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The Motor Unit and Muscle Action

Any action—ascending a flight of stairs, typing on a keyboard, even holding a pose—requires coordinating the movement of body parts. This is accomplished by the interaction of the nervous system with muscle. The role of the nervous system is to activate the muscles that provide the forces needed to move in a particular way. This is not a simple task. Not only must the nervous system decide which muscles to activate, how much to activate them, and the sequence in which they must be activated in order to move one part of the body, but it must also control the influence of the resultant muscle forces on other body parts and maintain the required posture.

This chapter examines how the nervous system controls muscle force and how the force exerted by a limb depends on muscle structure. We also describe how muscle activation changes to perform different types of movement.

The Motor Unit Is the Elementary Unit of Motor Control

A Motor Unit Consists of a Motor Neuron and Multiple Muscle Fibers

The nervous system controls muscle force with signals sent from motor neurons in the spinal cord or brain stem to the muscle fibers. A motor neuron and the muscle fibers it innervates are known as a motor unit, the basic functional unit by which the nervous system controls movement, a concept proposed by Charles Sherrington in 1925.

A typical muscle is controlled by a few hundred motor neurons whose cell bodies are clustered in a motor nucleus in the spinal cord or brain stem. The axon of each motor neuron exits the spinal cord through the ventral root or through a cranial nerve in the brain stem and runs in a peripheral nerve to the

muscle. When the axon reaches the muscle, it branches and innervates from a few to several thousand muscle fibers (Figure 31–1).

Once synaptic input depolarizes the membrane potential of a motor neuron above threshold, the neuron generates an action potential that is propagated along the axon to its terminals in the muscle. The action potential releases acetylcholine at the neuromuscular synapse, triggering an action potential at the sarcolemma of the muscle fiber (Chapter 12). A muscle fiber has electrical properties similar to those of a large-diameter, unmyelinated axon, and thus, action potentials propagate along the sarcolemma, although more slowly due to the higher capacitance of the fiber resulting from the transverse tubules (see Figure 31–9). Because the action potentials in all the muscle fibers of a motor unit occur at approximately the same time, they contribute to extracellular currents that sum to generate a field potential near the active muscle fibers.

Most muscle contractions involve the activation of many motor units, whose currents sum to produce signals (*compound action potentials*) that can be detected by electromyography. The electromyogram (EMG) is typically large and can be easily recorded with electrodes

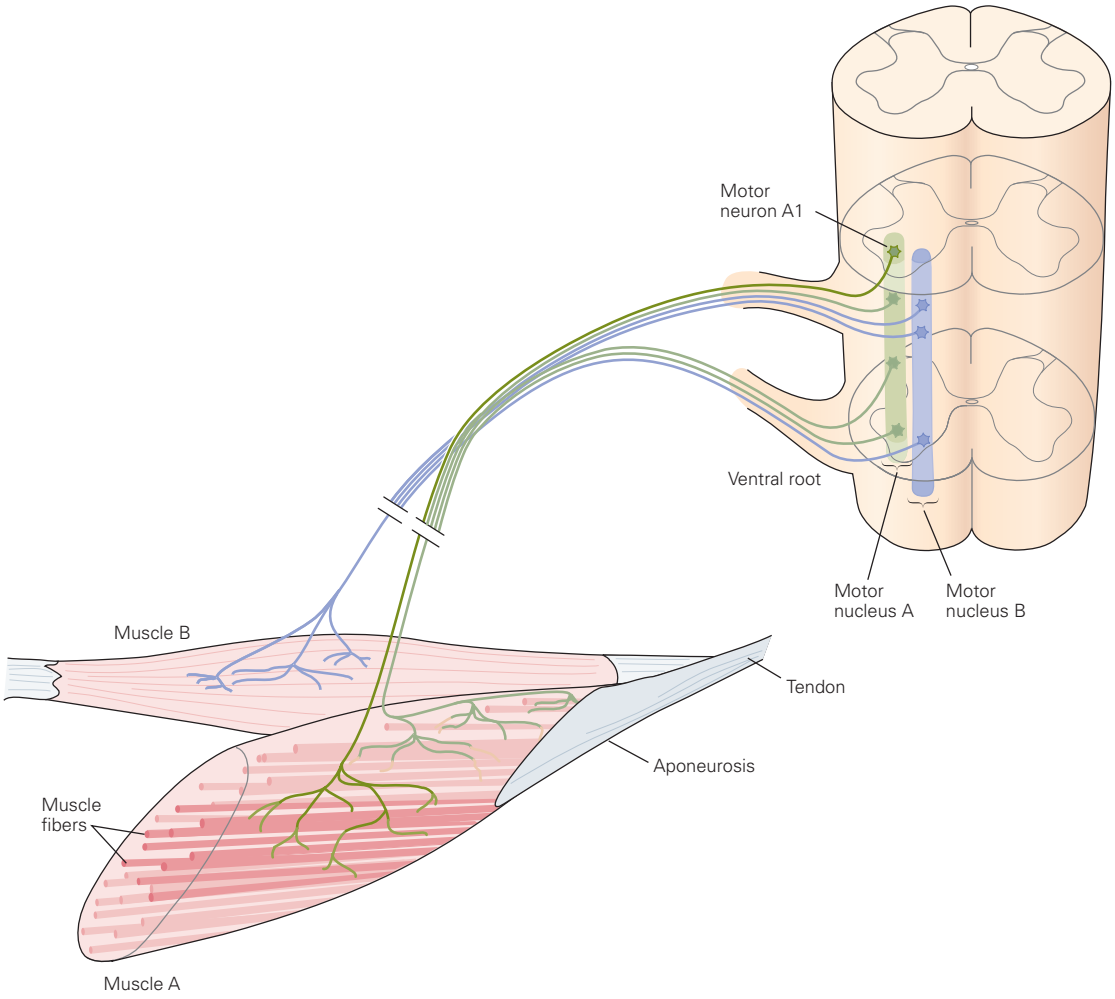

Figure 31–1 A typical muscle consists of many thousands of muscle fibers working in parallel and organized into a smaller number of motor units. A motor unit comprises a motor neuron and the muscle fibers it innervates, illustrated here by motor neuron A1. The motor neurons innervating one muscle are usually clustered into an elongated motor nucleus that may extend over one to four segments within the ventral spinal cord. The axons from a motor nucleus exit the spinal cord in several ventral roots and peripheral nerves but are collected into one nerve bundle near the target muscle. In the figure, motor nucleus A includes all those motor neurons innervating muscle A; likewise, motor nucleus B includes all the motor neurons that innervate muscle B. The extensively branched dendrites of each motor neuron (not shown in the figure) tend to intermingle with those of motor neurons from other nuclei.

placed on the skin over the muscle. The timing and amplitude of EMG activity, therefore, reflect the activation of muscle fibers by the motor neurons. EMG signals are useful for studying the neural control of movement and for diagnosing pathology (Chapter 57).

Each fiber in most mature vertebrate muscles is innervated by a single motor neuron. The number of muscle fibers innervated by one motor neuron, the *innervation number*, varies across muscles. In human skeletal muscles, the innervation number ranges from average values of 5 for an eye muscle to 1,800 for a leg muscle (Table 31–1). Because innervation number denotes the number of muscle fibers within a motor unit, differences in innervation number determine the differences in increments in force produced by activation of different motor units in the same muscle. Thus, the innervation number also indicates the fineness of control of the muscle at low forces; the smaller the innervation number, the finer the control achieved by varying the number of activated motor units.

The differences in innervation numbers between motor units in the same muscle can be substantial. For example, motor units of the first dorsal interosseous muscle of the hand have innervation numbers ranging from approximately 21 to 1,770. The strongest motor unit in the hand's first dorsal interosseous muscle can exert approximately the same force as the average motor unit in the leg's medial gastrocnemius muscle due to different ranges of innervation numbers in the two muscles.

Table 31–1 Innervation Numbers in Human Skeletal Muscles

Muscle	Alpha motor axons	Muscle fibers	Average innervation number
Biceps brachii	774	580,000	750
Brachioradialis	333	129,200	410
Cricothyroid	112	18,550	155
Gastrocnemius (medial)	579	1,042,000	1,800
Interossei dorsales (1)	119	40,500	340
Lumbricales (1)	96	10,269	107
Masseter	1,452	929,000	640
Opponens pollicis	133	79,000	595
Platysma	1,096	27,100	25
Posterior cricoarytenoid	140	16,200	116
Rectus lateralis	4,150	22,000	5
Temporalis	1,331	1,247,000	936
Tensor tympani	146	1,100	8
Tibialis anterior	445	272,850	613
Transverse arytenoid	139	34,470	247

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The muscle fibers of a single motor unit are distributed throughout the muscle and intermingle with fibers innervated by other motor neurons. The muscle fibers innervated by a single motor unit can be distributed across 8% to 75% of the volume in a limb muscle, with 2 to 5 muscle fibers belonging to the same motor unit among 100 muscle fibers. Therefore, the muscle fibers in a cross-section through the middle of an entire muscle are associated with 20 to 50 different motor units. This distribution and even the number of motor units change with age and with some neuromuscular disorders (Chapter 57). For example, muscle fibers that lose their innervation after the death of a motor neuron can be reinnervated by collateral sprouts from neighboring axons.

Some muscles comprise discrete compartments that are each innervated by a different primary branch of the muscle nerve. Branches of the median and ulnar nerves in the forearm, for example, innervate distinct compartments in three multitendon extrinsic hand muscles that enable the fingers to be moved relatively independently. The muscle fibers belonging to each motor unit in such muscles tend to be confined to one compartment. A muscle can therefore consist of several functionally distinct regions.

The Properties of Motor Units Vary

The force exerted by a muscle depends not only on the number of motor units that are activated during a contraction but also on three properties of motor units: contraction speed, maximal force, and fatigability. These properties are assessed by examining the force exerted by individual motor units in response to variations in the number and rate of evoked action potentials.

The mechanical response to a single action potential is known as a *twitch contraction.* The time it takes the twitch to reach its peak force, the *contraction time,* is one measure of the contraction speed of the muscle fibers that compose a motor unit. The motor units in a muscle typically exhibit a range of contraction times from slow to fast contracting. The mechanical response to a series of action potentials that produce overlapping twitches is known as a *tetanic contraction* or *tetanus.*

The force exerted during a tetanic contraction depends on the extent to which the twitches overlap

and summate (ie, the force varies with the contraction time of the motor unit and the rate at which the action potentials are evoked). At lower rates of stimulation, the ripples in the tetanus denote the peaks of individual twitches (Figure 31–2A). The peak force achieved during a tetanic contraction varies as a sigmoidal function of action potential rate, with the shape of the curve depending on the contraction time of the motor unit (Figure 31–2B). Maximal force is reached at lower action potential rates for slow-contracting motor units than the rates needed to achieve maximal force in fast-contracting units.

The functional properties of motor units vary across the population and between muscles. At one end of the distribution, motor units have long twitch contraction times and produce small forces, but are less fatigable. At the other end of the distribution, motor units have short contraction times, produce large forces, and are

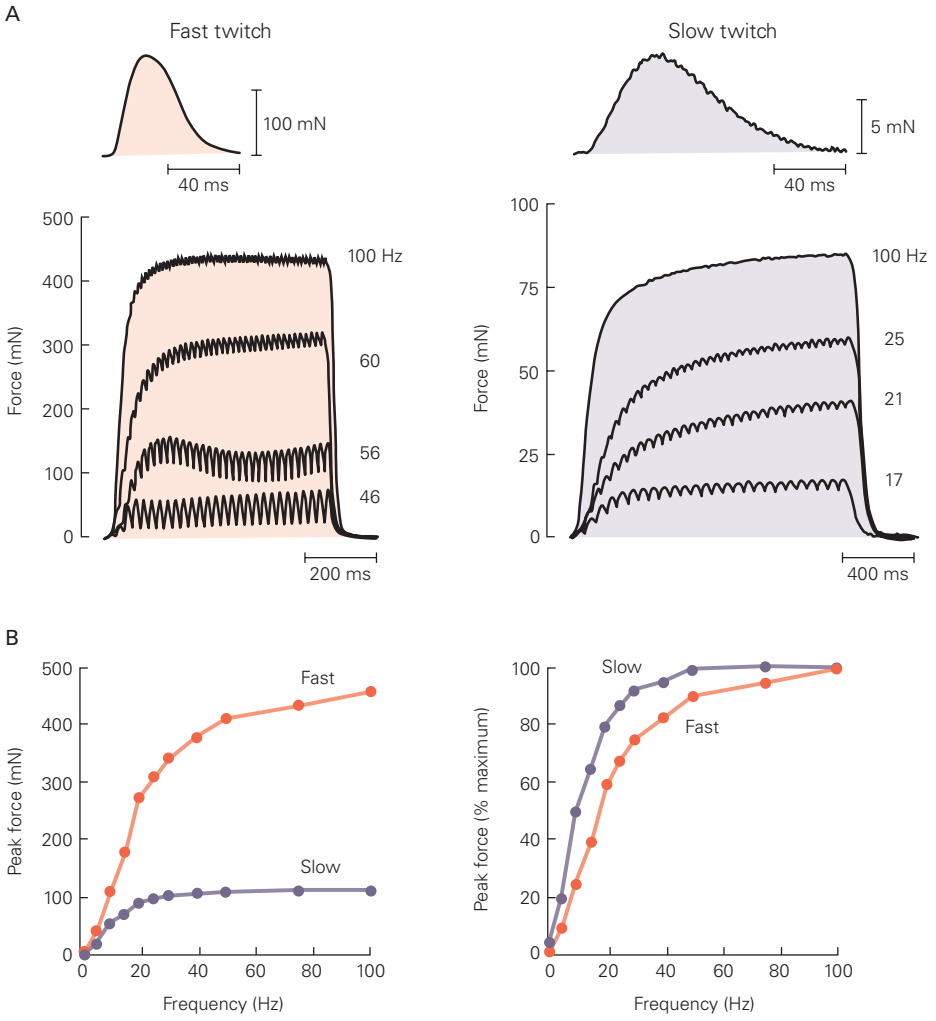


Figure 31–2 The force exerted by a motor unit varies with the rate at which its neuron generates action potentials.

