Control of Body Movement

CHAPTER 10

Tracking and striking a soccer ball require a sophisticated system of motor control. ©Erik Isakson/Blend Images/Getty Images

Previous chapters described the complex structure and functions of the nervous system (Chapters 6–8) and skeletal muscles (Chapter 9). In this chapter, you will learn how those systems interact with each other in the initiation and control of body movements. Consider the events associated with reaching out and grasping an object. The trunk is inclined toward the object, and the wrist, elbow, and shoulder are extended (straightened) and stabilized to support the weight of the arm and hand, as well as the object. The fingers are extended to reach around the object and then flexed (bent) to grasp it. The degree of extension will depend upon the size of the object, and the force of flexion will depend upon its weight and consistency (for example, you would grasp an egg less tightly than a rock). Through all this, the body maintains upright posture and balance despite its continuously shifting position.

As described in Chapter 9, the building blocks for these movements—as for all movements—are motor units, each comprising one motor neuron together with all the skeletal muscle fibers innervated by that neuron. The motor neurons are the final common pathway out of the central nervous system because all neural influences on skeletal muscle converge on the motor neurons and can only affect skeletal muscle through them. All the motor neurons that supply a given muscle make up the **motor neuron pool** for the muscle. The cell bodies of the

pool for a given muscle are close to each other either in the ventral horn of the spinal cord or in the brainstem.

Within the brainstem or spinal cord, the axon terminals of many neurons synapse on a motor neuron to control its activity. The precision and speed of normally coordinated actions are produced by a balance of excitatory and inhibitory inputs onto motor neurons. For example, if inhibitory synaptic input to a given motor neuron is removed, the excitatory input to that neuron will be unopposed and the motor neuron firing will increase, leading to increased contraction. It is important to realize that movements—even simple movements such as flexing a finger—are rarely achieved by just one muscle. Body movements are achieved by activation, in a precise sequence, of many motor units in various muscles.

This chapter deals with the interrelated neural inputs that converge upon motor neurons to control their activity, and features several of the general principles of physiology described in Chapter 1. Throughout the chapter, signaling along individual neurons and within complex neural networks demonstrates the general principle of physiology that information flow between cells, tissues, and organs is an essential feature of homeostasis and allows for integration of physiological processes. Inputs to motor neurons can be either excitatory or inhibitory, a good example of the general principle of physiology that most physiological functions are controlled by multiple regulatory systems, often working in opposition. Finally, the challenge of maintaining posture and balance against gravity relates to the general principle of physiology that physiological processes are dictated by the laws of chemistry and physics. We first present a general model of how the motor system functions and then describe each component of the model in detail. Keep in mind that many of the contractions that skeletal muscles execute—particularly the muscles involved in postural support—are isometric (Chapter 9). These isometric contractions serve to stabilize body parts rather than to move them but are included in the discussion because they are essential in the overall control of body movements. ■

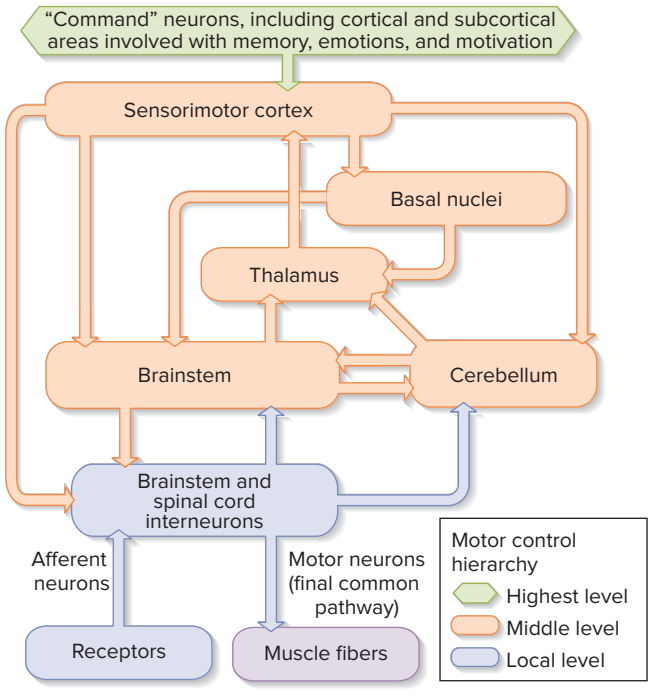
10.1 Motor Control Hierarchy

The neurons involved in controlling skeletal muscles can be thought of as being organized in a hierarchical fashion, with each level of the hierarchy having a certain task in motor control (**Figure 10.1**). To begin a consciously planned movement, a general intention such as "pick up sweater" or "write signature" or "answer telephone" is generated at the highest level of the motor control hierarchy. These higher centers include many regions of the brain (described in detail later), including cortical and subcortical areas involved in memory, emotions, and motivation.

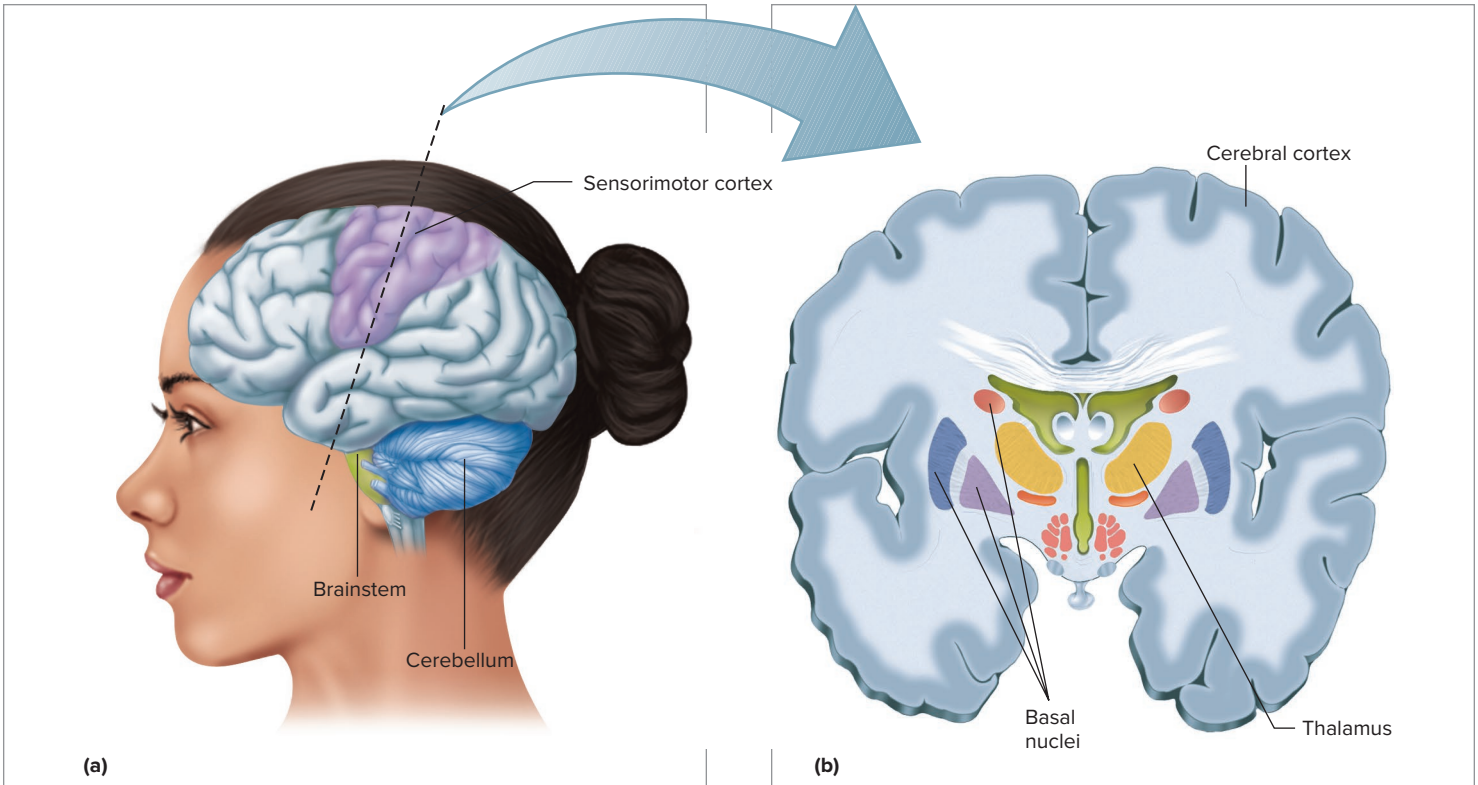
Information is relayed from these higher-center "command" neurons to parts of the brain that make up the middle level of the motor control hierarchy. The middle-level structures specify the individual postures and movements needed to carry out the intended action. In our example of picking up a sweater, structures of the middle hierarchical level coordinate the commands that tilt the body and extend the arm and hand toward the sweater and shift the body's weight to maintain balance. The middle-level hierarchical structures are located in sensory and motor regions of the cerebral cortex as well as in the cerebellum, subcortical nuclei, and brainstem (see Figure 10.1 and **Figure 10.2**). These structures have extensive interconnections, as the arrows in Figure 10.1 indicate.

