ratios in the human lower limb determined in vivo. J Biomech 2006; 39: 1663–1668.
100. Marshall RN, Mazur SM, Taylor NAS: Three-dimensional surfaces for human muscle kinetics. Eur J Appl Physiol 1990; 61: 263–270.
101. McCall GE, Byrnes WC, Dickinson A, et al.: Muscle fiber hypertrophy, hyperplasia, and capillary density in college men after resistance training. J Appl Physiol 1996; 81: 2004–2012.
102. Mero A, Kuitunen S, Harland M, et al.: Effects of muscle-tendon length on joint moment and power during sprint starts. J Sports Sci 2006; 24: 165–173.
103. Miller M, Holmback AM, Downham D, Lexell J: Voluntary activation and central activation failure in the knee extensors in young women and men. Scand J Med Sci Sports 2006; 16: 274–281.
104. Miller AEJ, MacDougall JD, Tarnopolsky MA, Sale DG: Gender differences in strength and muscle fiber characteristics. Eur J Appl Physiol. 1993; 66: 254–262.
105. Milner-Brown HS, Stein RB, Yemm R: The orderly recruitment of human motor units during voluntary isometric contractions. J Physiol 1973; 230: 359–370.
106. Mizner RL, Petterson SC, Stevens JE, et al.: Early quadriceps strength loss after total knee arthroplasty: the contributions of muscle atrophy and failure of voluntary muscle activation. J Bone Joint Surg Am 2005; 87: 1047–1053.
107. Mizuno M, Secher NH, Quistorff B: 31P-NMR spectroscopy, rsEMG, and histochemical fiber types of human wrist flexor muscles. J Appl Physiol. 1994; 76: 531–538.
108. Moritani T: 1998 ISEK Congress Keynote Lecture: The use of electromyography in applied physiology. International Society of Electrophysiology and Kinesiology. J Electromyogr Kinesiol. 1998; 8: 363–381.
109. Moritani T, DeVries HA: Reexamination of the relationship between the surface integrated electromyogram (IEMG) and force of isometric contraction. Am J Phys Med. 1978; 57: 263–277.
110. Moss CL: Comparison of the histochemical and contractile properties of human gastrocnemius muscle. J Orthop Sports Phys Ther 1991; 13: 322–327.
111. Mulrow CD, Gerety MB, Kanten D, et al.: A randomized trial of physical rehabilitation for very frail nursing home residents. JAMA 1994; 271: 519–524.
112. Murphy AJ, Wilson GJ, Pryor JF, Newton RU: Isometric assessment of muscular function: the effect of joint angle. J Appl Biomech 1995; 11: 205–214.
113. Murray WM, Delp SL, Buchanan TS: Variation of muscle moment arms with elbow and forearm position. J Biomech 1995; 28: 513–525.
114. Narici M: Human skeletal muscle architecture studied in vivo by non-invasive imaging techniques: functional significance and applications. J Electromyogr Kinesiol 1999; 9: 97–103.
115. Narici MV, Roi GS, Landoni L, et al.: Changes in force, cross-sectional area and neural activation during strength training and detraining of the human quadriceps. Eur J Appl Physiol. 1989; 59: 310–319.
116. Neumann DA, Soderberg GL, Cook TM: Comparison of maximal isometric hip abductor muscle torques between hip sides. Phys Ther 1988; 68: 496–502.
117. Olson VL, Smidt GL, Johnston RC: The maximum torque generated by the eccentric, isometric, and concentric contractions of the hip abductor muscles. Phys Ther 1972; 52: 149–158.
118. Osternig LR, Bates BT, James SL: Isokinetic and isometric torque force relationships. Arch Phys Med Rehabil 1977; 58: 254–257.
119. Payne VG, Morrow JR Jr, Johnson L, Dalton SN: Resistance training in children and youth: a meta-analysis. Res Q Exerc Sport 1997; 68: 80–88.
120. Powell PL, Roy RR, Kanim P, et al.: Predictability of skeletal muscle tension from architectural determinations in guinea pig hindlimbs. J Appl Physiol 1984; 57: 1715–1721.
121. Ralston HJ, Inman VT, Strait LA, Shaffrath MD: Mechanics of human isolated voluntary muscle. Am J Phys 1947; 151: 612–620.
122. Ralston HJ, Polissar MJ, Inman VT, et al.: Dynamic features of human isolated voluntary muscle in isometric and free contractions. J Appl Physiol 1949; 1: 526–533.
123. Raschke U, Chaffin DB: Support for a linear length-tension relation of the torso extensor muscles: an investigation of the length and velocity EMG-force relationships. J Biomech 1996; 29: 1597–1604.
124. Rassier DE, Herzog W: Effects of shortening on stretch-induced force enhancement in single skeletal muscle fibers. J Biomech 2004; 37: 1305–1312.
125. Rassier DE, MacIntosh BR, Herzog W: Length dependence of active force production in skeletal muscle. J Appl Physiol. 1999; 86: 1445–1457.
126. Raty HP, Kujala U, Videman T, et al.: Associations of isometric and isoinertial trunk muscle strength measurements and lumbar paraspinal muscle cross-sectional areas. J Spinal Disord 1999; 12: 266–270.