A. Traces show the forces exerted by fast- and slow-contracting motor units in response to a single action potential (**top trace**) and a series of action potentials (set of **four traces below**). The time to the peak twitch force, or contraction time, is briefer in the faster unit. The rates of the action potentials used to evoke the tetanic contractions range from 17 to 100 Hz in the slow-contracting unit to 46 to 100 Hz in the fast-contracting unit. The peak tetanic force evoked by 100-Hz stimulation is greater for the fast-contracting unit. Note the different force scales for the two sets of traces. (Adapted, with permission, from Botterman, Iwamoto, and Gonyea 1986; adapted from Fuglevand, Macefield, and Bigland-Ritchie 1999; and Macefield, Fuglevand, and Bigland-Ritchie 1996.)

B. Relation between peak force and the rate of action potentials for fast- and slow-contracting motor units. The absolute force (*left plot*) is greater for the fast-contracting motor unit at all frequencies. At lower stimulus rates (*right plot*), the force evoked in the slow-contracting motor unit (longer contraction time) sums to a greater relative force (percent of peak force) than in the fast-contracting motor unit (shorter contraction time).

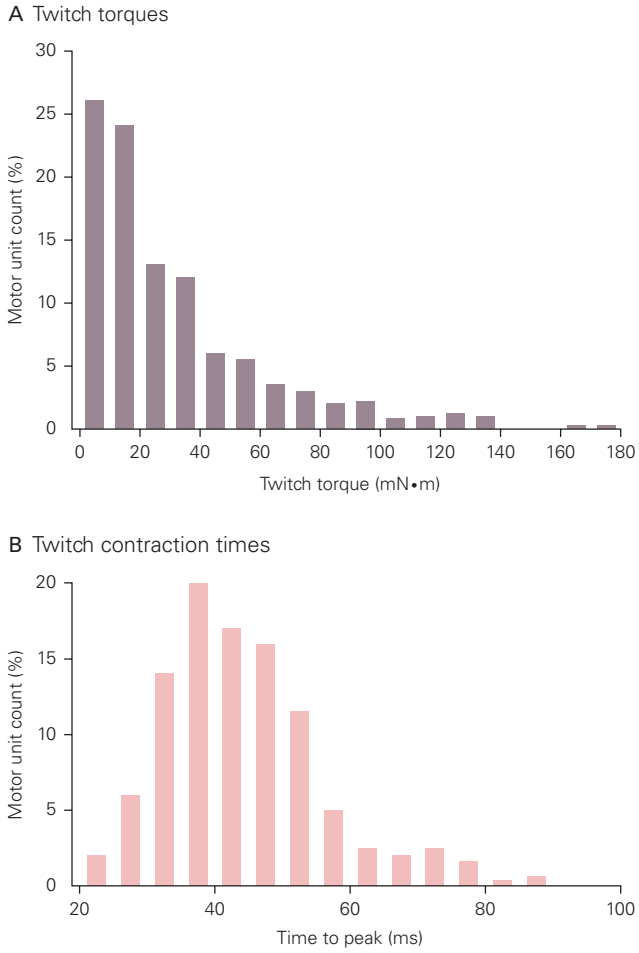


Figure 31–3 **Most human motor units produce low forces and have intermediate contraction times.** (Reproduced, with permission, from Van Cutsem et al. 1997. © Canadian Science Publishing.)

A. Distribution of twitch torques for 528 motor units in the tibialis anterior muscle obtained from 10 subjects.

B. Distribution of twitch contraction times for 528 motor units in the tibialis anterior muscle.

more fatigable. The order in which motor units are recruited during a voluntary contraction begins with the slow-contracting, low-force units and proceeds up to the fast-contracting, high-force units. As observed by Jacques Duchateau and colleagues, most motor units in humans produce low forces and have intermediate contraction times (Figure 31–3).

The range of contractile properties exhibited by motor units is partly attributable to differences in the structural specializations and metabolic properties of muscle fibers. One commonly used scheme to characterize muscle fibers is based on their reactivity to histochemical assays for the enzyme myosin adenosine triphosphatase (ATPase), which is used as an index of contractile speed. Histochemical stains for myosin ATPase can identify two types of muscle fibers: type I (low levels of myosin ATPase) and type II (high levels of myosin ATPase). Slow-contracting motor units contain type I muscle fibers, and fast-contracting units include type II fibers. The type II fibers can be further classified as being less fatigable (type IIa) or more fatigable (type IIb, IIx, or IId), due to the association between myosin ATPase content and the relative abundance of oxidative enzymes. Another commonly used scheme distinguishes muscle fibers on the basis of genetically defined isoforms of the myosin heavy chain (MHC). Muscle fibers in slow-contracting motor units express MHC-I, those in the less fatigable fast-contracting units express MHC-IIA, and those in the more fatigable fast-contracting units express MHC-IIX.

In actuality, the contractile properties of single muscle fibers are less distinct than the two classification schemes suggest (Figure 31–4). In addition to the variability in the contractile properties of each type of muscle fiber (MHC-I, -IIA, or -IIX), some muscle fibers co-express more than one MHC isoform. Such hybrid muscle fibers exhibit contractile properties that are intermediate between the muscle fibers that compose a

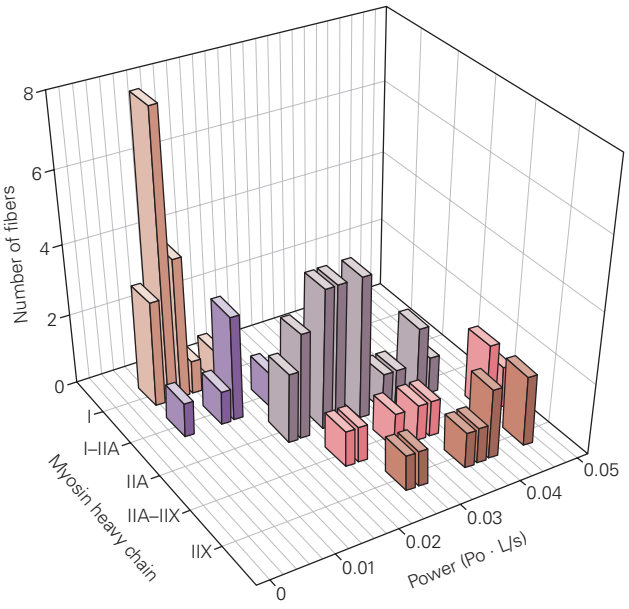


Figure 31–4 **The contractile properties of muscle fiber types are distributed continuously.** Peak power produced by segments of single muscle fibers from the vastus lateralis muscle with different types of myosin heavy chain (MHC) isoforms. Two types of hybrid fibers (I-IIA and IIA-IIX) contain isoforms of both types of MHCs. Power is calculated as the product of peak tetanic force (P_o) and maximal shortening velocity (segment length per second [L/s]). (Adapted, with permission, from Bottinelli et al. 1996. Copyright © 1996 The Physiological Society.)

single isoform. The relative proportion of hybrid fibers in a muscle increases with age. As with the distribution of contractile properties across motor units (Figure 31–3), the distribution across individual muscle fibers is also continuous, from slow to fast contracting and from least to most powerful (Figure 31–4).

Physical Activity Can Alter Motor Unit Properties

Alterations in habitual levels of physical activity can influence the three contractile properties of motor units (contraction speed, maximal force, and fatigability). A decrease in muscle activity, such as occurs with aging, bed rest, limb immobilization, or space flight, reduces the maximal capabilities of all three properties. The effects of increased physical activity vary with the intensity and duration of the activity. Brief sets of strong contractions performed a few times each week can increase motor unit force (strength training); brief sets of rapid contractions performed a few times each week can increase motor unit discharge rate (power training); and prolonged periods of weaker contractions can reduce motor unit fatigability (endurance training).

Changes in the contractile properties of motor units involve adaptations in the structural specializations and biochemical properties of muscle fibers. The improvement in contraction speed caused by power training, for example, is associated with an increase in the maximal shortening velocity of a muscle fiber caused by an increase in the quantity of myosin ATPase in the fiber. Similarly, the increase in maximal force is associated with the enlarged size and increased intrinsic force capacity of the muscle fibers produced by an increase in the number and density of the contractile proteins.

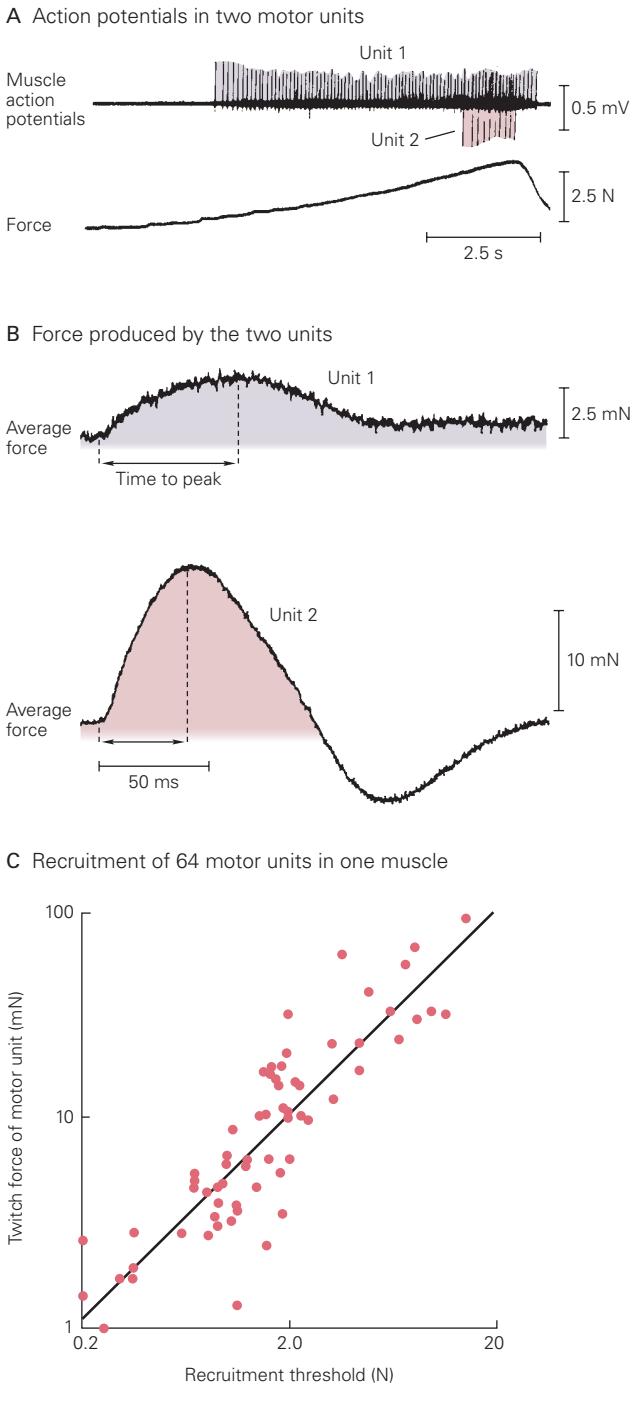
In contrast, decreases in the fatigability of a muscle fiber can be caused by many different adaptations, such as increases in capillary density, number of mitochondria, efficiency of the processes involved in activating the contractile proteins (excitation-contraction coupling), and oxidative capacity of the muscle fibers. Although the adaptive capabilities of muscle fibers decline with age, the muscles remain responsive to exercise even at 90 years of age.

Despite the efficacy of strength, power, and endurance training in altering the contractile properties of muscle fibers, these training regimens have little effect on the composition of a muscle's fibers. Although several weeks of exercise can change the relative proportion of type IIA and IIX fibers, it produces no change in the proportion of type I fibers. All fiber types adapt in response to exercise, although to varying extents depending on the type of exercise. For example, strength training of leg muscles for 2 to 3 months can increase the cross-sectional area of type I fibers by 0% to 20% and of type II fibers by 20% to 60%, increase the proportion of type IIa fibers by approximately 10%, and decrease the proportion of type IIx fibers by a similar amount. Furthermore, endurance training may increase the enzyme activities of oxidative metabolic pathways without noticeable changes in the proportions of type I and type II fibers, but the relative proportions of type IIa and IIx fibers do change as a function of the duration of each exercise session. Conversely, although several weeks of bed rest or limb immobilization do not change the proportions of fiber types in a muscle, they do decrease the size and intrinsic force capacity of muscle fibers. Adaptations in fiber type properties and proportions in turn alter the distribution of contractile properties in muscle fibers (Figure 31–4) and motor units (Figure 31–3).

Although physical activity has little influence on the proportion of type I fibers in a muscle, more substantial interventions can have an effect. Space flight, for example, exposes muscles to a sustained decrease in gravity that reduces the proportion of type I fibers in some leg muscles and decreases contractile properties. Similarly, surgically changing the nerve that innervates a muscle alters the pattern of activation and eventually causes the muscle to exhibit properties similar to those of the muscle that was originally innervated by the transplanted nerve. Connecting a nerve that originally innervated a rapidly contracting leg muscle to a slowly contracting leg muscle, for example, will cause the slower muscle to become more like a faster muscle. In contrast, a history of performing powerful contractions with leg muscles is associated with a modest reduction in the proportion of type I fibers, a marked increase in the proportion of type IIx fibers, and a huge increase in the power that can be produced by the type IIa and IIx fibers.

Muscle Force Is Controlled by the Recruitment and Discharge Rate of Motor Units

The force exerted by a muscle during a contraction depends on the number of motor units that are activated and the rate at which each of the active motor neurons discharges action potentials. Force is increased during a muscle contraction by the activation of additional motor units, which are recruited progressively from the weakest to the strongest (Figure 31–5). A motor unit's recruitment threshold is the force during the contraction at which the motor unit is activated. Muscle force decreases gradually by terminating the activity of motor units in the reverse order from strongest to weakest.