As the neurons in the middle level of the hierarchy receive input from the command neurons, they simultaneously receive afferent information from receptors in the muscles, tendons, joints, and skin, as well as from the vestibular apparatus and eyes. Utilizing this afferent input, middle-level neurons build an internal model of the pattern of neural activity that will be required to perform a movement (sometimes referred to as a **motor program**). The model integrates information about the starting position of body parts, the nature of the space they will move through, and environmental elements with which they will interact (such as the properties of a diving board). The importance of sensory pathways in planning movements is demonstrated by the fact that when these pathways are impaired, a person has not only sensory deficits but also slow and uncoordinated voluntary movement.

The information determined by the motor program is transmitted via **descending pathways** to the local level of the motor control hierarchy. There, the axons of the motor neurons projecting to the muscles exit the brainstem or spinal cord. The local level of the hierarchy includes afferent neurons, motor neurons, and interneurons. Local-level neurons determine exactly which motor neurons will be activated to achieve the desired action and when this will happen. Note in Figure 10.1 that the descending pathways to the local level arise only in the sensorimotor cortex and brainstem. The term **sensorimotor cortex** is used to describe the widespread regions of the frontal



AP|R **Figure 10.1** Simplified hierarchical organization of the neural systems controlling body movement. Motor neurons control all the skeletal muscles of the body. The sensorimotor cortex includes those parts of the cerebral cortex that act together to control skeletal muscle activity. The middle level of the hierarchy also receives input from the vestibular apparatus and eyes (not shown in the figure).



AP|R **Figure 10.2** (a) Side view of the brain showing three of the five components of the middle level of the motor control hierarchy. (Figure 10.9 shows details of the sensorimotor cortex.) (b) Cross section of the brain showing the cerebral cortex, thalamus, and basal nuclei.

and parietal lobes that act together to control muscle movement. Other brain areas, notably the basal nuclei (also referred to as the basal ganglia), thalamus, and cerebellum, exert their effects on the local level only indirectly via the descending pathways from the cerebral cortex and brainstem.

The motor programs are continuously adjusted during the course of most movements. As the initial motor program begins and the action gets underway, brain regions at the middle level of the hierarchy continue to receive a constant stream of updated afferent information about the movements taking place. Afferent information about the position of the body and its parts in space is called **proprioception.** Say, for example, that the sweater you are picking up is wet and heavier than you expected so that the initially determined strength of muscle contraction is not sufficient to lift it. Any discrepancies between the intended and actual movements are detected, program corrections are determined, and the corrections are relayed to the local level of the hierarchy and the motor neurons. Reflex circuits acting entirely at the local level are also important in refining ongoing movements. Thus, some proprioceptive inputs are processed and influence ongoing movements without ever reaching the level of conscious perception.

If a complex movement is repeated often, learning takes place and the movement becomes skilled. Then, the initial information from the middle hierarchical level is more accurate and fewer corrections need to be made. Movements performed at high speed without concern for fine control are made solely according to the initial motor program.

Table 10.1 summarizes the structures and functions of the motor control hierarchy.

TABLE 10.1 Conceptual Motor Control Hierarchy for Voluntary Movements

I. Higher centers
- A. Function: forms complex plans according to individual's intention and communicates with the middle level via command neurons.
- B. Structures: areas involved with memory, emotions and motivation, and sensorimotor cortex. All these structures receive and correlate input from many other brain structures.

II. The middle level
- A. Function: converts plans received from higher centers to a number of smaller motor programs that determine the pattern of neural activation required to perform the movement. These programs are broken down into subprograms that determine the movements of individual joints. The programs and subprograms are transmitted through descending pathways to the local control level.
- B. Structures: sensorimotor cortex, cerebellum, parts of basal nuclei, some brainstem nuclei.

III. The local level
- A. Function: specifies tension of particular muscles and angle of specific joints at specific times necessary to carry out the programs and subprograms transmitted from the middle control levels.
- B. Structures: brainstem or spinal cord interneurons, afferent neurons, motor neurons.

Voluntary and Involuntary Actions

Given such a highly interconnected and complicated neuroanatomical basis for the motor system, it is difficult to use the phrase **voluntary movement** with any real precision. We will use it, however, to refer to actions that have the following characteristics: (1) The movement is accompanied by a conscious awareness of what we are doing and why we are doing it, and (2) our attention is directed toward the action or its purpose.

The term *involuntary,* on the other hand, describes actions that do not have these characteristics. *Unconscious, automatic,* and *reflex* often serve as synonyms for *involuntary,* although in the motor system, the term *reflex* has a more precise meaning.

Despite our attempts to distinguish between voluntary and involuntary actions, almost all motor behavior involves both components, and it is not easy to make a distinction between the two. For example, some highly conscious acts with a repetitive nature, such as walking, are initiated by preprogrammed pattern-generating circuits in the brain and spinal cord. The alternating pattern of contraction of muscles activated by those circuits is then subconsciously varied in response to unique situations, as might occur when you encounter obstacles or uneven terrain while walking.

