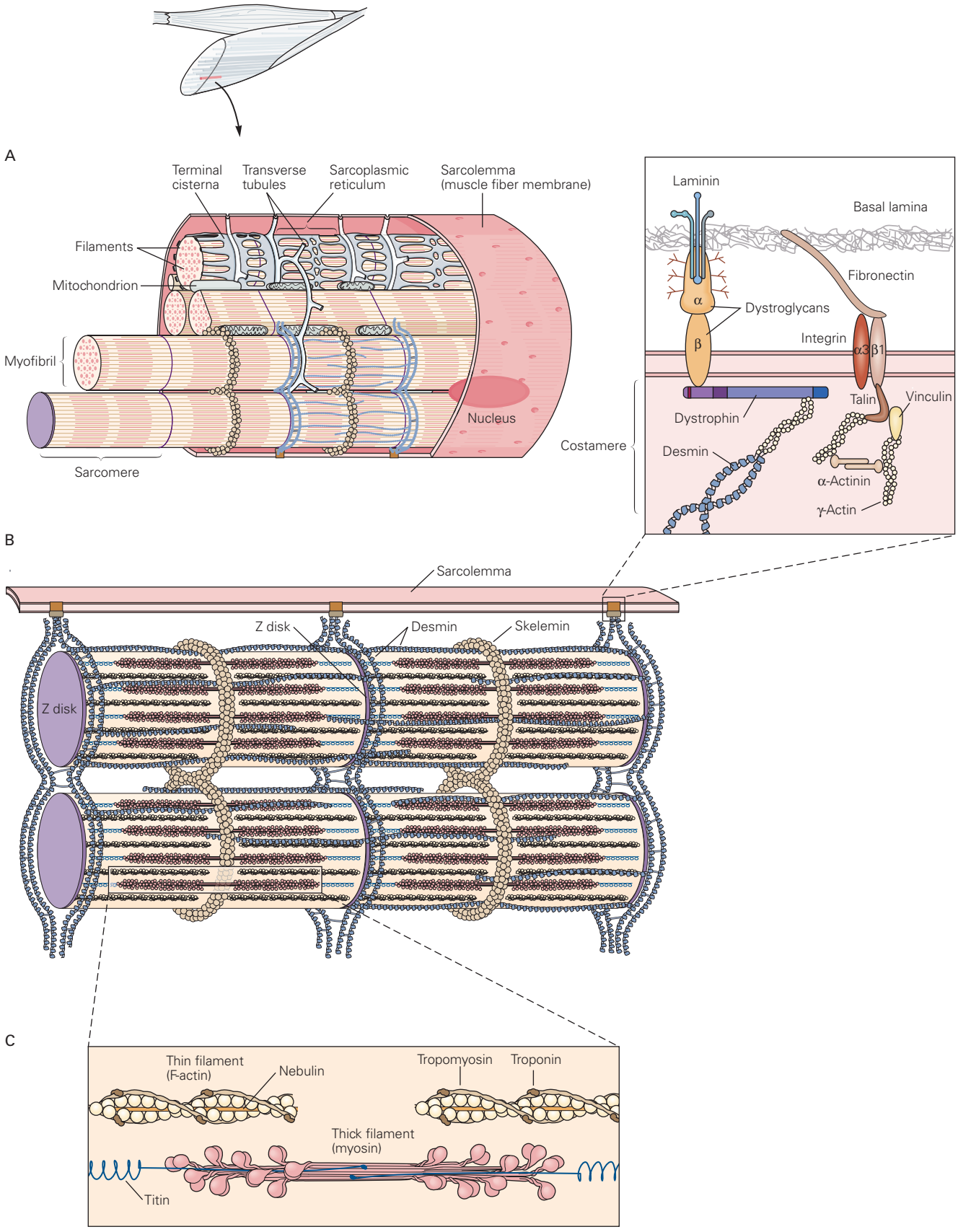

A
Terminal cisterna
Transverse tubules
Sarcoplasmic reticulum
Sarcolemma (muscle fiber membrane)
Filaments
Mitochondrion
Myofibril
Nucleus
Sarcomere
Laminin
Basal lamina
Fibronectin
α
Dystroglycans
β
Integrin
α3β1
Talin
Vinculin
Dystrophin
Costamere
Desmin
α-Actinin
γ-Actin
B
Sarcolemma
Z disk
Desmin
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.