The order in which motor units are recruited is highly correlated with several indices of motor unit size, including the size of the motor neuron cell bodies, the diameter and conduction velocity of the axons, and the amount of force that the muscle fibers can exert. Because individual sources of synaptic input are broadly distributed across most neurons in a motor nucleus, the orderly recruitment of motor neurons is not accomplished by the sequential activation of different sets of synaptic inputs that target specific motor neurons. Rather, recruitment order is determined by intrinsic differences in the responsiveness of individual motor neurons to relatively uniform synaptic input.

One of these factors is the anatomical size of a neuron's soma and dendrites. Smaller neurons have a higher input resistance (R_{in}) to current and, due to Ohm's law ($\Delta V_m = I_{syn} \times R_{in}$), experience a greater change in membrane potential (ΔV_m) in response to a given synaptic current (I_{syn}). Consequently, increases in the net excitatory input to a motor nucleus cause the levels of depolarization to reach threshold in an ascending order of motor neuron size: Contraction force is increased by recruiting the smallest motor neuron first and the largest motor neuron last (Figure 31–6). This effect is known as the size principle of motor neuron recruitment, a concept enunciated by Elwood Henneman in 1957.

The size principle has two important consequences for the control of movement by the nervous system. First, the sequence of motor neuron recruitment is determined by the properties of the spinal neurons and not by supraspinal regions of the nervous system. This means that the brain cannot selectively activate specific motor units. Second, the axons arising from small motor neurons are thinner than those associated with

Figure 31–5 (Left) Motor units that exert low forces are recruited before those that exert greater forces. (Adapted, with permission, from Desmedt and Godaux 1977; Milner-Brown, Stein, and Yemm 1973. Copyright © 1973 The Physiological Society.)

A. Action potentials in two motor units were recorded concurrently with a single intramuscular electrode while the subject gradually increased muscle force. Motor unit 1 began discharging action potentials near the beginning of the voluntary contraction, and its discharge rate increased during the contraction. Motor unit 2 began discharging action potentials near the end of the contraction.

B. Average twitch forces for motor units 1 and 2 as extracted with an averaging procedure during the voluntary contraction.

C. The plot shows the net muscle forces at which 64 motor units in a hand muscle of one person were recruited (recruitment threshold) during a voluntary contraction relative to the twitch forces of the individual motor units.

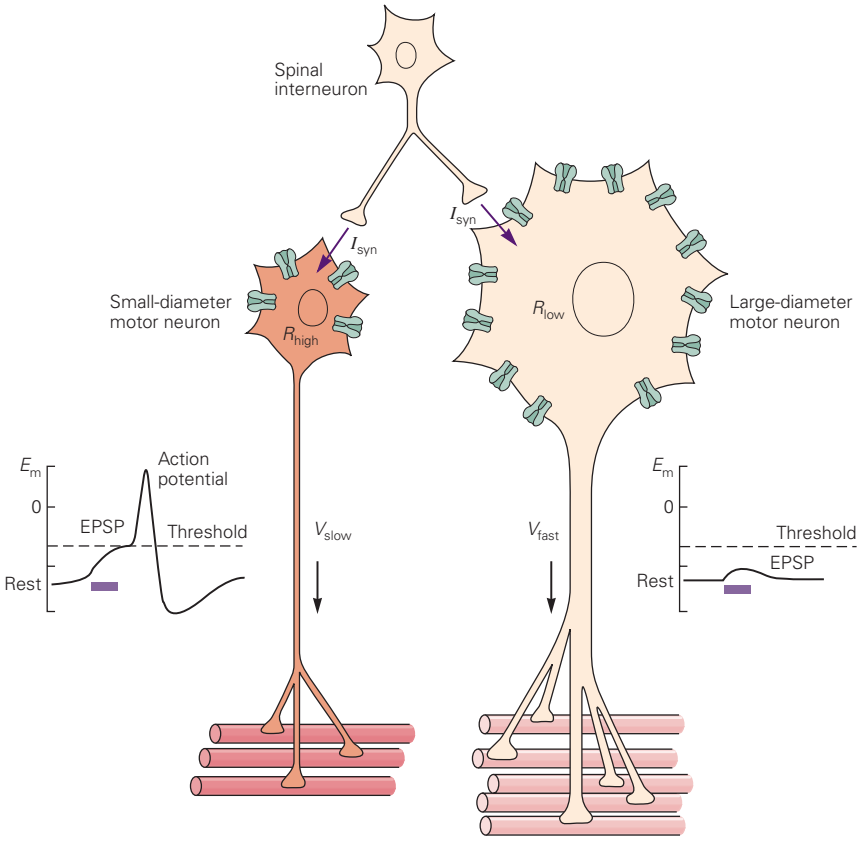


Figure 31–6 The size principle of motor neuron recruitment. Two motor neurons of different sizes have the same resting membrane potential (V_r) and receive the same excitatory synaptic current (I_{syn}) from a spinal interneuron. Because the small motor neuron has a smaller surface area, it has fewer parallel ion channels and therefore a higher input resistance (R_{high}). According to Ohm's law ($V = IR$), I_{syn} in the small neuron produces a large excitatory postsynaptic potential (**EPSP**) that reaches threshold, resulting in the discharge of an action potential. However, the axon of the small motor neuron has a small diameter and thus conducts the action potential at a relatively low velocity (V_{slow}) and to fewer muscle fibers. In contrast, the large motor neuron has a larger surface area, which results in a lower transmembrane resistance (R_{low}) and a smaller EPSP that does not reach threshold in response to I_{syn}; however, when synaptic input does reach threshold, the action potential is conducted relatively rapidly (V_{fast}) (Chapter 9).

large motor neurons and innervate fewer muscle fibers. Because the number of muscle fibers innervated by a motor neuron is a key determinant of motor unit force, motor units are activated in order of increasing strength, so the earliest recruited motor units are the weakest ones.

As suggested by Edgar Adrian in the 1920s, the muscle force at which the last motor unit in a motor nucleus is recruited varies between muscles. In some hand muscles, all the motor units have been recruited when the force reaches approximately 60% of maximum during a slow muscle contraction. In the biceps brachii, deltoid, and tibialis anterior muscles, recruitment continues up to approximately 85% of the maximal force. Beyond the upper limit of motor unit recruitment, changes in muscle force depend solely on variations in the rate at which motor neurons generate action potentials. Over most of the operating range of a muscle, the force it exerts depends on concurrent changes in discharge rate and the number of active motor units (Figure 31–7). Except at low forces, however, variation in

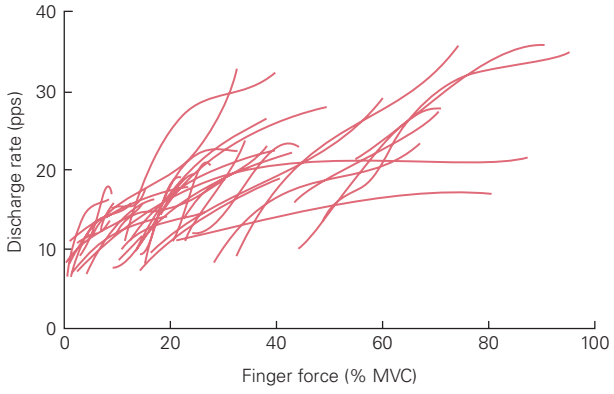


Figure 31–7 Muscle force can be adjusted by varying the number of active motor units and their discharge rate. Each line shows the discharge rate (pulses per second [**pps**]) for a single motor unit in a hand muscle over a range of finger forces (maximal voluntary contraction [**MVC**]). The finger force was produced by the action of a single hand muscle. The leftmost point of each line indicates the threshold force at which the motor unit is recruited, whereas the rightmost point corresponds to the peak force at which the motor unit could be identified. The range of discharge rates was often less for motor units with lower recruitment thresholds. Increases in finger force were produced by concurrent increases in discharge rate and the number of activated motor units. (Adapted, with permission, from Moritz et al. 2005.)

discharge rate has a greater influence on muscle force than does changes in the number of active motor units.

The order in which motor units are recruited does not change with contraction speed. Due to the time involved in excitation-contraction coupling, faster contractions require the action potential for each motor unit to be generated earlier than during a slow contraction. As a result of this adjustment, the upper limit of motor unit recruitment during the fastest muscle contractions is approximately 40% of maximum. Consequently, it is possible to manipulate the rate at which motor units are recruited by varying contraction speed.

The Input–Output Properties of Motor Neurons Are Modified by Input From the Brain Stem

The discharge rate of motor neurons depends on the magnitude of the depolarization generated by excitatory inputs and the intrinsic membrane properties of the motor neurons in the spinal cord. These properties can be profoundly modified by input from monoaminergic neurons in the brain stem (Chapter 40). In the absence of this input, the dendrites of motor neurons passively transmit synaptic current to the cell body, resulting in a modest depolarization that immediately ceases when the input stops. Under these conditions, the relation between input current and discharge rate is linear over a wide range.

The input–output relation becomes nonlinear, however, when the monoamines serotonin and norepinephrine induce a huge increase in conductance by activating L-type Ca^{2+} channels that are located on the dendrites of the motor neurons. The resulting inward Ca^{2+} currents can enhance synaptic currents by three- to five-fold (Figure 31–8). In an active motor neuron, this augmented current can sustain an elevated discharge rate after a brief depolarizing input has ended, a behavior known as *self-sustained firing*. A subsequent brief inhibitory input, such as from a spinal reflex pathway, can terminate such self-sustained firing.

Because the properties of motor neurons are strongly influenced by monoamines, the excitability of the pool of motor neurons innervating a single muscle is partly under control of the brain stem. In the awake state, moderate levels of monoaminergic input to the motor neurons of slowly contracting motor units promote self-sustained firing. This is probably the source of the sustained force exerted by slower motor units to maintain posture (Chapter 36). Conversely, the withdrawal of monoaminergic drive during sleep decreases excitability and helps ensure a relaxed motor state. Thus, monoaminergic input from the brain stem can adjust the gain of the motor unit pool to meet the demands of different tasks. This flexibility does not compromise the size principle of orderly recruitment because the threshold for activation of the persistent inward currents is lowest in the motor neurons of slower contracting motor units, which are the first recruited even in the absence of monoamines.

Muscle Force Depends on the Structure of Muscle

Muscle force depends not only on the amount of motor neuron activity but also on the arrangement of the fibers in the muscle. Because movement involves the controlled variation of muscle force, the nervous system must take into account the structure of muscle to achieve specific movements.

The Sarcomere Is the Basic Organizational Unit of Contractile Proteins

Individual muscles contain thousands of fibers that vary from 1 to 50 mm in length and from 10 to 60 μm in diameter. The variation in fiber dimensions reflects differences in the quantity of contractile protein. Despite this quantitative variation, the organization of contractile proteins is similar in all muscle fibers. The proteins are arranged in repeating sets of thick and thin filaments, each set known as a *sarcomere* (Figure 31–9). The in vivo length of a sarcomere, which is bounded by Z disks, ranges from 1.5 to 3.5 μm within and across muscles. Sarcomeres are arranged in series to form a *myofibril,* and the myofibrils are aligned in parallel to form a muscle fiber (myocyte).

The force that each sarcomere can generate arises from the interaction of the contractile thick and thin filaments. The thick filament consists of several hundred myosin molecules arranged in a structured sequence. Each myosin molecule comprises paired coiled-coil domains that terminate in a pair of globular heads. The myosin molecules in the two halves of a thick filament point in opposite directions and are progressively displaced so that the heads, which extend away from the filament, protrude around the thick filament (Figure 31–9C). The thick filament is anchored in the middle of the sarcomere by the protein titin, which connects each end of the thick filament with neighboring strands of actin in the thin filament and with the Z-disc. To maximize the interaction between the globular heads of myosin and the thin filaments, six thin filaments surround each thick filament.

The primary components of the thin filament are two helical strands of fibrous F-actin, each of which

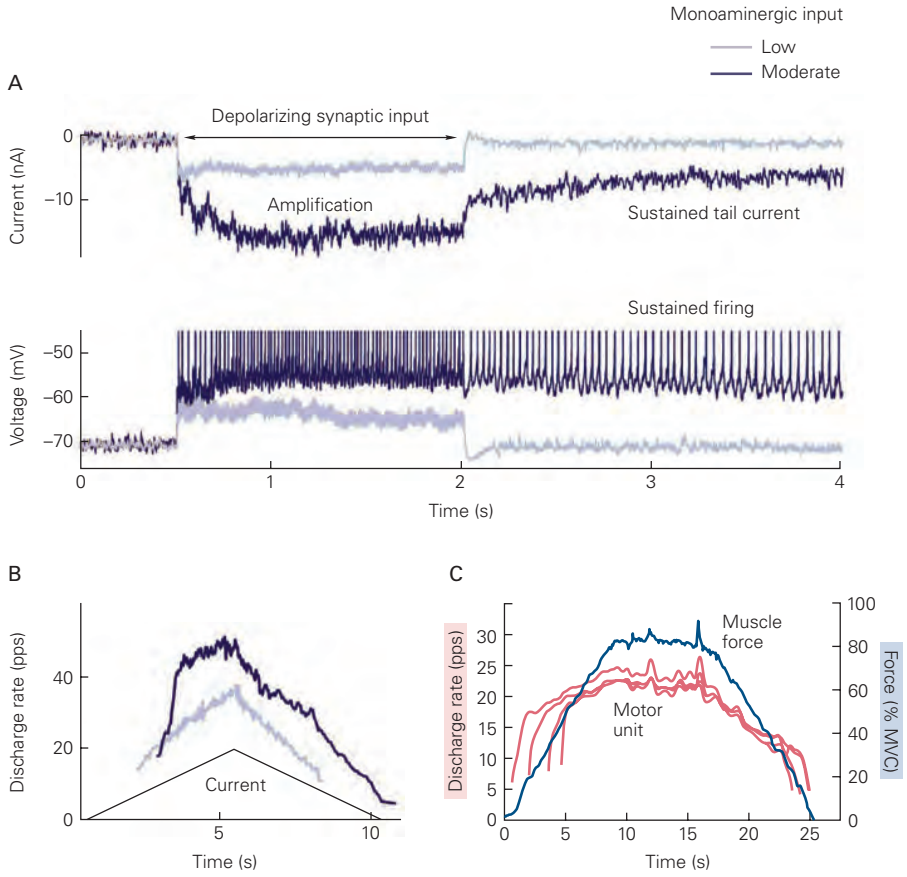


Figure 31–8 Monoaminergic input enhances the excitability of motor neurons. (Part A, adapted, with permission, of Heckman et al. 2009. Copyright © 2009 International Federation of Clinical Neurophysiology; Part B, data from CJ Heckman; Part C, adapted, with permission, from Erim et al. 1996. Copyright © 1996 John Wiley & Sons, Inc.)