Most motor behavior, therefore, is neither purely voluntary nor purely involuntary but has elements of both. Moreover, actions shift along this continuum according to the frequency with which they are performed. When a person first learns to drive a car with a manual transmission, for example, shifting gears requires a great deal of conscious attention. With practice, those same actions become automatic. On the other hand, reflex behaviors that are generally involuntary can, with special effort, sometimes be voluntarily modified or even prevented.

We now turn to an analysis of the individual components of the motor control system. We will begin with local control mechanisms because their activity serves as a base upon which the descending pathways exert their influence. Keep in mind throughout these descriptions that motor neurons always form the final common pathway to the muscles.

10.2 Local Control of Motor Neurons

The local control systems are the relay points for instructions to the motor neurons from centers higher in the motor control hierarchy. In addition, the local control systems are very important in adjusting motor unit activity to unexpected obstacles to movement and to painful stimuli in the surrounding environment.

To carry out these adjustments, the local control systems use information carried by afferent fibers from sensory receptors in the muscles, tendons, joints, and skin of the body parts to be moved. As noted earlier, the afferent fibers also transmit information to higher levels of the hierarchy.

Interneurons

Most of the synaptic input to motor neurons from the descending pathways and afferent neurons does not go directly to motor neurons but, rather, goes to interneurons that synapse with the motor neurons. Interneurons comprise 90% of spinal cord neurons, and they are of several types. Some are near the motor neuron they synapse upon and thus are called local interneurons. Others have processes that extend up or down short distances in the spinal cord and brainstem, or even throughout much of the length of the central nervous system. The interneurons with longer processes are important for integrating complex movements such as stepping forward with your left foot as you throw a baseball with your right arm.

The interneurons are important elements of the local level of the motor control hierarchy, integrating inputs not only from higher centers and peripheral receptors but from other interneurons as well (**Figure 10.3**). They are crucial in determining which muscles are activated and when. This is especially important in coordinating repetitive, rhythmic activities like walking or running, for which spinal cord interneurons encode pattern generator circuits responsible for activating and inhibiting limb movements in an alternating sequence. Moreover, interneurons can act as "switches" that enable a movement to be turned on or off under the command of higher motor centers. For example, if you pick up a hot plate, a local reflex arc will be initiated by pain receptors in the skin of your hands, normally causing you to drop the plate. If it contains your dinner, however, descending commands can inhibit the local activity and you can hold onto the plate until you reach a location where you can put it down safely. The integration of various inputs by local interneurons is a prime example of the general principle of physiology that most physiological functions are controlled by multiple regulatory systems, often working in opposition.

Local Afferent Input

As just noted, afferent fibers sometimes impinge on the local interneurons. (In one case that will be discussed shortly, they synapse directly on motor neurons.) The afferent fibers carry information from sensory receptors located in three places: (1) in the skeletal muscles controlled by the motor neurons; (2) in other muscles, such as those with antagonistic actions; and (3) in the tendons, joints, and skin of body parts affected by the action of the muscle.

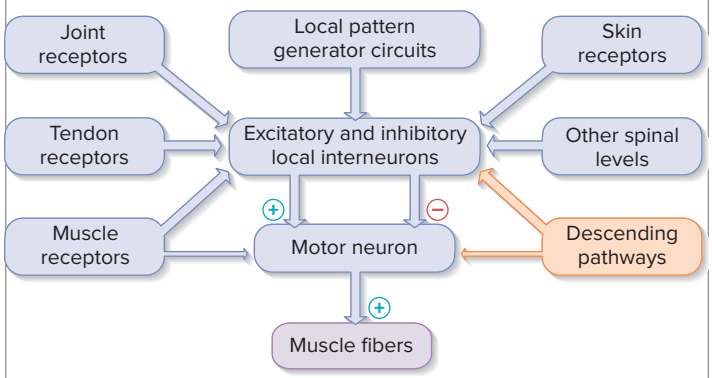


Figure 10.3 Converging inputs to local interneurons that control motor neuron activity. Plus signs indicate excitatory synapses and minus sign an inhibitory synapse. Neurons in addition to those shown may synapse directly onto motor neurons.

PHYSIOLOGICAL INQUIRY

- Many spinal cord interneurons release the neurotransmitter glycine, which opens chloride ion channels on postsynaptic cell membranes. Given that the plant-derived chemical strychnine blocks glycine receptors, predict the symptoms of strychnine poisoning.

Answer can be found at end of chapter.

These receptors monitor the length and tension of the muscles, movement of the joints, and the effect of movements on the overlying skin. In other words, the movements themselves give rise to afferent input that, in turn, influences how the movement proceeds. As we will see next, their input sometimes provides negative feedback control over the muscles and also contributes to the conscious awareness of limb and body position.

Length-Monitoring Systems Stretch receptors embedded within muscles monitor muscle length and the rate of change in muscle length. These receptors consist of peripheral endings of afferent nerve fibers wrapped around modified muscle fibers, several of which are enclosed in a connective-tissue capsule. The entire apparatus is collectively called a **muscle spindle** (Figure 10.4). The modified muscle fibers within the spindle are known as **intrafusal fibers.** The skeletal muscle fibers that form the bulk of the muscle and generate its force and movement (which were the focus of Chapter 9) are the **extrafusal fibers.**

Within a given spindle are two kinds of stretch receptors. One, the nuclear chain fiber, responds best to how much a muscle is stretched; whereas the other, the nuclear bag fiber, responds to both the magnitude of a stretch and the speed with which it occurs. Although the two kinds of stretch receptors are separate entities, we will refer to them collectively as the **muscle-spindle stretch receptors.**

The muscle spindles are attached by connective tissue in parallel to the extrafusal fibers. Thus, an external force stretching the muscle also pulls on the intrafusal fibers, stretching them and activating their receptor endings (Figure 10.5a). The more or the faster the muscle is stretched, the greater the rate of receptor firing.

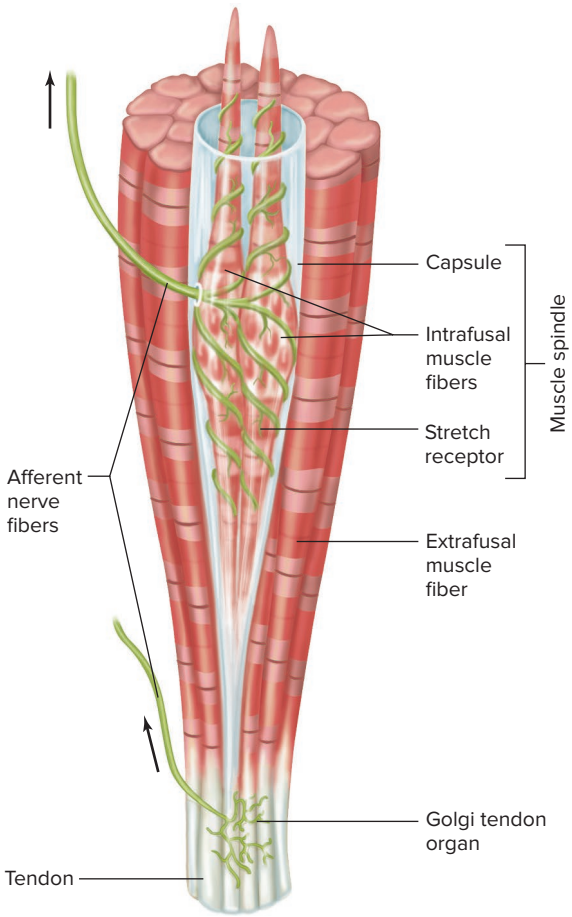


Figure 10.4 A muscle spindle and Golgi tendon organ. The muscle spindle is exaggerated in size compared to the extrafusal muscle fibers. The Golgi tendon organ will be discussed later in the chapter.