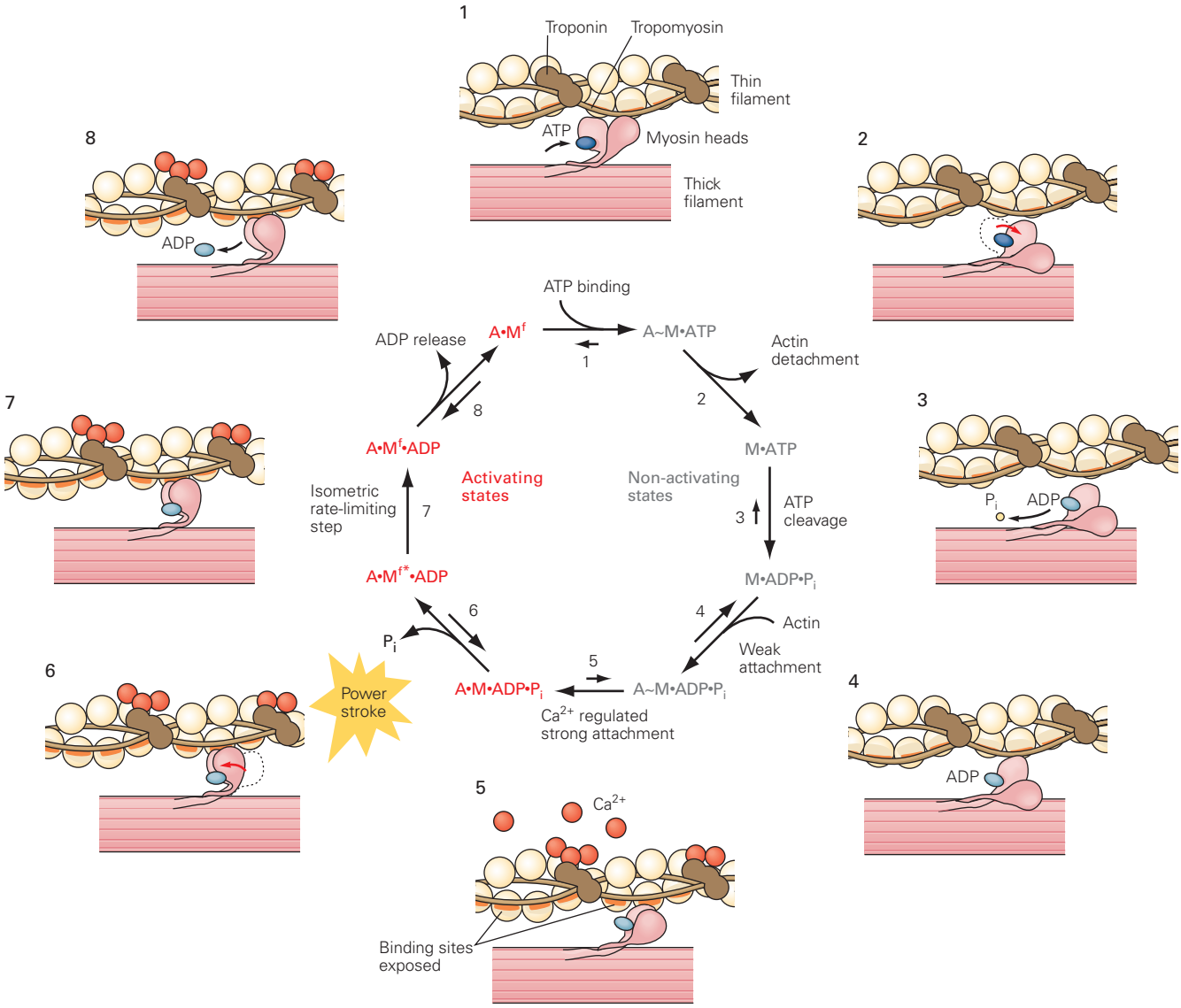


Figure 31–10 **The cross-bridge cycle.** Several nonactivating states are followed by several activating states triggered by Ca^{2+}. The cycle begins at the top (**step 1**) with the binding of adenosine triphosphate (**ATP**) to the myosin head. The myosin head detaches from actin (**step 2**), ATP 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