A. Membrane currents and potentials in spinal motor neurons of adult cats that were either deeply anesthetized (low monoaminergic drive) or decerebrate (moderate monoaminergic drive). When monoaminergic input is absent or low, a brief excitatory input produces an equally brief synaptic current during voltage clamp (**upper record**). This current is not sufficient to bring the membrane potential of the neuron to threshold for generating action potentials in the unclamped condition (**lower record**). The same brief excitatory input during moderate levels of monoaminergic input activates a persistent inward current in the dendrites, which amplifies the excitatory synaptic current and decays slowly following cessation of synaptic input (**upper record**). This persistent inward current causes a high discharge rate during the input and sustains a lesser discharge rate after the input ceases (**lower record**). A brief inhibitory input will return the neuron to its resting state.

B. High levels of monoaminergic input to a motor neuron give rise to a persistent inward current in response to injected current, resulting in a much greater discharge rate for a given amount of current.

C. The **blue** trace represents the force exerted by the dorsiflexor muscle during a contraction that gradually increased to 80% of maximal voluntary isometric contraction (**MVC**) force in a human subject. Each of the four **pink** traces indicates the change in the rate at which a single motor unit discharged action potentials during the contraction. The leftmost point (start) of each of these four traces shows the time when the motor unit was recruited, and the rightmost point (end) denotes the time at which the motor neuron stopped discharging action potentials. The rapid increase in discharge rate during the increase in muscle force is similar to the change in rate observed in the presence of moderate levels of monoaminergic input (see part B).

contains approximately 200 actin monomers. Superimposed on F-actin are tropomyosin and troponin, proteins that control the interaction between actin and myosin. Tropomyosin consists of two coiled strands that lie in the groove of the F-actin helix; troponin is a small molecular complex that is attached to tropomyosin at regular intervals (Figure 31–9C).

The thin filaments are anchored to the Z disk at each end of the sarcomere, whereas the thick filaments occupy the middle of the sarcomere (Figure 31–9B). This organization accounts for the alternating light and dark bands of striated muscle. The light band contains only thin filaments, whereas the dark band contains both thick and thin filaments. When a muscle is

activated, the width of the light band decreases but the width of the dark band does not change, suggesting that the thick and thin filaments slide relative to one another during a contraction. This led to the *sliding filament hypothesis* of muscle contraction proposed by A. F. Huxley and H. E. Huxley in the 1950s.

The sliding of the thick and thin filaments is triggered by the release of Ca^{2+} from within the sarcoplasm of a muscle fiber in response to an action potential that travels along the fiber's membrane, the sarcolemma. Varying the amount of Ca^{2+} in the sarcoplasm controls the interaction between the thick and thin filaments. The Ca^{2+} concentration in the sarcoplasm is kept low under resting conditions by active pumping of Ca^{2+} into the sarcoplasmic reticulum, a network of longitudinal tubules and chambers of smooth endoplasmic reticulum. Calcium is stored in the terminal cisternae, which are located next to intracellular extensions of the sarcolemma known as transverse tubules (T-tubules). The transverse tubules, terminal cisternae, and sarcoplasmic reticulum constitute an activation system that transforms an action potential into the sliding of the thick and thin filaments (Figure 31–9A).

As an action potential propagates along the sarcolemma, it invades the transverse tubules and causes the rapid release of Ca^{2+} from the terminal cisternae into the sarcoplasm. Once in the sarcoplasm, Ca^{2+} diffuses among the filaments and binds reversibly to troponin, which results in the displacement of the troponin–tropomyosin complex and enables the sliding of the thick and thin filaments. Because a single action potential does not release enough Ca^{2+} to bind all available troponin sites in skeletal muscle, the strength of a contraction increases with the action potential rate.

The sliding of the filaments depends on mechanical work performed by the globular heads of myosin, work that uses chemical energy contained in adenosine triphosphate (ATP). The actions of the myosin heads are regulated by the *cross-bridge cycle*, a sequence of detachment, activation, and attachment (Figure 31–10). In each cycle, a globular head undergoes a displacement of 5 to 10 nm. Contractile activity continues as long as Ca^{2+} and ATP are present in the cytoplasm in sufficient amounts.

Once the contractile proteins have been activated by the release of Ca^{2+}, sarcomere length may increase, remain the same, or decrease depending on the magnitude of the load against which the muscle is acting. The force generated by an activated sarcomere when its length does not change or decreases can be explained by the cross-bridge cycle involving the thick and thin filaments. When the length of the activated sarcomere increases, however, the force developed by the extension of titin adds significantly to the sarcomere force. The force produced by titin during the stretch of an activated sarcomere is augmented by its ability to increase stiffness, which is accomplished when titin binds Ca^{2+} and then attaches at specific locations on actin to reduce the length that it can be stretched. The force produced by activated sarcomeres therefore depends on the interactions of three filaments (actin, myosin, and titin).

Noncontractile Elements Provide Essential Structural Support

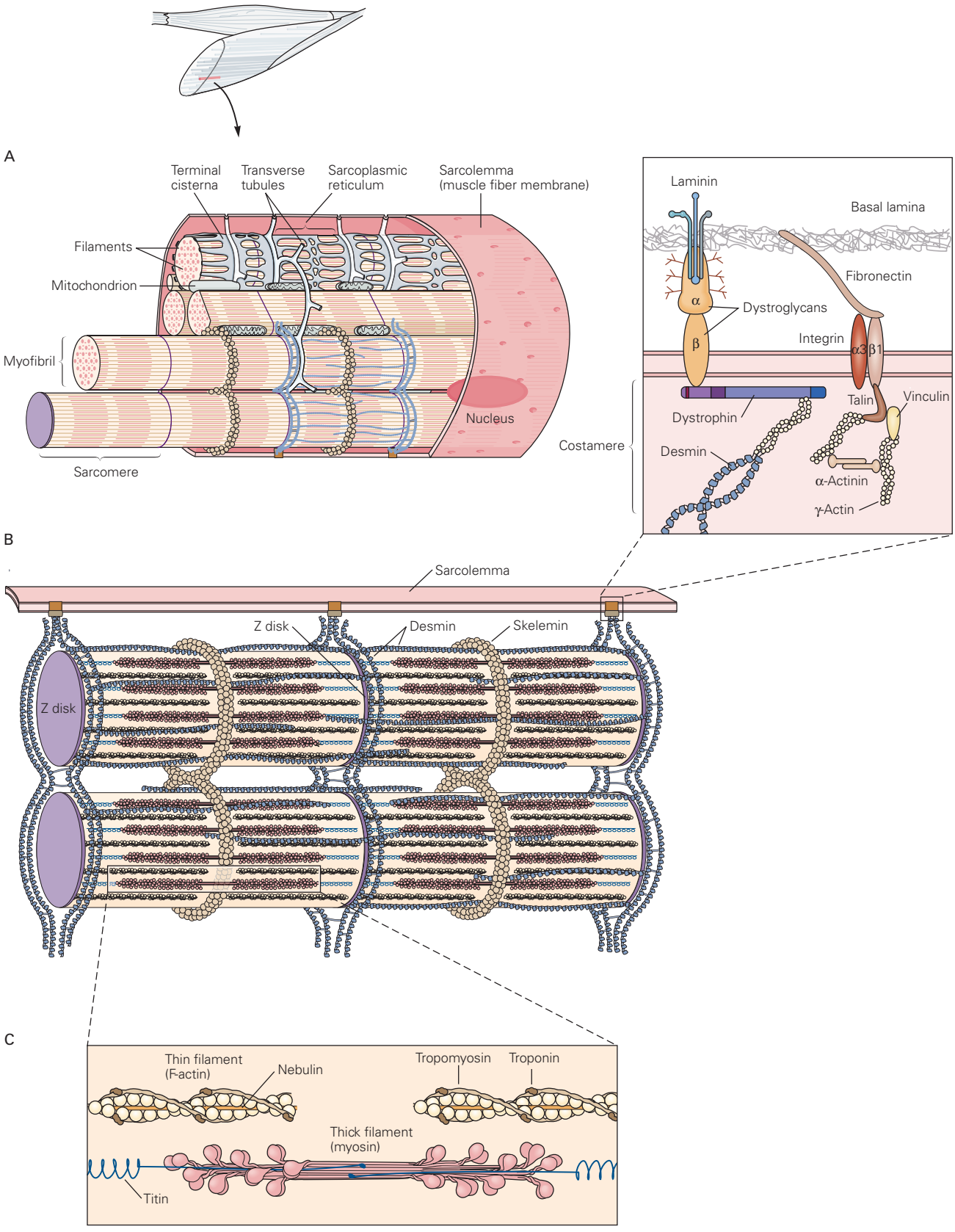
Structural elements of the muscle fiber maintain the alignment of the contractile proteins within the fiber and facilitate the transmission of force from the sarcomeres to the skeleton. A network of proteins (nebulin, titin) maintains the orientation of the thick and thin filaments within the sarcomere, whereas other proteins (desmin, skelemins) constrain the lateral alignment of the myofibrils (Figure 31–9B). These proteins contribute to the elasticity of muscle and maintain the appropriate alignment of cellular structures when the muscle acts against an external load.

Although some of the force generated by the cross bridges is transmitted along the sarcomeres in series, most of it travels laterally from the thin filaments to an extracellular matrix that surrounds each muscle fiber, through a group of transmembrane and membrane-associated proteins called a *costamere* (see inset for Figure 31–9B). The lateral transmission of force follows two pathways through the costamere, one through a dystrophin–glycoprotein complex and the other through vinculin and members of the integrin family. Mutations of genes that encode components of the dystrophin–glycoprotein complex cause muscular dystrophies in humans, which are associated with substantial decreases in muscle force.

Contractile Force Depends on Muscle Fiber Activation, Length, and Velocity

The force that a muscle fiber can exert depends on the number of cross bridges formed and the force produced by each cross bridge. These two factors are influenced by the Ca^{2+} concentration in the sarcoplasm, the amount of overlap between the thick and thin filaments, and the velocity with which the thick and thin filaments slide past one another.

The influx of Ca^{2+} that activates formation of the cross bridges is transitory because continuous pump activity quickly returns Ca^{2+} to the sarcoplasmic reticulum. The release and reuptake of Ca^{2+} in response to a single action potential occurs so quickly that only some


A
Terminal cisterna
Transverse tubules
Sarcoplasmic reticulum
Sarcolemma (muscle fiber membrane)
Filaments
Mitochondrion
Myofibril
Nucleus
Sarcomere
Costamere
Laminin
Basal lamina
Fibronectin
α
Dystroglycans
β
Integrin
α3β1
Talin
Vinculin
Dystrophin
Desmin
α-Actinin
γ-Actin
B
Sarcolemma
Z disk
Desmin
Skelemin
Z disk
C
Thin filament (F-actin)
Nebulin
Tropomyosin
Troponin
Thick filament (myosin)
Titin

of the potential cross bridges are formed. This explains why the peak force of a twitch is less than the maximal force of the muscle fiber (see Figure 31–2A). Maximal force can be achieved only with a series of action potentials that sustains the Ca^{2+} concentration in the sarcoplasm, thus maximizing cross bridge formation.

Although Ca^{2+} activates formation of the cross bridges, cross bridges can be formed only when the thick and thin filaments overlap. This overlap varies as the filaments slide relative to one another (Figure 31–11A). The amount of overlap between actin and myosin is optimal at an intermediate sarcomere length (L_o), and the relative force is maximal. Increases in sarcomere length reduce the overlap between actin and myosin and the force that can be developed. Decreases in sarcomere length cause the thin filaments to overlap, reducing the number of binding sites available to the myosin heads. Although many muscles operate over a narrow range of sarcomere lengths (approximately 94 ± 13% L_o, mean ± standard deviation), among muscles, there is considerable diversity in sarcomere lengths during movement.

Because structures that connect the contractile proteins to the skeleton also influence the force that a muscle can exert, muscle force increases with length over its operating range. This property enables muscle to function like a spring and to resist changes in length. Muscle stiffness, which corresponds to the slope of the relation between muscle force and muscle length (N/m), depends on the structure of the muscle. A stiffer muscle, similar to a stronger spring, is more resistant to changes in length.

Once activated, cross bridges perform work and cause the thick and thin filaments to slide relative to one another. Due to the elasticity of intracellular cytoskeletal proteins and the extracellular matrix, sarcomeres will shorten when the cross bridges are activated and the length of the muscle fiber is held fixed (*isometric contraction*). When the length of the muscle fiber is not kept constant, the direction and rate of change in sarcomere length depend on the amount of muscle fiber force relative to the magnitude of the load against which the fiber acts. Sarcomere length decreases when the muscle fiber force exceeds the load (*shortening contraction*) but increases when the force is less than the load (*lengthening contraction*). The maximal force that a muscle fiber can exert decreases as shortening velocity increases but increases as lengthening velocity increases (Figure 31–11B).