Extrafusal fibers of a muscle are activated by large-diameter motor neurons called **alpha motor neurons.** If action potentials along alpha motor neurons cause contraction of the extrafusal fibers, the resultant shortening of the muscle removes tension on the spindle and slows the rate of firing in the stretch receptor (Figure 10.5b). If muscles were always activated as shown in Figure 10.5b, however, slackening of muscle spindles would reduce the available sensory information about muscle length during rapid shortening contractions. A mechanism called **alpha–gamma coactivation** prevents this loss of information. The two ends of intrafusal muscle fibers are activated by smaller-diameter neurons called **gamma motor neurons (Figure 10.5c).** The cell bodies of alpha and gamma motor neurons to a given muscle lie close together in the spinal cord or brainstem. Both types are activated by interneurons in their immediate vicinity and sometimes directly by neurons of the descending pathways. The contractile ends of intrafusal fibers are not large or strong enough to contribute to force or shortening of the whole muscle. However, they can maintain tension and stretch in the central receptor region of the intrafusal fibers. Activating gamma motor neurons alone therefore increases the sensitivity of a muscle to stretch. Coactivating gamma motor neurons and alpha motor neurons prevents the central region of the muscle spindle from going slack during a shortening contraction (see Figure 10.5c). This ensures that information about muscle length will be continuously available to provide for adjustment during ongoing actions and to plan and program future movements.

The Stretch Reflex When the afferent fibers from the muscle spindle enter the central nervous system, they divide into branches that take different paths. In Figure 10.6, path A makes excitatory synapses directly onto motor neurons that return to the muscle that was stretched, thereby completing a reflex arc known as the **stretch reflex.**

This reflex is important in maintaining balance and posture, and is probably most familiar in the form of the **knee-jerk reflex,** part of a routine medical examination. The examiner taps the patellar tendon (see Figure 10.6), which passes over the knee and connects extensor muscles in the thigh to the tibia in the lower leg. As the tendon is pushed in by tapping, the thigh muscles it is attached to are stretched and all the stretch receptors within these muscles are activated. This stimulates a burst of action potentials in the afferent nerve fibers from the stretch receptors, and these action potentials activate excitatory synapses on the motor neurons that control these same muscles. The motor units are stimulated, the thigh muscles contract, and the patient's lower leg briefly extends. The proper performance of the knee jerk tells the physician that the afferent fibers, the balance of synaptic input to the motor neurons, the motor neurons, the neuromuscular junctions, and the muscles themselves are functioning normally.

Because the afferent nerve fibers in the stretched muscle synapse directly on the motor neurons to that muscle without any interneurons, this type of reflex is called a **monosynaptic reflex.** Stretch reflexes have the only known monosynaptic reflex arcs. All other reflex arcs are **polysynaptic;** they have at least one interneuron—and usually many—between the afferent and efferent neurons.

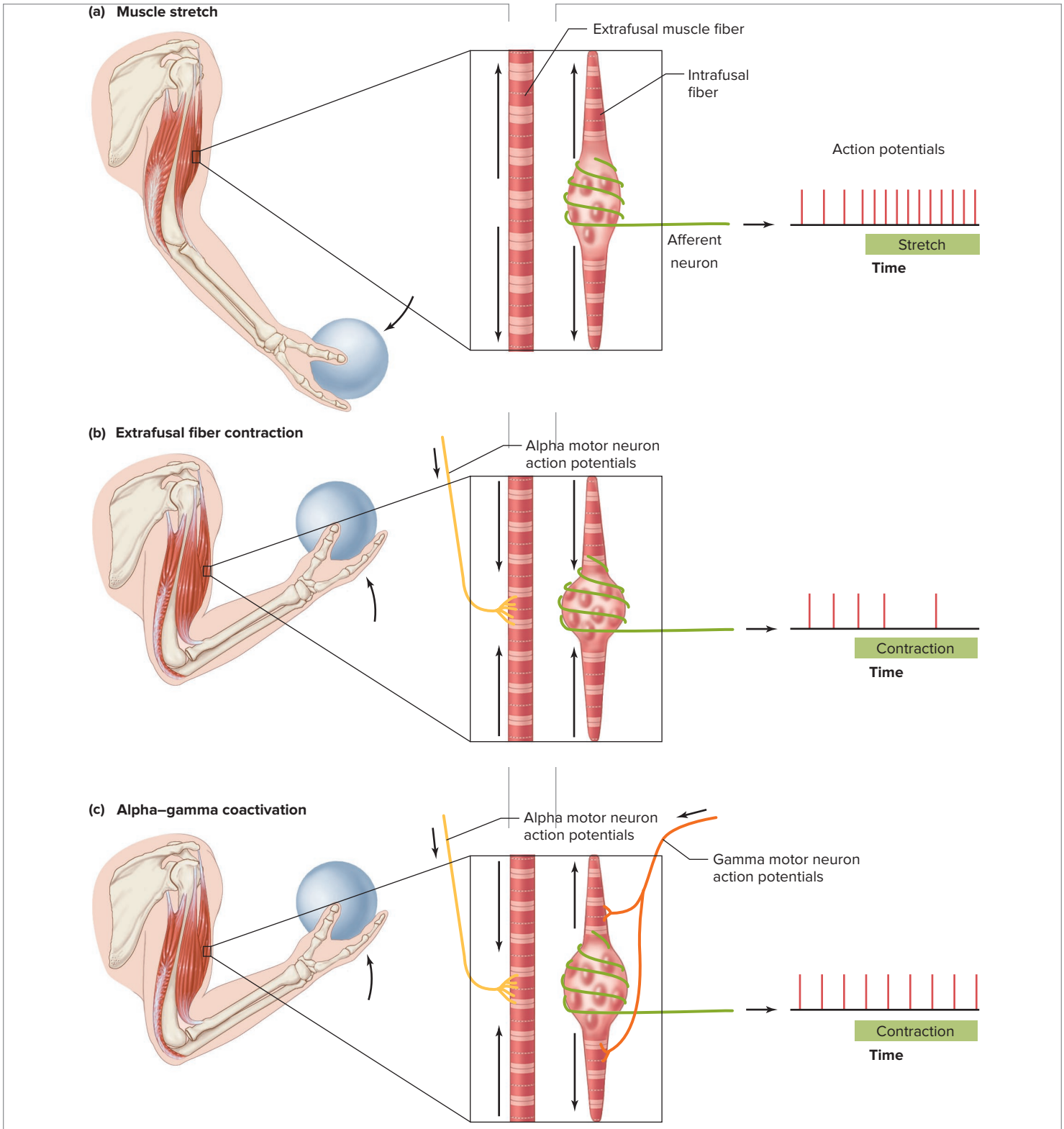
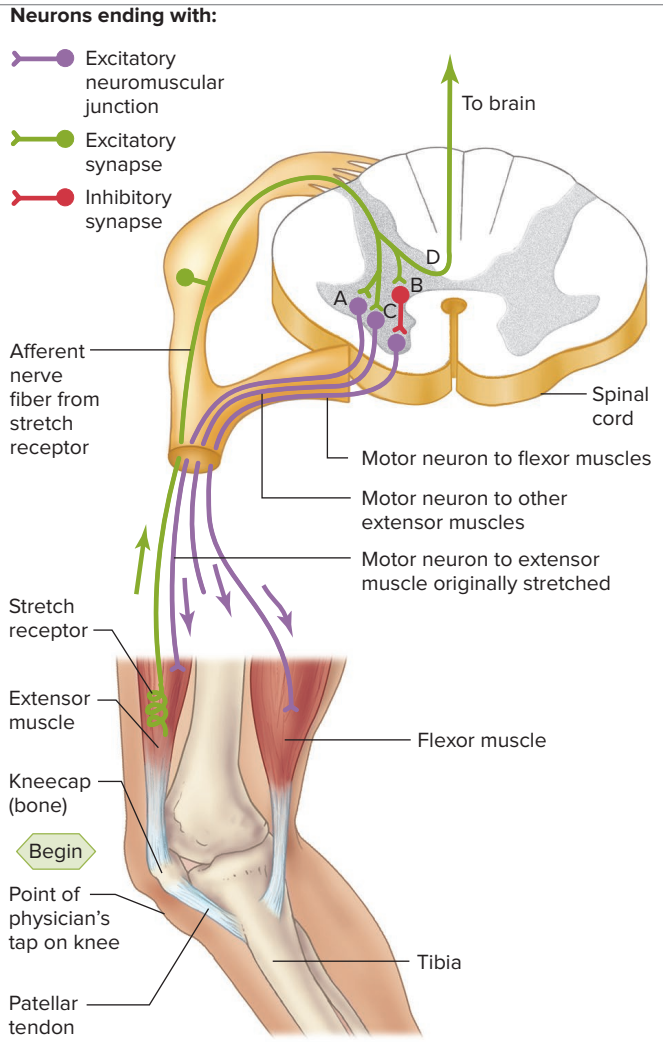