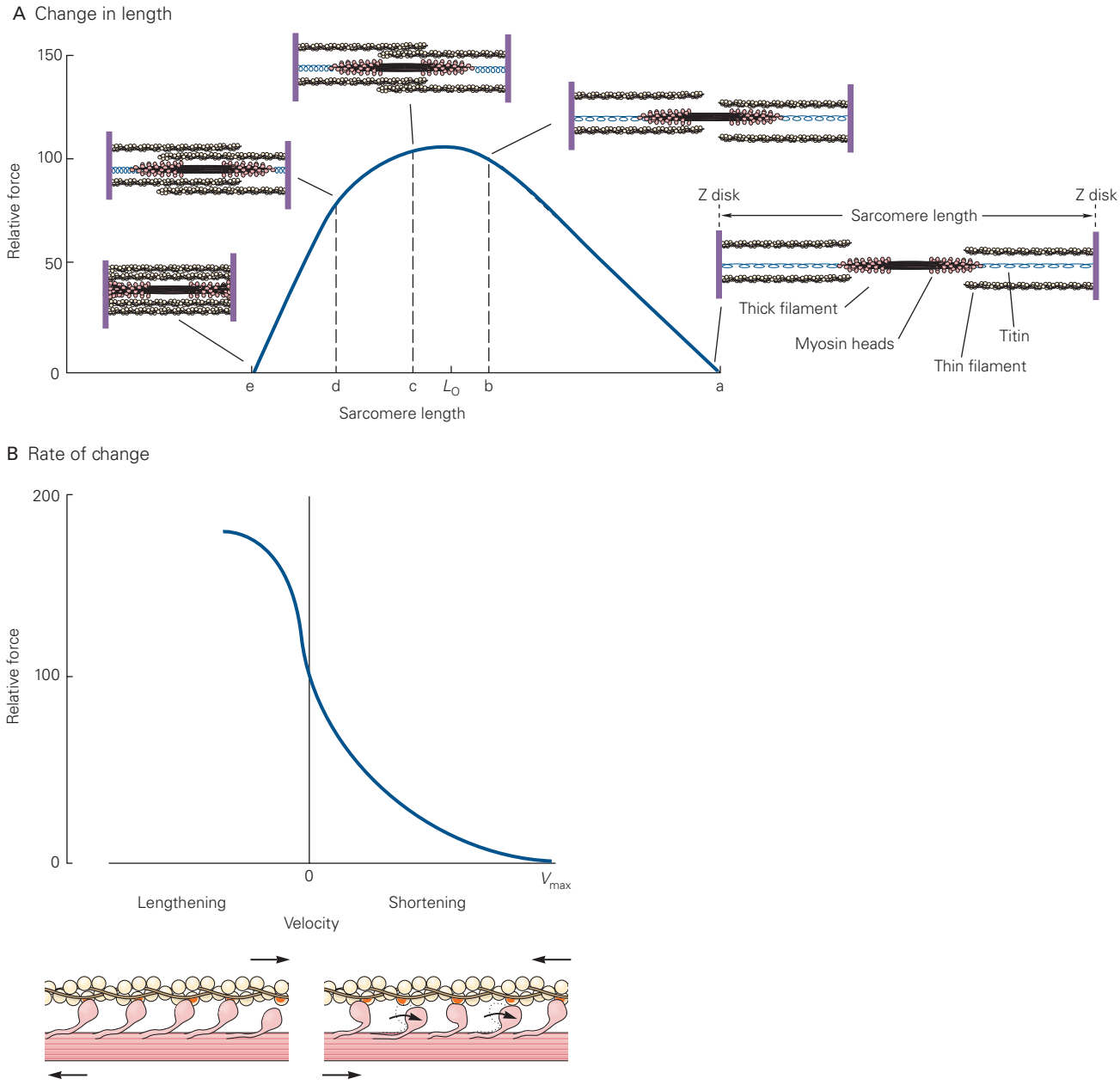


Figure 31–11 Contractile force varies with the change in sarcomere length and velocity.