The maximal rate at which a muscle fiber can shorten is limited by the peak rate at which cross bridges can form. The variation in fiber force as contraction velocity changes is largely caused by differences in the average force exerted by each cross bridge. For example, the decrease in force during a shortening contraction is attributable to a reduction in cross-bridge displacement during each power stroke and the failure of some myosin heads to find attachment sites. Conversely, the increase in force during a lengthening contraction reflects the stretching of incompletely activated sarcomeres, the more rapid reattachment of cross bridges after they have been pulled apart, and the attachment of Ca^{2+} to titin.

The rate of cross-bridge cycling depends not only on contraction velocity but also on the preceding activity of the muscle. For example, the rate of cross-bridge cycling increases after a brief isometric contraction. When a muscle is stretched while in this state, such as would occur during a postural disturbance, muscle stiffness is enhanced, and the muscle is more effective at resisting the change in length. This property is known as *short-range stiffness*. Conversely, the cross-bridge cycling rate decreases after shortening contractions, and the muscle does not exhibit short-range stiffness.

Figure 31–9 (Opposite) **The sarcomere is the basic functional unit of muscle.** (Adapted from Bloom and Fawcett 1975.)

A. This section of a muscle fiber shows its anatomical organization. Several myofibrils lie side by side in a fiber, and each myofibril is made up of sarcomeres arranged end to end and separated by Z disks (see part **B**). The myofibrils are surrounded by an activation system (the transverse tubules, terminal cisternae, and sarcoplasmic reticulum) that initiates muscle contraction.

B. Sarcomeres are connected to one another and to the muscle fiber membrane by the cytoskeletal lattice. The cytoskeleton influences the length of the contractile elements, the thick and thin filaments (see part **C**). It maintains the alignment of these filaments within a sarcomere, connects adjacent myofibrils, and transmits force to the extracellular matrix of connective tissue through costameres. One consequence of this organization is that the force generated by the contractile elements in a sarcomere can be transmitted along and across sarcomeres (through desmin and skelemin), within and between sarcomeres (through nebulin and titin), and to the sarcolemma through the costameres. The Z disk is a focal point for many of these connections.

C. The thick and thin filaments comprise different contractile proteins. The thin filament includes polymerized actin along with the regulatory proteins tropomyosin and troponin. The thick filament is an array of myosin molecules; each molecule includes a stem that terminates in a pair of globular heads. The protein titin maintains the position of each thick filament in the middle of the sarcomere.

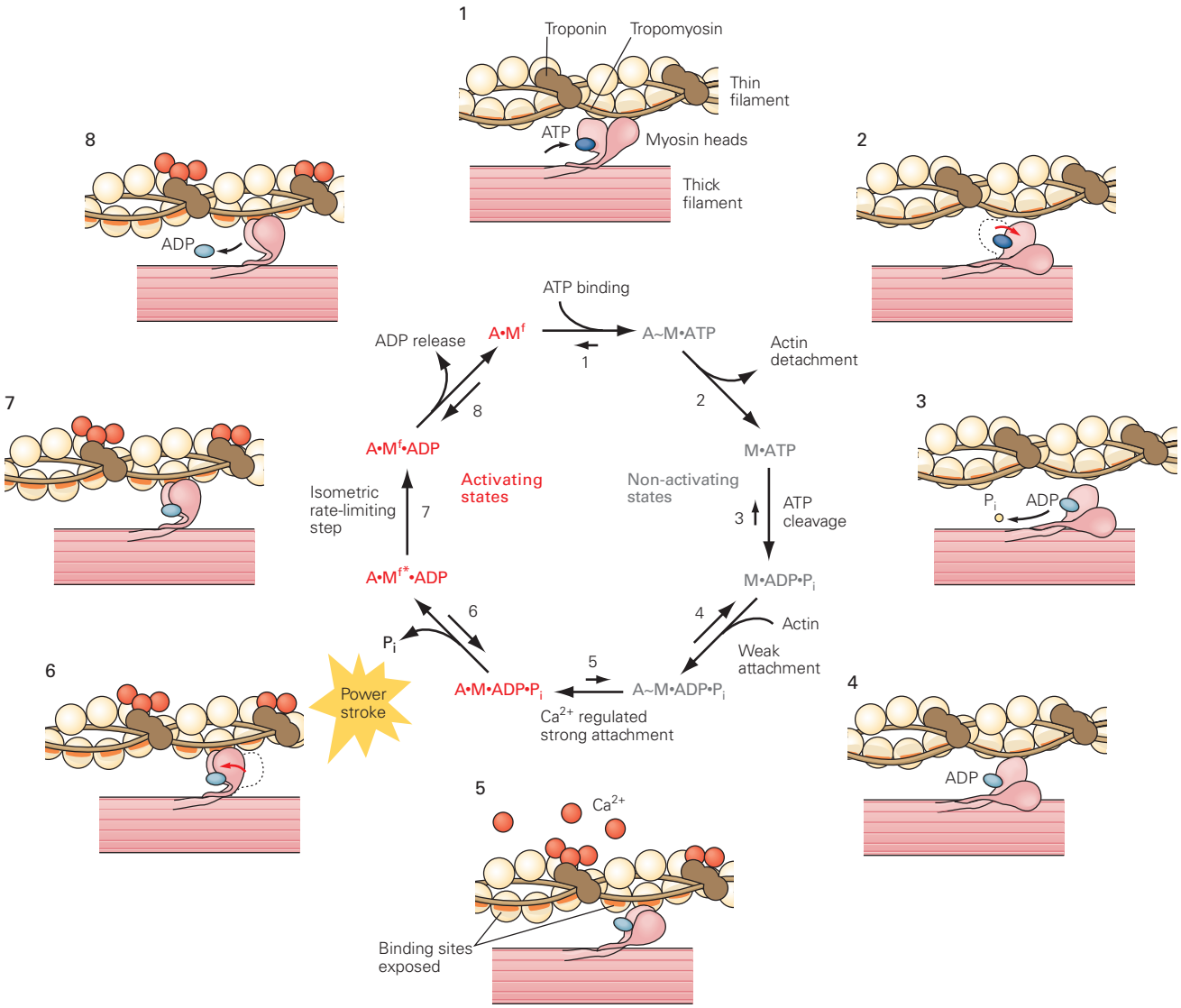


Figure 31–10 The cross-bridge cycle. Several nonactivating states are followed by several activating states triggered by Ca^{2+}. The cycle begins at the top (**step 1**) with the binding of adenosine triphosphate (**ATP**) to the myosin head. The myosin head detaches from actin (**step 2**), ATP is cleaved to phosphate (P_i) and adenosine diphosphate (**ADP**) (**step 3**), and the myosin becomes weakly bound to actin (**step 4**). The binding of Ca^{2+} to troponin causes tropomyosin to slide over actin and enables the two myosin heads to close (**step 5**). This results in the release of P_i and the extension of the myosin neck, the power stroke of the cross-bridge cycle (**step 6**). Each cross-bridge exerts a force of approximately 2 pN during a structural change (**step 7**) and the release of ADP (**step 8**). (•, strong binding; ~, weak binding; M^f, cross-bridge force of myosin; and M^f*, force-bearing state of myosin.) (Adapted, with permission, from Gordon, Regnier, and Homsher 2001.)

Muscle Torque Depends on Musculoskeletal Geometry

The anatomy of a muscle has a pronounced effect on its force capacity, range of motion, and shortening velocity. The anatomical features that influence function include the arrangement of the sarcomeres in each muscle fiber, the organization of the muscle fibers within the muscle, and the location of the muscle's attachments on the skeleton. These features vary widely among muscles.

At the level of a single muscle fiber, the number of sarcomeres in series and in parallel can vary. The number of sarcomeres in series determines the length

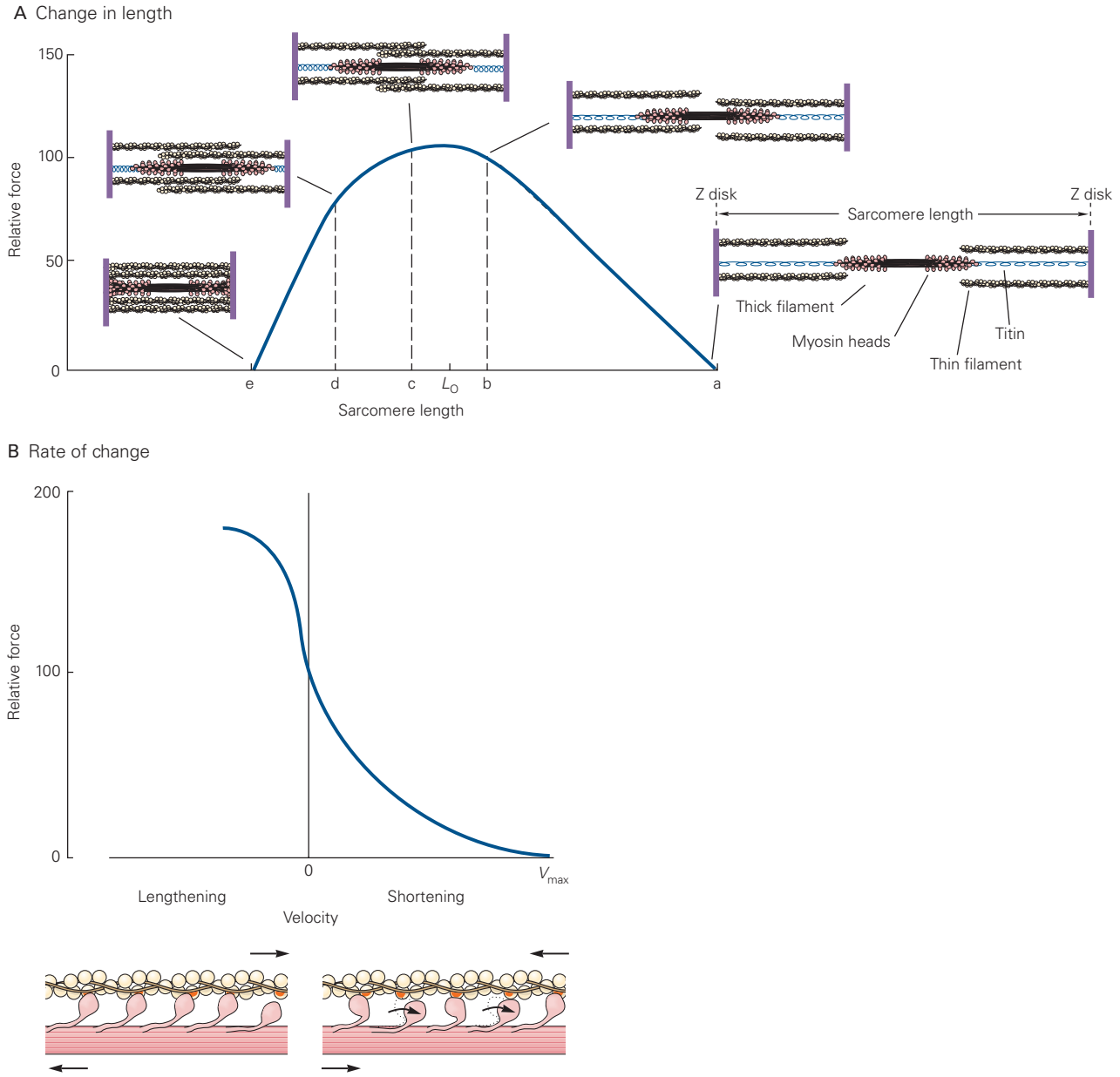


Figure 31–11 Contractile force varies with the change in sarcomere length and velocity.

A. At an intermediate sarcomere length, L_o, the amount of overlap between actin and myosin is optimal and the relative force is maximal. When the sarcomere is stretched beyond the length at which the thick and thin filaments overlap (length **a**), cross bridges cannot form and no force is exerted. As sarcomere length decreases and the overlap of the thick and thin filaments increases (between lengths **a** and **b**), the force increases because the number of cross bridges increases. With further reductions in length (between lengths **c** and **e**), the extreme overlap of the thin filaments with each other occludes potential attachment sites and the force decreases.

B. Contractile force varies with the rate of change in sarcomere length. Relative to the force that a sarcomere can exert during an isometric contraction (zero velocity), the peak force declines as the rate of shortening increases. Muscle force reaches a minimum at the maximal shortening velocity (V_{max}). In contrast, when the sarcomere is lengthened while being activated, the peak force increases to values greater than those during an isometric contraction. Shortening causes the myosin heads to spend more time near the end of their power stroke, where they produce less contractile force, and more time detaching, recocking, and reattaching, during which they produce no force. When the muscle is actively lengthened, the myosin heads spend more time stretched beyond their angle of attachment and little time unattached because they do not need to be recocked after being pulled away from the actin in this manner. Titin also contributes significantly to sarcomere force during lengthening contractions.