Figure 10.5 Alpha-gamma coactivation of muscle cells maintains muscle spindle sensitivity to muscle length. (a) Passive stretch of the muscle by an external load activates the spindle stretch receptors and causes an increased rate of action potentials in the afferent nerve. (b) Contraction of the extrafusal fibers removes tension on the stretch receptors and decreases the rate of action potential firing. (c) Simultaneous activation of alpha and gamma motor neurons results in maintained stretch of the central region of intrafusal fibers, and afferent information about muscle length continues to reach the central nervous system.

In path B of Figure 10.6, the branches of the afferent nerve fibers from stretch receptors end on inhibitory interneurons. When activated, these inhibit the motor neurons controlling antagonistic muscles whose contraction would interfere with the reflex response. In the knee jerk, for example, neurons to muscles that flex the knee are inhibited. This component of the stretch reflex is polysynaptic.



AP|R **Figure 10.6** Neural pathways involved in the knee-jerk reflex. Tapping the patellar tendon stretches the extensor muscle, causing (paths A and C) compensatory contraction of this and other extensor muscles, (path B) relaxation of flexor muscles, and (path D) information about muscle length to go to the brain. Arrows indicate direction of action potential propagation.

PHYSIOLOGICAL INQUIRY

- Based on this figure and Figure 10.5, hypothesize what might happen if you could suddenly stimulate gamma motor neurons to leg flexor muscles in a resting subject.

Answer can be found at end of chapter.

The divergence of neuronal pathways to influence both the agonist and antagonist muscles of a particular body movement is called **reciprocal innervation.** This is characteristic of many movements, not just the stretch reflex, and in some circumstances antagonist muscle groups are simultaneously contracted to stiffen a limb joint.

Path C in Figure 10.6 activates motor neurons of **synergistic muscles**—that is, muscles whose contraction assists the intended motion. In the example of the knee-jerk reflex, this would include other muscles that extend the leg.

Path D of Figure 10.6 is not explicitly part of the stretch reflex; it demonstrates that information about changes in muscle length ascends to higher centers. The axon of the afferent neuron continues to the brainstem and synapses there with interneurons that form the next link in the pathway that conveys information about the muscle length to areas of the brain dealing with motor control. This information is especially important during slow, controlled movements such as the performance of an unfamiliar action. Ascending paths also provide information that contributes to the conscious perception of the position of a limb.

Tension-Monitoring Systems Any given set of inputs to a given set of motor neurons can lead to various degrees of tension in the muscles they innervate. The tension depends on muscle length, the load on the muscles, and the degree of muscle fatigue. Therefore, feedback is necessary to inform the motor control systems of the tension actually achieved.

Some of this feedback is provided by vision (you can see whether you are lifting or lowering an object) as well as by afferent input from skin, muscle, and joint receptors. An additional receptor type specifically monitors the stretching of muscle tendons, which is related to how much tension the contracting motor units are exerting and external forces acting on the muscle.

The receptors employed in this tension-monitoring system are the **Golgi tendon organs,** which are endings of afferent nerve fibers that wrap around collagen bundles in the tendons near their junction with the muscle (see Figure 10.4). These collagen bundles are slightly bowed in the resting state. When the muscle is stretched or the attached extrafusal muscle fibers contract, tension is exerted on the tendon. This tension straightens the collagen bundles and distorts the receptor endings, activating them. The tendon is typically stretched much more by an active contraction of the muscle than when the whole muscle is passively stretched (**Figure 10.7**). When activated, the Golgi tendon organs initiate action potentials that are transmitted to the central nervous system.

Branches of afferent neurons from Golgi tendon organs ascend to the brain to provide conscious perception of muscle force, and that information can be used to modify an ongoing motor program. Branches also project widely to interneurons in the spinal cord, where they contribute to reflexive control of muscles. The muscles affected can include not only the one associated with a given tendon organ, but also muscles that move other joints of a limb. Combining muscle tension information from Golgi tendon organs with muscle length information from the muscle spindles allows reflexive coordination of limb flexion, extension, and stiffness during walking and running.