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.

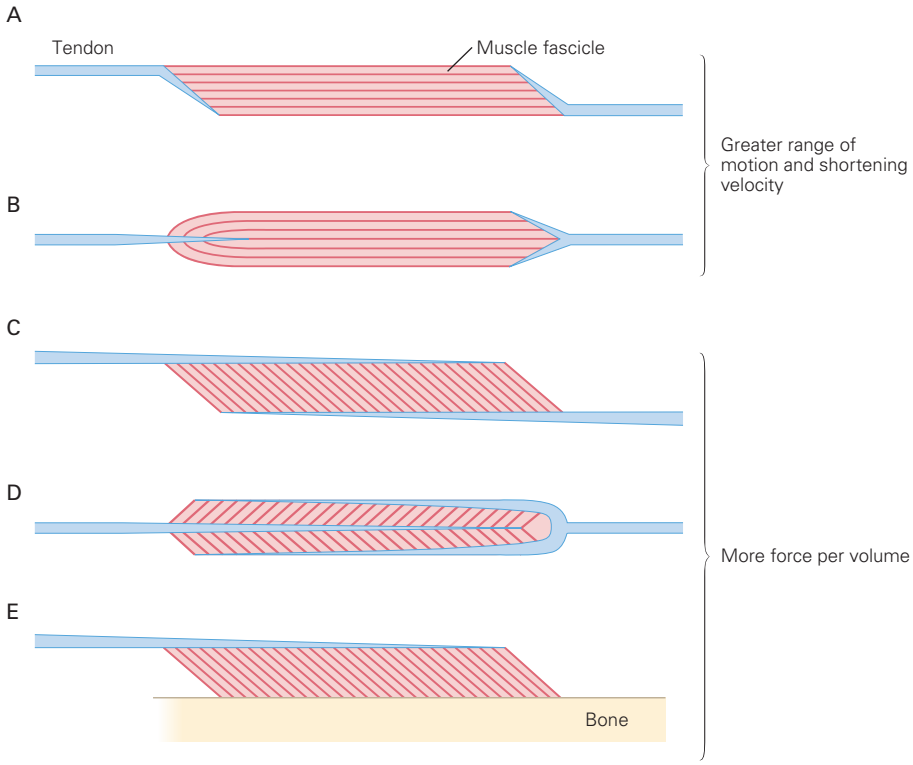


Figure 31–12 Five common arrangements of tendon and muscle. The fundamental distinction between these arrangements is whether or not the muscle fascicles are aligned with the line of pull of the muscles. The fascicles in muscles A and B are parallel to the line of pull (longitudinal axis of the muscle), 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,