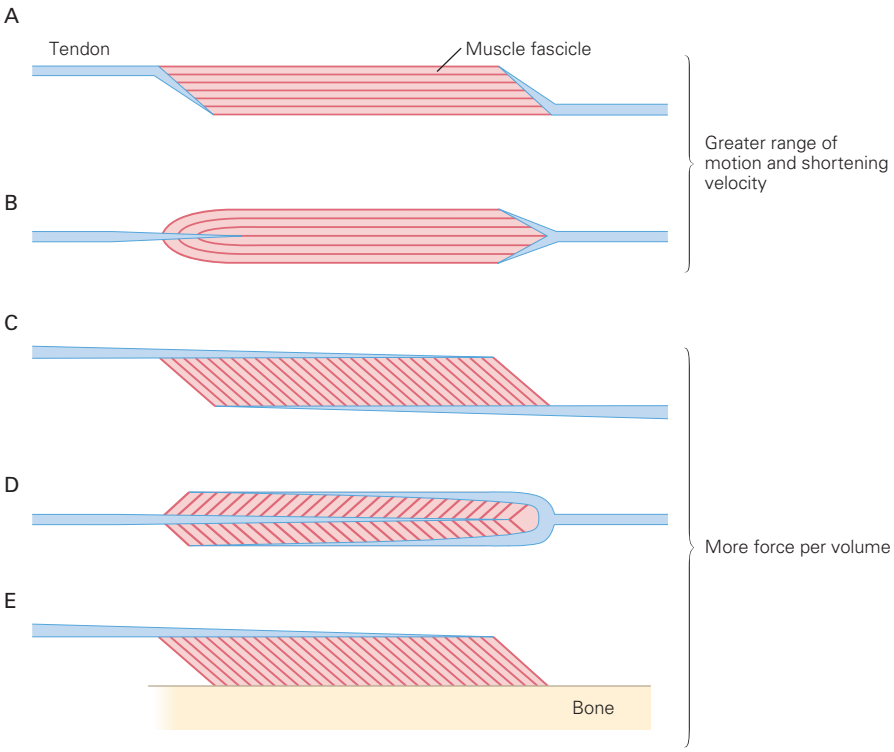


Figure 31–12 Five common arrangements of tendon and muscle. The fundamental distinction between these arrangements is whether or not the muscle fascicles are aligned with the line of pull of the muscles. The fascicles in muscles A and B are parallel to the line of pull (longitudinal axis of the muscle), whereas the fascicles in muscles C, D, and E are rotated away from the line of pull. The magnitude of this rotation is expressed as the pennation angle. (Reproduced, with permission, from Alexander and Ker 1990.)

of the myofibril and thus the length of the muscle fiber. Because one sarcomere can shorten by a certain length with a given maximal velocity, both the range of motion and the maximal shortening velocity of a muscle fiber are proportional to the number of sarcomeres in series. The force that a myofibril can exert is equal to the average sarcomere force and is not influenced by the number of sarcomeres in series. Rather, the force capacity of a fiber depends on the number of sarcomeres in parallel and hence on the diameter or cross-sectional area of the fiber. At the level of the muscle, the functional attributes of the fibers are modified by the orientation of the fascicles (bundles of muscle fibers) to the line of pull of the muscle and the length of the fiber relative to the muscle length. In most muscles, the fascicles are not parallel to the line of pull but fan out in feather-like (pennate) arrangements (Figure 31–12).

The relative orientation, or pennation angle of the fascicles, ranges from close to 0° (biceps brachii, sartorius) to approximately 30° (soleus). Because more fibers can fit into a given volume as the pennation angle increases, muscles with large pennation angles typically have more fascicles in parallel and hence large cross-sectional areas when measured perpendicular to the long axis of individual muscle fibers. Given the linear relation between cross-sectional area (quantity of contractile proteins in parallel) and maximal force (~22.5 N • cm^{-2}), these muscles are capable of a greater maximal force. However, the fibers in pennate muscles are generally short and have a lesser maximal shortening velocity than those in nonpennate muscles.

The functional consequences of this anatomical arrangement can be seen by comparing the contractile properties of two muscles with different numbers of fibers and fiber lengths. If the two muscles have identical fiber lengths but one has twice as many fibers, the range of motion of the two muscles will be similar because it is a function of fiber length, but the maximal force capacity will vary in proportion to the number of muscle fibers. If the two muscles have identical numbers of fibers but the fibers in one muscle are twice as long, the muscle with the longer fibers will have a greater range of motion and a greater maximal shortening velocity, even though the two muscles have a similar force capacity. Because of this effect, the muscle with longer fibers is able to exert more force and produce more power (the product of force and velocity) at a given absolute shortening velocity (Figure 31–13).

Muscle fiber lengths and cross-sectional areas vary substantially throughout the human body, which suggests that the contractile properties of individual muscles also differ markedly (Table 31–2). In the leg,

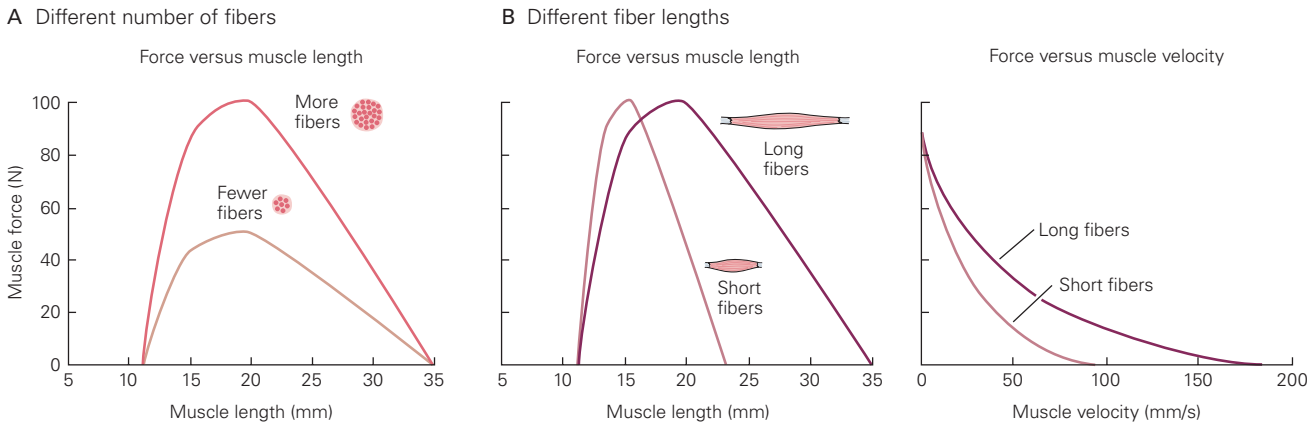


Figure 31–13 Muscle dimensions influence the peak force and maximal shortening velocity. (Reproduced, with permission, from Lieber and Fridén 2000. Copyright © 2000 John Wiley & Sons, Inc.)

A. Muscle force at various muscle lengths for two muscles with similar fiber lengths but different numbers of muscle fibers (different cross-sectional area). The muscle with twice as many fibers exerts greater force.

B. Muscle force at various muscle lengths for two muscles with the same cross-sectional area but different fiber lengths. The muscle with longer fibers (approximately twice as long as those of the other muscle) has an increased range of motion (left plot). It also has a greater maximal shortening velocity and exerts greater force at a given absolute velocity (right plot).

Table 31–2 Average Architectural Properties for Some Human Skeletal Leg Muscles

Muscle	Mass (g)	Muscle length (cm)	Fiber length (cm)	Pennation angle (°)	Cross-sectional area (cm^2)
Thigh					
Sartorius	78	45	40	1	2
Rectus femoris	111	36	8	14	14
Vastus lateralis	376	27	10	18	35
Vastus intermedius	172	41	10	5	17
Vastus medialis	239	44	10	30	21
Gracilis	53	29	23	8	2
Adductor longus	75	22	11	7	7
Adductor brevis	55	15	10	6	5
Adductor magnus	325	38	14	16	21
Biceps femoris (long)	113	35	10	12	11
Biceps femoris (short)	60	22	11	12	5
Semitendinosus	100	30	19	13	5
Semimembranosus	134	29	7	15	18
Lower leg					
Tibialis anterior	80	26	7	10	11
Extensor hallucis longus	21	24	7	9	3
Extensor digitorum longus	41	29	7	11	6
Peroneus longus	58	27	5	14	10
Peroneus brevis	24	24	5	11	5
Gastrocnemius (medial)	113	27	5	10	21
Gastrocnemius (lateral)	62	22	6	12	10
Soleus	276	41	4	28	52
Flexor hallucis longus	39	27	5	17	7
Flexor digitorum longus	20	27	4	14	4
Tibialis posterior	58	31	4	14	14

Source: Adapted, with permission, from Ward et al. 2009.

for example, pennation angle ranges from 1° (sartorius) to 30° (vastus medialis), fiber length ranges from 4 mm (soleus) to 40 mm (sartorius), and cross-sectional area ranges from 2 cm^2 (sartorius) to 52 cm^2 (soleus). In addition, the fact that muscle fiber length is usually less than muscle length indicates that muscle fibers are connected serially within a muscle. Functionally coupled muscles tend to have complementary combinations of these properties. For example, the three vasti muscles have similar muscle fiber lengths (10 cm), but they differ in pennation angle (intermedius is the smallest) and cross-sectional area (lateralis is the largest). A similar relation exists for soleus and the two heads (medial and lateral) of gastrocnemius.

Movement involves the muscle-controlled rotation of adjacent body segments, which means that the capacity of a muscle to contribute to a movement also depends on its location relative to the joint that it spans. The rotary force exerted by a muscle about a joint is referred to as *muscle torque* and is calculated as the product of the muscle force and the *moment arm*, the shortest perpendicular distance from the line of pull of the muscle to the joint's center (Figure 31–14).

The moment arm usually changes as a joint rotates through its range of motion; the amount of change depends on where the muscle is attached to the skeleton relative to the joint. If the force exerted by a muscle remains relatively constant throughout the joint's range of motion, muscle torque varies in direct proportion to the change in the moment arm. For many muscles, the moment arm is maximal in the middle of the range of motion, which usually corresponds to the position of maximal muscle force and hence greatest muscle torque.

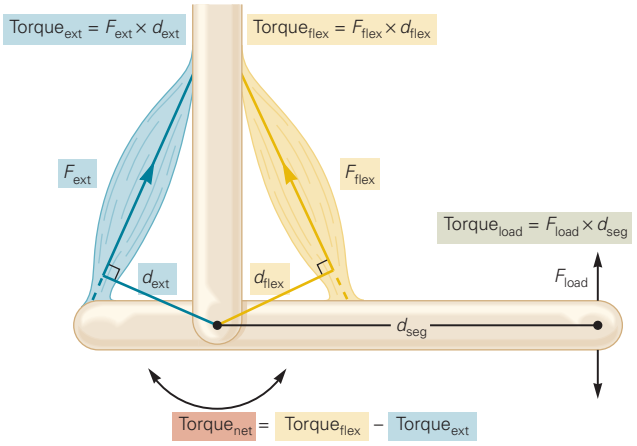


Figure 31–14 Muscle torque varies over the range of motion about a joint. A muscle's torque about a joint is the product of its contractile force (F) and its moment arm relative to the joint (d). The moment arm is the shortest perpendicular distance from the line of pull of the muscle to the center of rotation of the joint. Because the moment arm changes when the joint rotates, muscle torque varies with angular displacement about the joint. The net torque about a joint, which determines the mechanical action, is the difference in the torques exerted by opposing muscles, such as extensors (ext) and flexors (flex). Similarly, a force applied to the limb (F_{load}) will exert a torque about the joint that depends on F_{load} and its distance from the joint (d_{seg}).

Different Movements Require Different Activation Strategies

The human body has approximately 600 muscles, each with a distinct torque profile about one or more joints. To perform a desired movement, the nervous system must activate an appropriate combination of muscles with adequate intensity and timing of activity. The activation must be appropriate for the contractile properties and musculoskeletal geometry of many muscles, as well as the mechanical interactions between body segments. As a result of these demands, activation strategies differ with the details of the movement.

Contraction Velocity Can Vary in Magnitude and Direction

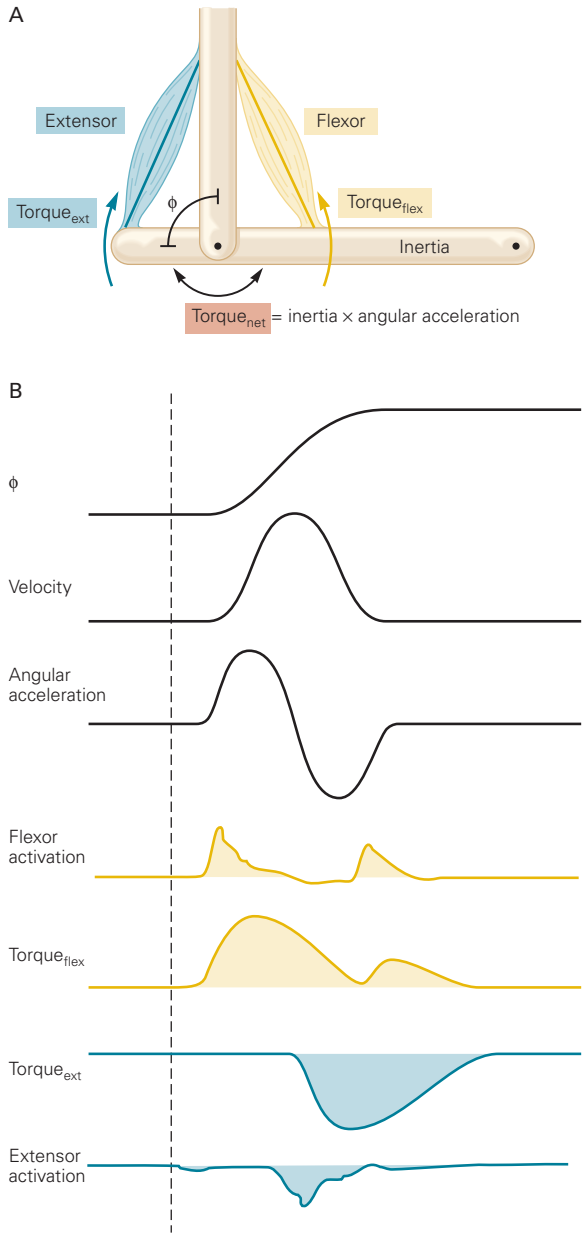
Movement speed depends on the contraction velocity of a muscle. The only ways to vary contraction velocity are to alter either the number of motor units recruited or the rates at which they discharge action potentials. The velocity of a contraction can vary in both magnitude and direction (see Figure 31–11B). To control the velocity of a contraction, the nervous system must scale the magnitude of the net muscle torque relative to the load torque (Figure 31–14), which includes both the weight of the body part and any external load acting on the body.

When muscle torque exceeds load torque, the muscle shortens as it performs a shortening contraction. When muscle torque is less than load torque, the muscle lengthens as it performs a lengthening contraction. For the example shown in Figure 31–14, the load is lifted with a shortening contraction of the flexor and lowered with a lengthening contraction of the flexor. Both types of contractions are common in daily activities.