The Withdrawal Reflex In addition to the afferent information from the spindle stretch receptors and Golgi tendon organs of activated muscles, other input is transmitted to the local motor control systems. For example, painful stimulation of the skin, as occurs from stepping on a tack, activates the flexor muscles and inhibits the extensor muscles of the ipsilateral leg (on the same side of the body). The resulting action moves the affected limb away from the harmful stimulus and is thus known as a **withdrawal reflex** (**Figure 10.8**). The same stimulus causes just the opposite response in the contralateral leg (on the opposite side of the body from the stimulus); motor neurons to the extensors are activated while the flexor muscle motor neurons are inhibited. This **crossed-extensor reflex** enables the contralateral leg to

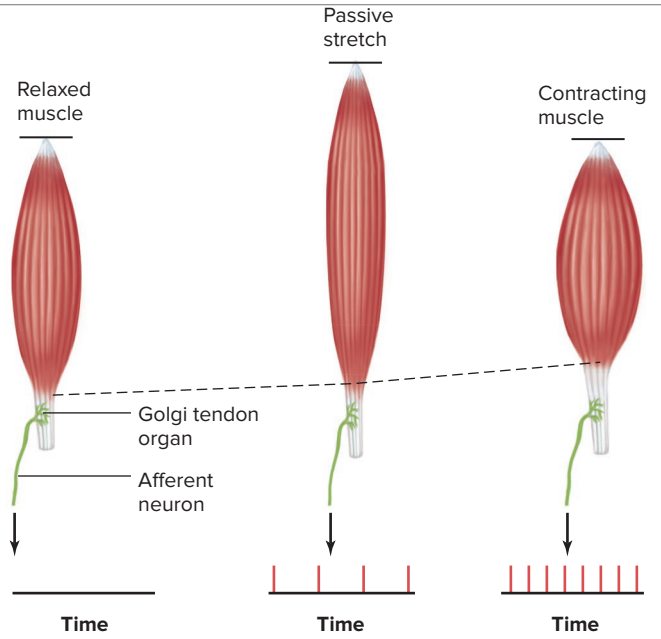


Figure 10.7 Activation of Golgi tendon organs. Compared to when a muscle is contracting, passive stretch of the relaxed muscle produces less stretch of the tendon and fewer action potentials from the Golgi tendon organ.

PHYSIOLOGICAL INQUIRY

- Which of these conditions would result in the greatest action potential frequency in afferent neurons from muscle-spindle receptors?

Answer can be found at end of chapter.

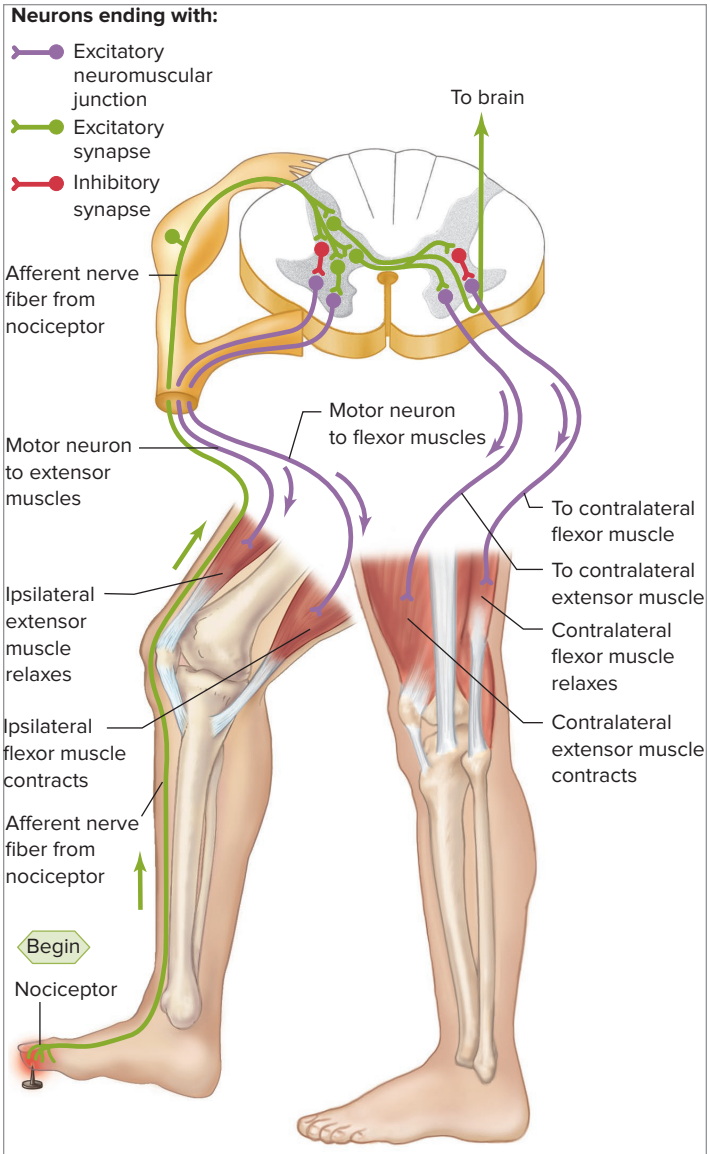
support the body's weight as the injured foot is lifted by flexion (see Figure 10.8). This concludes our discussion of the local level of motor control.

10.3 The Brain Motor Centers and the Descending Pathways They Control

We now turn our attention to the motor centers in the brain and the descending pathways that direct the local control system (review Figure 10.1).

Cerebral Cortex

A network of connected neurons in the frontal and parietal lobes of the cerebral cortex has a critical function in both the planning and ongoing control of voluntary movements, functioning in both the highest and middle levels of the motor control hierarchy. A large number of neurons that give rise to descending pathways for motor control come from two areas of sensorimotor cortex on the posterior part of the frontal lobe: the **primary motor cortex** (sometimes called simply the **motor cortex**) and the **premotor area** (Figure 10.9).