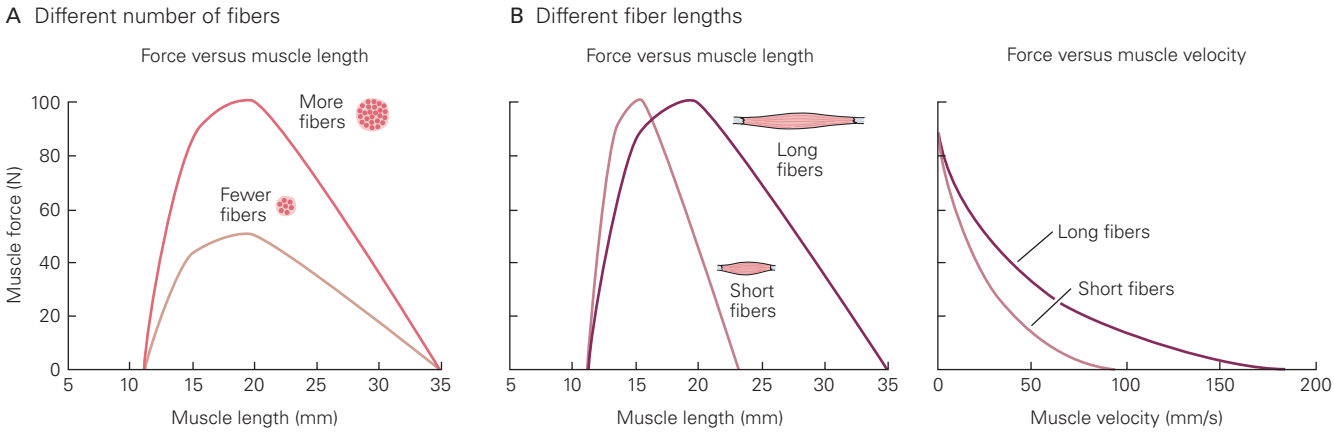


Figure 31–13 **Muscle dimensions influence the peak force and maximal shortening velocity.** (Reproduced, with permission, from Lieber and Fridén 2000. Copyright © 2000 John Wiley & Sons, Inc.)

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.

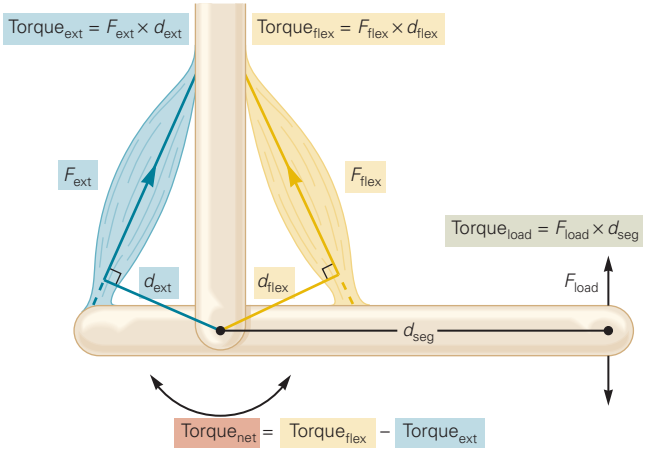


Figure 31–14 Muscle torque varies over the range of motion about a joint. A muscle's torque about a joint is the product of its contractile force (F) and its moment arm relative to the joint (d). The moment arm is the shortest perpendicular distance from the line of pull of the muscle to the center of 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

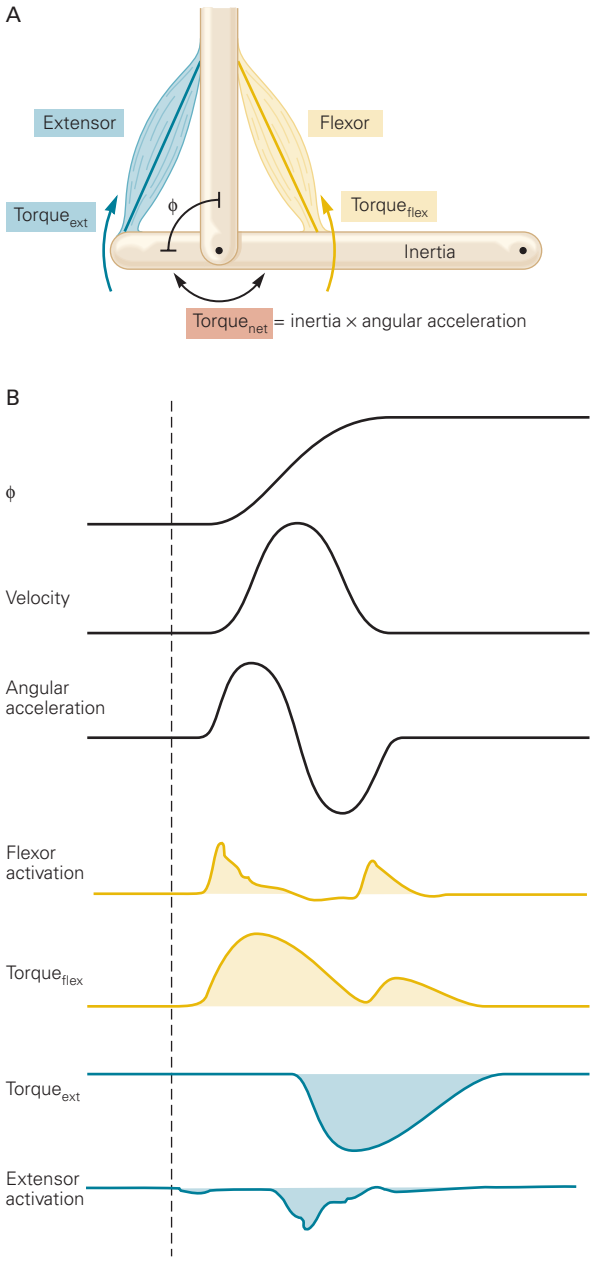
Movement speed depends on the contraction velocity of a muscle. The only ways to vary contraction velocity are to alter either the number of motor units recruited or the rates at which they discharge action potentials. The velocity of a contraction can vary in both magnitude and direction (see Figure 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