Shortening and lengthening contractions are not simply the result of adjusting motor unit activity so that the net muscle torque is greater or less than the load torque. When the task involves lifting a load with a prescribed trajectory, activation of the motor units must be aligned so that the sum of the rise times produce the appropriate torque so as to match the desired trajectory while lifting (shortening contractions), whereas while lowering the load (lengthening contractions), the sum of the decay times must be similarly

controlled. The nervous system accomplishes this with different descending input and sensory feedback during the two types of contractions. Because of these differences in required motor unit activity, the control of the two types of contraction respond differently to stresses imposed on the system. Declines in the capacity to control motor unit activity, such as observed in older adults and persons performing rehabilitation exercises after an orthopedic procedure, are associated with greater difficulty in performing lengthening contractions.

The amount of motor unit activity relative to the load also influences the contraction velocity. This effect depends on both the number of motor units recruited and the maximal rates at which the motor units can discharge action potentials. As described previously, physical training with rapid contractions, such as power training, increases the rate at which motor units can discharge trains of action potentials, which can be mimicked by step injections of current into a motor neuron. Changes in the maximal shortening velocity of a muscle after a change in the habitual level of physical activity are the result, at least partly, of factors that influence the ability of motor neurons to discharge action potentials at high rates.



Movements Involve the Coordination of Many Muscles

In the simplest case, muscles span a single joint and cause the attached body segments to accelerate about a single axis of rotation. Because muscles can exert only a pulling force, motion about a single axis of rotation requires at least two muscles or groups of muscles when the action involves shortening contractions (Figure 31–15A).

Because most muscles attach to the skeleton slightly off center from the axis of rotation, they can cause movement about more than one axis of rotation. If one of the actions is not required, the nervous system must activate other muscles to control the unwanted movement. For example, activation of the radial flexor muscle of the wrist can cause the wrist to flex and abduct. If the intended action is only wrist flexion, then the abduction action must be opposed by another muscle, such as the ulnar flexor muscle, which causes wrist flexion and adduction. Depending on the geometry of the articulating surfaces and the attachment sites of the muscles, the multiple muscles that span a joint are capable of producing movements about one to three axes of rotation. Furthermore, some structures

Figure 31–15 (Left) Antagonist muscles spanning a single joint control movement of a limb about a single axis of rotation.

A. According to Newton's law of acceleration (force = mass × acceleration), force is required to change the velocity of a mass. Muscles exert a torque to accelerate the inertial mass of the skeletal segment around a joint. For angular motion, Newton's law is written as torque = rotational inertia × angular acceleration.

B. The angular velocity for movement of a limb from one position to another has a bell-shaped profile. Acceleration in one direction is followed by acceleration in the opposite direction—the flexor and extensor muscles are activated in succession. The records here show the activation profiles and associated muscle torques for a fast elbow flexion movement. Because contractile force decays relatively slowly, the flexor muscle is usually activated a second time to counter the prolonged acceleration generated by the extensor muscle and to stop the limb at the intended joint angle.

can be displaced linearly (eg, the scapula on the trunk), adding to the degrees of freedom about a joint.

The off-axis attachment of muscles enhances the flexibility of the skeletal motor system; the same movement can be achieved by activating different combinations of muscles. However, this additional flexibility requires the nervous system to control the unwanted actions. A solution used by the nervous system is to organize relations among selected muscles to produce specific actions. A particular sequence of muscle activations is known as a *muscle synergy,* and movement is produced through the coordinated activation of these synergies. For example, EMG recordings of human subjects suggest that variations of movements with the same general purpose, such as grasping various objects with the hand, reaching and pointing in different directions, or walking and running at several speeds, are controlled by approximately five muscle synergies.

The number of muscles that participate in a movement also varies with the speed of the movement. For example, slow lifting of a load requires only that the muscle torque slightly exceed the load torque (see Figure 31–14), and thus, only the flexor muscle is activated. This strategy is used when lifting a handheld weight with the elbow flexor muscles. In contrast, to perform this movement rapidly with an abrupt termination at an intended joint angle, both the flexor and extensor muscles must be activated. First, the flexor muscle is activated to accelerate the limb in the direction of flexion, followed by activation of the extensor muscle to accelerate the limb in the direction of extension, and finally a burst of activity by the flexor muscle to increase the angular momentum of the limb and the handheld weight in the direction of flexion so that it arrives at the desired joint angle (Figure 31–15B). The amount of extensor muscle activity increases with the speed of the movement.

Increases in movement speed introduce another factor that the nervous system must control: unwanted accelerations in other body segments. Because body parts are connected to one another, motion in one part can induce motion in another. The induced motion is often controlled with lengthening contractions, such as

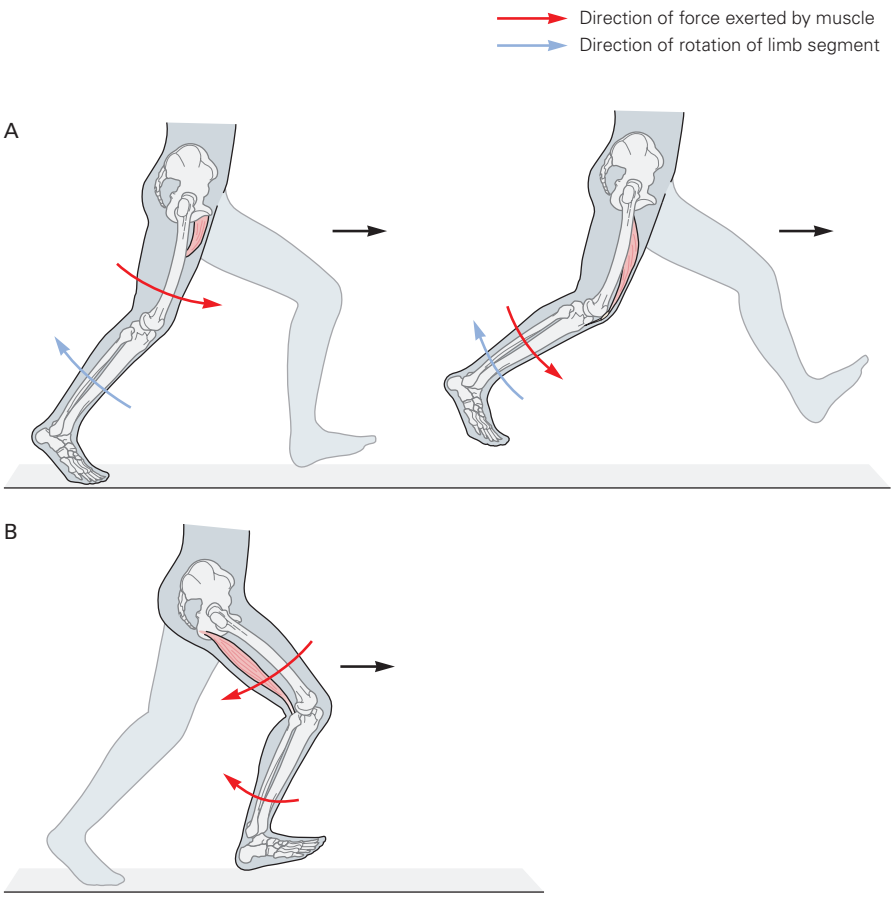


Figure 31–16 A single muscle can influence the motion about many joints.

A. Muscles that cross one joint can accelerate an adjacent body segment. For example, at the beginning of the swing phase while running, the hip flexor muscles are activated to accelerate the thigh forward (**red arrow**). This action causes the lower leg to rotate backward (**blue arrow**) and the knee joint to flex. To control the knee joint flexion during the first part of the swing phase, the knee extensor muscles are activated and undergo a lengthening contraction to accelerate the lower leg forward (**red arrow**) while it continues to rotate backward (**blue arrow**).

B. Many muscles cross more than one joint to exert an effect on more than one body segment. For example, the hamstring muscles of the leg accelerate the hip in the direction of extension and the knee in the direction of flexion (**red arrows**). During running, at the end of the swing phase, the hamstring muscles are activated and undergo lengthening contractions to control the forward rotation of the leg (hip flexion and knee extension). This strategy is more economical than activating individual muscles at the hip and knee joints to control the forward rotation of the leg.

those experienced by thigh muscles during the swing phase of running (Figure 31–16A).

Muscles that span more than one joint can be used to control these motion-dependent interactions between body parts. At the end of the swing phase in running, activation of the hamstring muscles causes both the thigh and lower leg to accelerate backward (Figure 31–16B). If a hip extensor muscle is used to accelerate the thigh backward instead of the hamstring muscles, the lower leg would accelerate forward, requiring activation of a knee flexor muscle to control the unwanted lower leg motion so that the foot could be placed on the ground. Use of the two-joint hamstring muscles is a more economical strategy, but one that can subject the hamstrings to high stresses during fast movements, such as sprinting. The control of such motion-dependent interactions often involves lengthening contractions, which maximize muscle stiffness and the ability of muscle to resist changes in length.

For most movements, the nervous system must establish rigid connections between some body parts for two reasons. First, as expressed in Newton's law of action and reaction, a reaction force must provide a foundation for the acceleration of a body part. For example, in a reaching movement performed by a person standing upright, the ground must provide a reaction force against the feet. The muscle actions that produce the arm movement exert forces that are transmitted through the body to the feet and are opposed by the ground. Different substrates provide different amounts of reaction force, which is why ice or sand can influence movement capabilities.

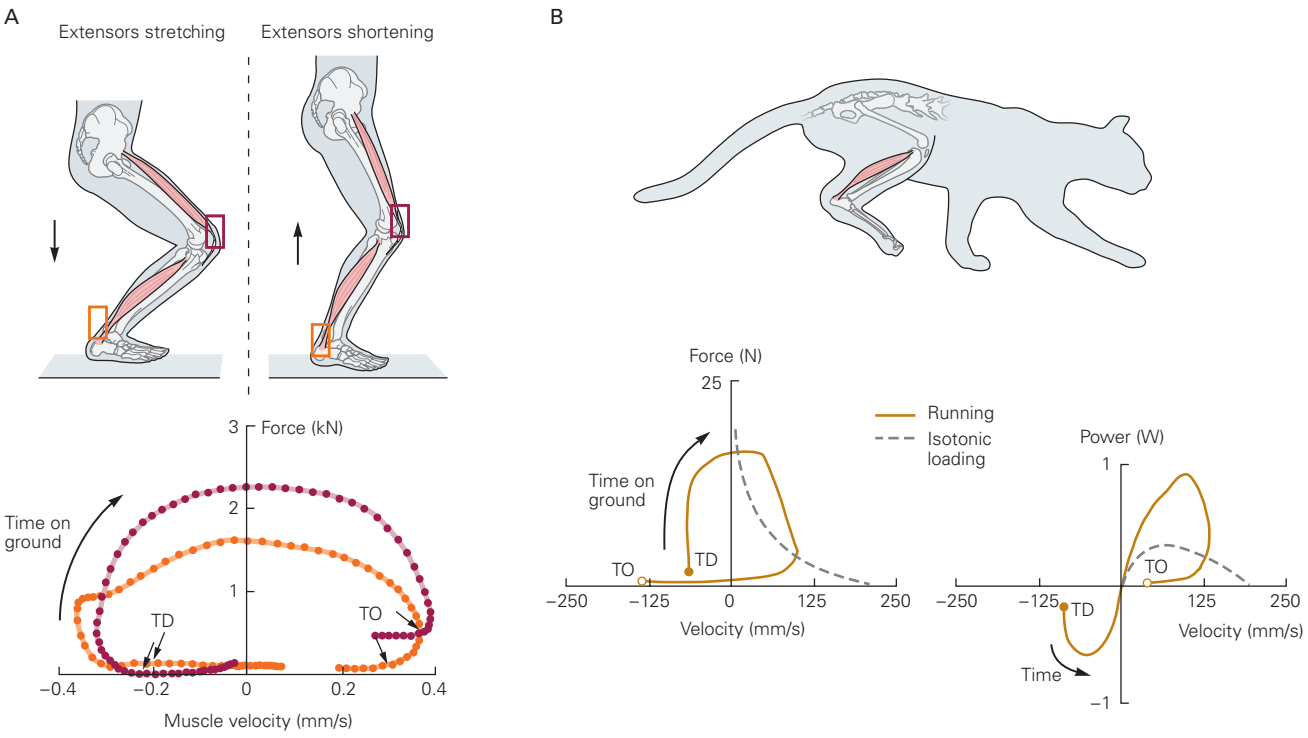


Figure 31–17 **An initial phase of negative work augments subsequent positive work performed by the muscle.** (Reproduced, with permission, from Finni, Komi, and Lepola 2000. Copyright © 2000, Springer-Verlag; Gregor et al. 1988.)

A. The force in the Achilles tendon (**orange**) and patellar tendon (**purple**) vary during the ground-contact phase of two-legged hopping. The feet contact the ground at touchdown (**TD**) and leave the ground at toe-off (**TO**). For approximately the first half of the movement, the quadriceps and triceps surae muscles lengthen, performing negative work (negative velocity). The muscles perform positive work when they shorten (positive velocity). The sites of force transducer measurements are indicated by rectangles.

B. The force exerted by the soleus muscle of a cat running at moderate speed varies from the instant the paw touches the ground (**TD**) until it leaves the ground (**TO**). The force exerted by the muscle during the shortening contraction (positive velocity) is greater than the peak forces measured when the muscle contracts maximally against various constant loads (isotonic loading). Negative velocity reflects a lengthening contraction in the soleus muscle. The power produced by the soleus muscle of the cat during running is greater than that produced in an isolated-muscle experiment (**dashed line**). The phase of negative power corresponds to the lengthening contraction just after the paw is placed on the ground (TD), when the muscle performs negative work.

Second, uncertain conditions are usually accommodated by stiffening the joints through concurrent activation of the muscles that produce force in opposite directions. Coactivation of opposing muscles occurs often when a support surface is unsteady, when the body might experience an unexpected perturbation, or when lifting a heavy load. Because coactivation increases the energetic cost of performing a task, one characteristic of skilled performance is the ability to accomplish a task with minimal activation of muscles that produce opposing actions.