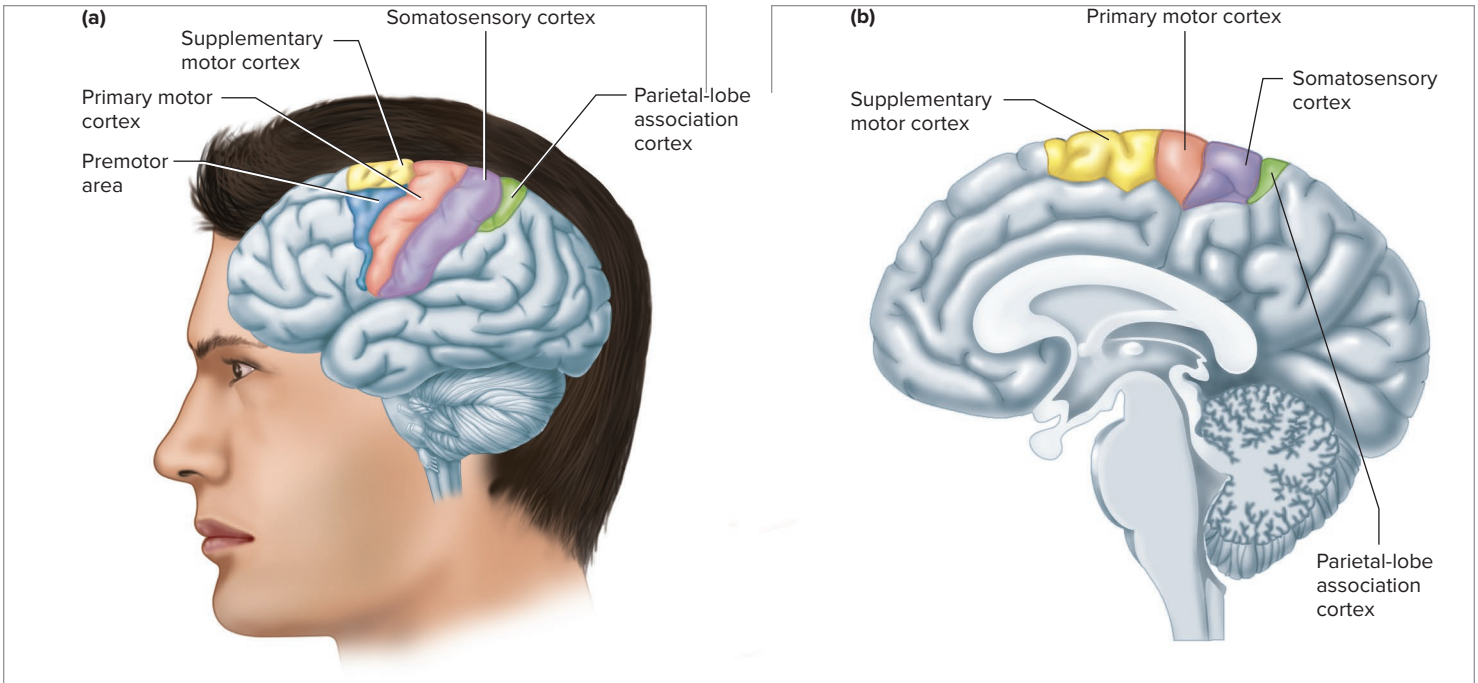
AP|R **Figure 10.8** In response to pain detected by nociceptors (Chapter 7), the ipsilateral flexor muscle's motor neuron is stimulated (withdrawal reflex). In the case illustrated, the opposite limb is extended (crossed-extensor reflex) to support the body's weight. Arrows indicate direction of action potential propagation.

PHYSIOLOGICAL INQUIRY

- While crawling across a floor, a child accidentally places her right hand onto a piece of broken glass. How will the flexor muscles of her left arm respond?

Answer can be found at end of chapter.

Other areas of sensorimotor cortex shown in Figure 10.9 include the **supplementary motor cortex,** which lies mostly on the surface of the frontal lobe where the cortex folds down between the two hemispheres, the **somatosensory cortex,** and parts of the **parietal-lobe association cortex.** The neurons of the motor cortex that control muscle groups in various parts of the body are arranged anatomically into a **somatotopic map**



AP|R **Figure 10.9** (a) The major motor areas of the cerebral cortex. (b) Midline view of the right side of the brain showing the supplementary motor cortex, which lies in the part of the cerebral cortex that is folded down between the two cerebral hemispheres. Other cortical motor areas also extend onto this area. The premotor, supplementary motor, primary motor, somatosensory, and parietal-lobe association cortices together make up the sensorimotor cortex.

(**Figure 10.10**), similar to that seen in the somatosensory cortex (review Figure 7.21).

Although these areas of the cortex are anatomically and functionally distinct, they are heavily interconnected, and individual muscles or movements are represented at multiple sites. Thus, the cortical neurons that control movement form a neural network, meaning that many neurons participate in each individual movement. In addition, any one neuron may function in more than one movement. The neural networks can be distributed across multiple sites in parietal and frontal cortex, including the sites named in the preceding two paragraphs. The interactions of the neurons within the networks are flexible so that the neurons are capable of responding differently under different circumstances. This adaptability enhances the possibility of integrating incoming neural signals from diverse sources and the final coordination of many parts into a smooth, purposeful movement. It probably also accounts for the remarkable variety of ways in which we can approach a goal. For example, you can comb your hair with the right hand or the left, starting at the back of your head or the front. This same adaptability also accounts for some of the learning that occurs in all aspects of motor behavior.

We have described the various areas of sensorimotor cortex as giving rise, either directly or indirectly, to pathways descending to the motor neurons. However, additional brain areas are involved in the initiation of intentional movements, such as the basal nuclei, cerebellum, and areas involved in memory, emotion, and motivation.

Association areas of the cerebral cortex also have other functions in motor control. For example, neurons of the parietal-lobe association cortex are important in the visual control of reaching and grasping. These neurons contribute to matching motor signals concerning the pattern of hand action with signals from the visual system concerning the three-dimensional features of the objects to be grasped. Imagine a glass of water sitting in front of you on your desk—you could reach out and pick it up much more smoothly with your eyes tracking your arm and hand movements than you could with your eyes closed.

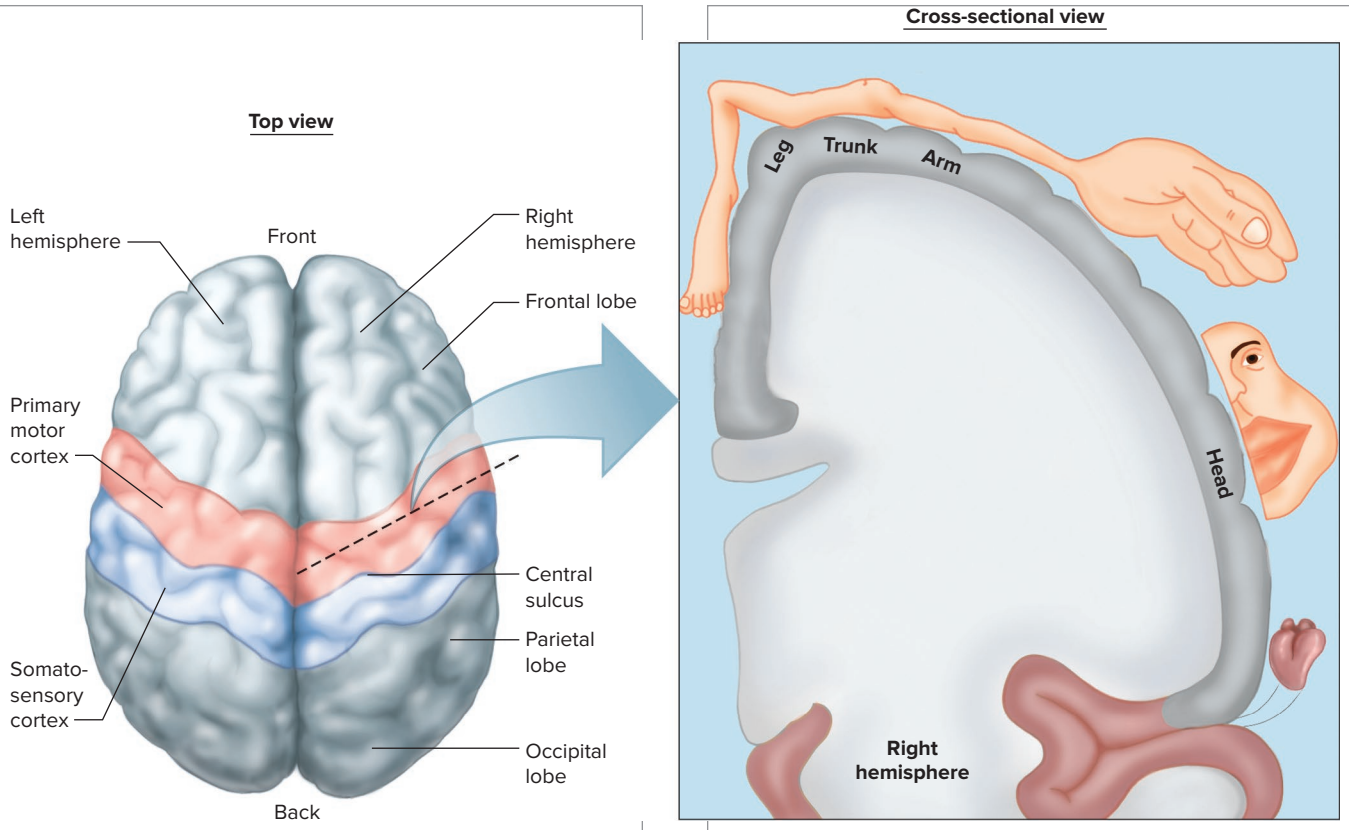
During activation of the cortical areas involved in motor control, subcortical mechanisms also become active. We now turn to these areas of the motor control system.