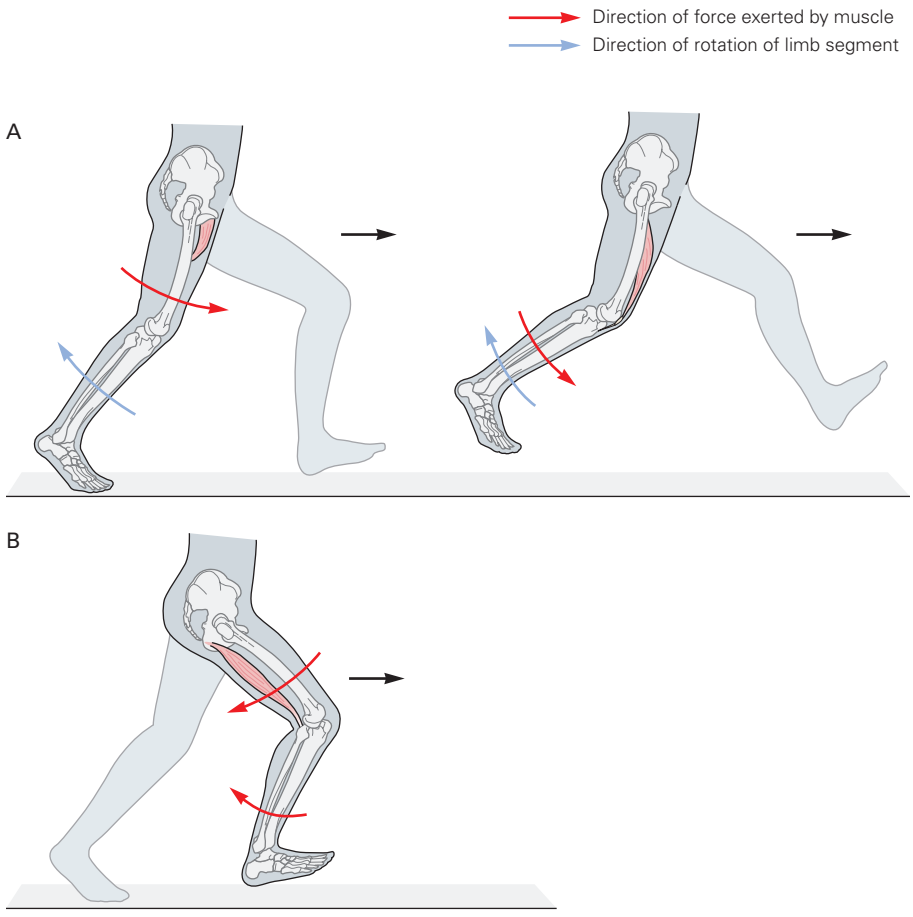


Figure 31–16 A single muscle can influence the motion about many joints.

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.