127. Rogers KL, Berger RA: Motor-unit involvement and tension during maximum voluntary concentric, eccentric and isometric contractions of elbow flexors. Med Sci Sports 1974; 6: 253–254.
128. Rousanoglou EN, Oskouei AE, Herzog W: Force depression following muscle shortening in sub-maximal voluntary contractions of human adductor pollicis. J Biomech 2007; 40: 1–8.
129. Scelsi R, Marchetti C, Poggi P: Histochemical and ultrastructural aspects of muscle vastus lateralis in sedentary old people (age 65–89). Acta Neuropathol 1980; 51: 99–105.
130. Seroussi RE, Pope MH: The relationship between trunk muscle electromyography and lifting moments in the sagittal and frontal planes. J Biomech 1987; 20: 135–146.
131. Shephard RJ, Sidney KH: Exercise and aging. Exerc Sports Sci Rev 1978; 6: 1–57.
132. Shklar A, Dvir Z: Isokinetic strength relationships in shoulder muscles. Clin Biomech 1995; 10: 369–373.
133. Sirca A, Kostevc V: The fibre type composition of thoracic and lumbar paravertebral muscles in man. J Anat 1985; 141: 131–137.
134. Solomonow M, Baratta R, Shoji H, D'Ambrosia R: The EMG-force relationships of skeletal muscle; dependence on contraction rate, and motor units control strategy. Electromyogr Clin Neurophysiol 1990; 30: 141–152.
135. Spoor C, Van Leeuwen J, Meskers C, et al.: Estimation of instantaneous moment arms of lower-leg muscles. J Biomech 1990; 23: 1247–1259.
136. Stokes IAF: Relationships of EMG to effort in the trunk under isometric conditions: force-increasing and decreasing effects and temporal delays. Clin Biomech 2005; 20: 9–15.

137. Stokes IAF, Moffroid M, Rush S, Haugh LD: EMG to torque relationship in rectus abdominis muscle: results with repeated testing. Spine 1989; 14: 857–861.
138. Suetta C, Aagaard P, Rosted A, et al.: Training-induced changes in muscle CSA, muscle strength, EMG, and rate of force development in elderly subjects after long-term unilateral disuse. J Appl Physiol 2004; 97: 1954–1961.
139. Tabary JC, Tabary C, Tardieu C, et al.: Physiological and structural changes in the cat's soleus muscle due to immobilization at different lengths by plaster casts. J Physiol 1972; 224: 231–244.
140. Tesch PA, Dudley GA, Duvoisin MR, et al.: Force and EMG signal patterns during repeated bouts of concentric or eccentric muscle actions. Acta Physiol Scand. 1990; 138: 263–271.
141. Thorstensson A, Grimby G, Karlsson J: Force-velocity relations and fiber composition in human knee extensor muscles. J Appl Physiol 1976; 40: 12–16.
142. Vogt R, Nix WA, Pfeifer B: Relationship between electrical and mechanical properties of motor units. J Neurol Neurosurg Psychiatry 1990; 53: 331–334.
143. Walker SM, Schrodt GR: I segment lengths and thin filament periods in skeletal muscle fibers of the rhesus monkey and human. Anat Rec 1974; 178: 63–81.
144. Webber S, Kriellaars D: Neuromuscular factors contributing to in vivo eccentric moment generation. J Appl Physiol. 1997; 83: 40–45.
145. Welle S, Totterman S, Thornton C: Effect of age on muscle hypertrophy induced by resistance training. J Gerontol A Biol Sci Med Sci 1996; 51: M270–M275
146. Wickiewicz TL, Roy RR, Powell PL, Edgerton VR: Muscle architecture of the human lower limb. Clin Orthop 1983; 179: 275–283.
147. Wilkie DR: The relation between force and velocity in human muscle. J Physiol 1950; 110: 249–286.
148. Williams M, Stutzman L: Strength variation through the range of joint motion. Phys Ther Rev 1959; 39: 145–152.
149. Williams P, Bannister L, Berry M, et al.: Gray's Anatomy, The Anatomical Basis of Medicine and Surgery, Br. ed. London: Churchill Livingstone, 1995.
150. Williams P, Kyberd P, Simpson H, et al.: The morphological basis of increased stiffness of rabbit tibialis anterior muscles during surgical limb-lengthening. J Anat 1998; 193: 131–138.
151. Wilson DL, Zhu Q, Duerk JL, et al.: Estimation of tendon moment arms from three-dimensional magnetic resonance images. Ann Biomed Eng 1999; 27: 247–256.
152. Winters J, Stark L, Seif-Naraghi A: An analysis of the sources of musculoskeletal system impedance. J Biomech 1988; 21: 12: 1011–1025.
153. Yang H, Alnaqeeb M, Simpson H, Goldspink G: Changes in muscle fibre type, muscle mass and IGF-I gene expression in rabbit skeletal muscle subjected to stretch. J Anat 1997; 190: 613–622.
154. Yeadon MR, King MA, Wilson C: Modelling the maximum voluntary joint torque/angular velocity relationship in human movement. J Biomech 2006; 39: 476–482.
155. Zajac FE: How musculotendon architecture and joint geometry affect the capacity of muscles to move and exert force on objects: a review with application to arm and forearm tendon transfer design. J Hand Surg [Am]. 1992; 17: 799–804.