Muscle Work Depends on the Pattern of Activation

Limb muscles in healthy young adults are active 10% to 20% of the time during waking hours. For much of this time, the muscles perform constant-length (*isometric*) contractions to maintain a variety of static body postures. In contrast, muscle length has to change during a movement so that the muscle can perform work to displace body parts. A muscle performs positive work and produces power during a shortening contraction, whereas it performs negative work and absorbs power during a lengthening contraction. The capacity of muscle to do positive work establishes performance capabilities, such as the maximal height that can be jumped.

The nervous system can augment the positive work capacity of muscle by commanding a brief period of negative work before performing positive work. This activation sequence, the *stretch–shorten cycle,* occurs in many movements. When a person jumps in place on two feet, for example, the support phase involves an initial stretch (lengthening) and subsequent shortening of the ankle extensor and knee extensor muscles (Figure 31–17A). The forces in the Achilles and patellar tendons increase during the stretch of the lengthening contraction and reach a maximum at the onset of the shortening phase. As a result, the muscles can perform more positive work and produce more power during the shortening contraction (Figure 31–17B).

Although negative work involves an increase in the length of the muscle, the length of the fascicles in the muscle often remains relatively constant, which indicates that the connective tissue structures are stretched prior to the shortening contraction. Thus, the capacity of the muscle to perform more positive work comes from strain energy that can be stored in the elastic elements of muscle and tendon during the stretch phase and released during the subsequent shortening phase. More strain energy can be stored in long tendons, but short tendons are more advantageous when the movement requires the rapid release of strain energy.

Highlights

1. The basic functional unit for the control of movement by the nervous system is the motor unit, which comprises a motor neuron and the muscle fibers it innervates.
2. The force exerted by a muscle depends in part on the number and properties of the motor units that are activated and the rates at which they discharge action potentials. The key motor unit properties include contraction speed, maximal force, and fatigability, all of which can be altered by physical activity. Motor unit properties vary continuously across the population that innervates each muscle; that is, there are not distinct types of motor units. Due to technological advances, it is becoming possible to characterize the adaptations exhibited by populations of motor units in response to different types of changes in physical activity.
3. Motor units tend to be activated in a stereotypical order that is highly correlated with motor neuron size. The rate at which motor units are recruited during a voluntary contraction increases with contraction speed.
4. The rate at which a motor unit discharges action potentials in response to a given synaptic input can be modulated by descending inputs from the brain stem. The modulatory input is likely critical for establishing the level of excitation in spinal pathways, but this has been difficult to demonstrate in humans.
5. Except at low muscle forces, variation in discharge rate has a greater influence on muscle force than does the number of activated motor units. Moreover, the variability in discharge rate of the motor unit population influences the level of fine motor control.
6. The sarcomere is the smallest element of muscle to include a complete set of contractile proteins. A transient connection between the contractile proteins myosin and actin, known as the cross-bridge cycle, enables muscle to exert a force. The organization of the sarcomeres within a muscle varies substantially and, in addition to motor unit activity, has a major effect on the contractile properties of the muscle.
7. For a given arrangement of sarcomeres, the force a muscle can exert depends on the activation of the cross bridges by Ca^{2+}, the amount of overlap between the thick and thin filaments, and the velocity of the moving filaments. Sarcomere force during lengthening contractions is augmented by a Ca^{2+}-mediated increase in titin stiffness. The force

produced by activated sarcomeres depends on the interactions of three filaments: actin, myosin, and titin.

8. Most of the force generated by activated sarcomeres is transmitted laterally through a network of noncontractile proteins that maintains the alignment of the thick and thin filaments.
9. The functional capability of a muscle depends on the torque that it can exert, which is influenced both by its contractile properties and by the location of its attachments on the skeleton relative to the joint that it spans.
10. To perform a movement, the nervous system activates multiple muscles and controls the torque exerted about the involved joints. The nervous system can vary the magnitude and direction of a movement by altering the amount of motor unit activity, and hence muscle torque, relative to the load acting on the body.
11. Although muscle exerts only a pulling force on the skeleton, it can do so whether the activated muscle shortens or is lengthened by a load torque that exceeds the muscle torque. The force capacity of muscle is greater during lengthening contractions. Motor unit activity differs during shortening and lengthening contractions, but little is known about how the synaptic inputs to motor neurons differ during these two types of contractions.
12. Faster movements elicit motion-dependent interactions between body parts that produce unwanted accelerations. These actions must be controlled by the nervous system to produce an intended movement.
13. The nervous system must coordinate the activity of multiple muscles to provide a mechanical link between moving body parts and the required support from the surroundings. The muscles engaged for each action, such as grasping, reaching, running, and walking, are organized into a few sets that exhibit a stereotypical pattern of activation, but it is not known why particular patterns are preferred.
14. The patterns of muscle activity vary substantially between movements and often include strategies that augment the work capacity of muscles. The patterns can be modified by experience, but little is known about the locus of the adaptations other than that both spinal and supraspinal pathways are involved.

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Selected Reading

Booth FW. 2015. Muscle adaptation to exercise: new Saltin's paradigms. Scand J Med Sci Sports Suppl 4:49–52.

Duchateau J, Enoka RM. 2011. Human motor unit recordings: origins and insights into the integrated motor system. Brain Res 1409:42–62.

Enoka RM. 2015. *Neuromechanics of Human Movement.* 5th ed. Champaign, IL: Human Kinetics.

Farina D, Negro F, Muceli S, Enoka RM. 2016. Principles of motor unit physiology evolve with advances in technology. Physiology 31:83–94.

Gordon AM, Regnier M, Homsher E. 2001. Skeletal and cardiac muscle contractile activation: tropomyosin "rocks and rolls." News Physiol Sci 16:49–55.

Heckman CJ, Enoka RM. 2012. Motor unit. Compr Physiol 2:2629–2682.

Herzog W, Schappacher G, DuVall M, Leonard TR, Herzog JA. 2016. Residual force enhancement following eccentric contractions: a new mechanism involving titin. Physiology 31:300–312.

Hunter SK, Pereira HM, Keenan KG. 2016. The aging neuromuscular system and motor performance. J Appl Physiol 121:982–995.

Huxley AF. 2000. Mechanics and models of the myosin motor. Philos Trans R Soc Lond B Biol Sci 355:433–440.

Lieber RL, Ward SR. 2011. Skeletal muscle design to meet functional demands. Philos Trans R Soc Lond B Biol Sci 366:1466–1476.

Merletti R, Muceli S. 2020. Tutorial. Surface EMG detection in space and time: best practices. J Electromyogr Kinesiol 49:102363.

Narici M, Franchi M, Maganaris C. 2016. Muscle structural assembly and functional consequences. J Exp Biol 219:276–284.

References

Alexander RM, Ker RF. 1990. The architecture of leg muscles. In: JM Winters, SL-Y Woo (eds). *Multiple Muscle Systems: Biomechanics and Movement Organization*, pp. 568–577. New York: Springer-Verlag.

Azizi E, Roberts TJ. 2012. Geared up to stretch: pennate muscle behavior during active lengthening. J Exp Biol 217:376–381.

Bloom W, Fawcett DW. 1975. *A Textbook of Histology.* 10th ed. Philadelphia, PA: Saunders.

Botterman BR, Iwamoto GA, Gonyea WJ. 1986. Gradation of isometric tension by different activation rates in motor units of cat flexor carpi radialis muscle. J Neurophysiol 56:494–506.

Bottinelli R, Canepari M, Pellegrino MA, Reggiani C. 1996. Force-velocity properties of human skeletal muscle fibres: myosin heavy chain isoform and temperature dependence. J Physiol 495:573–586.

Del Vecchio A, Negro F, Felici F, Farina D. 2017. Associations between motor unit action potential parameters and surface EMG features. J Appl Physiol 123:835–843.

Desmedt JE, Godaux E. 1977. Ballistic contractions in man: characteristic recruitment pattern of single motor units of the tibialis anterior muscle. J Physiol 264:673–693.

Duchateau J, Enoka RM. 2016. Neural control of lengthening contractions. J Exp Biol 219:197–204.
Enoka RM, Duchateau J. 2016. Translating muscle fatigue to human performance. Med Sci Sport Exerc 48:2228–2238.
Enoka RM, Duchateau J. 2017. Rate coding and the control of muscle force. Cold Spring Harb Perspect Med 27:1–12.
Enoka RM, Fuglevand AJ. 2001. Motor unit physiology: some unresolved issues. Muscle Nerve 24:4–17.
Erim Z, De Luca CJ, Mineo K, Aoki T. 1996. Rank-ordered regulation of motor units. Muscle Nerve 19:563–573.
Farina D, Merletti R, Enoka RM. 2014. The extraction of neural strategies from the surface EMG: an update. J Appl Physiol 117:1215–1230.
Finni T, Komi PV, Lepola V. 2000. In vivo human triceps surae and quadriceps muscle function in a squat jump and counter movement jump. Eur J Appl Physiol 83:416–426.
Fuglevand AJ, Macefield VG, Bigland-Ritchie B. 1999. Force-frequency and fatigue properties of motor units in muscles that control digits of the human hand. J Neurophysiol 81:1718–1729.
Gordon AM, Regnier M, Homsher R. 2001. Skeletal and cardiac muscle contractile activation: tropomyosin "rocks and rolls." News Physiol Sci 16:49–55.
Gregor RJ, Roy RR, Whiting WC, Lovely RG, Hodgson JA, Edgerton VR. 1988. Mechanical output of the cat soleus during treadmill locomotion: in vivo vs in situ characteristics. J Biomech 21:721–732.
Heckman CJ, Mottram C, Quinlan K, Theiss R, Schuster J. 2009. Motoneuron excitability: the importance of neuromodulatory inputs. Clin Neurophysiol. 120:2040–2054.
Henneman E, Somjen G, Carpenter DO. 1965. Functional significance of cell size in spinal motoneurons. J Neurophysiol 28:560–580.
Hepple RT, Rice CL. 2016. Innervation and neuromuscular control in ageing skeletal muscle. J Physiol 594:1965–1978.
Huxley AF, Simmons RM. 1971. Proposed mechanism of force generation in striated muscle. Nature 233:533–538.
Kubo K, Miyazaki D, Shimoju S, Tsunoda N. 2015. Relationship between elastic properties of tendon structures and performance in long distance runners. Eur J Appl Physiol 115:1725–1733.
Lai A, Schache AG, Lin YC, Pandy MG. 2014. Tendon elastic strain energy in the human ankle plantar-flexors and its role with increased running speed. J Exp Biol 217:3159–3168.
Lieber RL, Fridén J. 2000. Functional and clinical significance of skeletal muscle architecture. Muscle Nerve 23:1647–1666.
Lieber RL, Ward SR. 2011. Skeletal muscle design to meet functional demands. Philos Trans R Soc Lond B Biol Sci 366:1466–1476.
Liddell EGT, Sherrington CS. 1925. Recruitment and some other factors of reflex inhibition. Proc R Soc Lond B Biol Sci 97:488–518.
Maas H, Finni T. 2018. Mechanical coupling between muscle-tendon units reduces peak stresses. Exerc Sport Sci Rev 46:26–33.
Macefield VG, Fuglevand AJ, Bigland-Ritchie B. 1996. Contractile properties of single motor units in human toe extensors assessed by intraneural motor axon stimulation. J Neurophysiol 75:2509–2519.
Milner-Brown HS, Stein RB, Yemm R. 1973. The orderly recruitment of human motor units during voluntary isometric contraction. J Physiol 230:359–370.
Moritz CT, Barry BK, Pascoe MA, Enoka RM. 2005. Discharge rate variability influences the variation in force fluctuations across the working range of a hand muscle. J Neurophysiol 93:2449–2459.
O'Connor SM, Cheng EJ, Young KW, Ward SR, Lieber RL. 2016. Quantification of sarcomere length distribution in whole muscle frozen sections. J Exp Biol 219:1432–1436.
Overduin SA, d'Avella A, Roh J, Carmena JM, Bizzi E. 2015. Representation of muscle synergies in the primate brain. J Neurosci 35:12615–12624.
Palmisano MG, Bremner SN, Homberger TA, et al. 2015. Skeletal muscle intermediate filaments form a stress-transmitting and stress-signalling network. J Cell Sci 128: 219–224.
Person RS, Kudina LP. 1972. Discharge frequency and discharge pattern of human motor units during voluntary contraction of muscle. Electroencephalogr Clin Neurophysiol 32:471–483.
Sawicki GS, Robertson BD, Azizi E, Roberts TJ. 2015. Timing matters: tuning the mechanics of a muscle-tendon unit by adjusting stimulation phase during cyclic contractions. J Exp Biol 218:3150–3159.
Schache AG, Dorn TW, Williams GP, Brown NA, Pandy MG. 2014. Lower-limb muscular strategies for increasing running speed. J Orthop Sports Phys Ther 44:813–824.
Sherrington CS. 1925. Remarks on some aspects of reflex inhibition. Proc R Soc Lond B Biol Sci 97:519–545.
Trappe S, Costill D, Gallagher P, et al. 2009. Exercise in space: human skeletal muscle after 6 months aboard the International Space Station. J Appl Physiol 106:1159–1168.
Trappe S, Luden N, Minchev K, Raue U, Jemiolo B, Trappe TA. 2015. Skeletal muscle signature of a champion sprint runner. J Appl Physiol 118:1460–1466.
Van Cutsem M, Duchateau J, Hainaut K. 1998. Changes in single motor unit behaviour contribute to the increase in contraction speed after dynamic training in humans. J Physiol 513:295–305.
Van Cutsem M, Feiereisen P, Duchateau J, Hainaut K. 1997. Mechanical properties and behaviour of motor units in the tibialis anterior during voluntary contractions. Can J Appl Physiol 22:585–597.
Ward SR, Eng CM Smallwood LH, Lieber RL. 2009. Are current measurements of lower extremity muscle architecture accurate? Clin Orthop Rel Res 467:1074–1082.