Subcortical and Brainstem Nuclei

Numerous highly interconnected structures lie in the brainstem and within the cerebrum beneath the cortex, where they interact with the cortex to control movements. Their influence is transmitted indirectly to the motor neurons both by pathways that ascend to the cerebral cortex and by pathways that descend from some of the brainstem nuclei.

These structures may play a minor role in motivation and initiating movements, but they definitely are very important in planning and monitoring them. Their role is to establish the programs that determine the specific sequence of movements needed to accomplish a desired action. Subcortical and brainstem nuclei are also important in learning skilled movements.

Prominent among the subcortical nuclei are the paired **basal nuclei** (see Figure 10.2b), which consist of a closely related group of separate nuclei. As described in Chapter 6, these structures are often referred to as basal ganglia, but their presence within the central nervous system makes the term *nuclei* more anatomically correct. They form a link in some of the looping parallel circuits through which activity in the motor system is transmitted from a specific region of sensorimotor cortex to the basal nuclei, from there



AP|R **Figure 10.10** Somatotopic map of major body areas in the primary motor cortex. Within the broad areas, no one area exclusively controls the movement of a single body region and there is much overlap and duplication of cortical representation. Relative sizes of body structures are proportional to the number of neurons dedicated to their motor control. Only the right motor cortex, which principally controls muscles on the left side of the body, is shown.

PHYSIOLOGICAL INQUIRY

- What structural features of the primary motor cortex somatotopic map reflect the general principle of physiology that structure is a determinant of—and has coevolved with—function?

Answer can be found at end of chapter.

to the thalamus, and then back to the cortical area where the circuit started (review Figure 10.1). Some of these circuits facilitate movements, and others suppress them. This explains why brain damage to subcortical nuclei following a stroke or trauma can result in either hypercontracted muscles or flaccid paralysis—it depends on which specific circuits are damaged. The importance of the basal nuclei is particularly apparent in certain disease states, as we discuss next.

Parkinson's Disease In ***Parkinson's disease,*** the input to the basal nuclei is diminished, the interplay of the facilitatory and inhibitory circuits is unbalanced, and activation of the motor cortex (via the basal nuclei–thalamus limb of the circuit just mentioned) is reduced. Clinically, Parkinson's disease is characterized by a reduced amount of movement (***akinesia***), slow movements (***bradykinesia***), muscular rigidity, and a tremor at rest. Other motor and nonmotor abnormalities may also be present. For example, a common set of symptoms includes a change in facial expression resulting in a masklike, unemotional appearance, a shuffling gait with loss of arm swing, and a stooped and unstable posture.

Although the symptoms of Parkinson's disease reflect inadequate functioning of the basal nuclei, a major part of the initial defect arises in neurons of the **substantia nigra** ("black substance"), a brainstem nucleus that gets its name from the dark pigment in its cells. These neurons normally project to the basal nuclei, where they release dopamine from their axon terminals. The substantia nigra neurons degenerate in Parkinson's disease and the amount of dopamine they deliver to the basal nuclei is decreased. This decreases the subsequent activation of the sensorimotor cortex.

It is not currently known what causes the degeneration of neurons of the substantia nigra and the development of Parkinson's disease. In a small fraction of cases, there is evidence that it may have a genetic cause, based on observed changes in the function of genes associated with mitochondrial function, protection from oxidative stress, and removal of cellular proteins that have been targeted for metabolic breakdown. Scientists suspect that exposure to environmental toxins such as manganese, carbon monoxide, and some pesticides may also be a contributing factor to developing the disease. One chemical clearly linked to destruction of the substantia nigra is ***MPTP*** (***1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine***). MPTP is an impurity sometimes created in the manufacture of a synthetic heroin-like opioid drug, which when injected leads to a Parkinson's-like syndrome.

The drugs used to treat Parkinson's disease are all designed to restore dopamine activity in the basal nuclei. They fall into three main categories: (1) agonists (stimulators) of dopamine receptors, (2) inhibitors of the enzymes that metabolize dopamine at synapses, and (3) precursors of dopamine itself. The most widely prescribed drug is ***Levodopa*** (***L-dopa***), which falls into the third category. L-dopa enters the bloodstream, crosses the blood–brain barrier, and is converted in neurons to dopamine. (Dopamine itself is not used as medication because it cannot cross the blood–brain barrier and it has too many systemic side effects.) The newly formed dopamine activates receptors in the basal nuclei and improves the symptoms of the disease. Side effects sometimes occurring with L-dopa include hallucinations, like those seen in individuals with schizophrenia who have excessive dopamine activity (see Chapter 8), and spontaneous, abnormal motor activity. Other therapies for Parkinson's disease include the lesioning (destruction) of overactive areas of the basal nuclei and ***deep brain stimulation.*** The latter is accomplished by surgically implanting electrodes in regions of the basal nuclei; the electrodes are connected to an electrical pulse generator similar to a cardiac artificial pacemaker (Chapter 12). Although in many cases it relieves symptoms, the mechanism is not understood. Injection of undifferentiated stem cells capable of producing dopamine is also being explored as a possible treatment.

Cerebellum

The cerebellum is located dorsally to the brainstem (see Figure 10.2a and refer back to Chapter 6). It influences posture and movement indirectly by means of input to brainstem nuclei and (by way of the thalamus) to regions of the sensorimotor cortex that give rise to pathways that descend to the motor neurons. The cerebellum receives information from the sensorimotor cortex and also from the vestibular system, eyes, skin, muscles, joints, and tendons—that is, from some of the very receptors that movement affects.

One role of the cerebellum in motor functioning is to provide timing signals to the cerebral cortex and spinal cord for precise execution of the different phases of a motor program, in particular, the timing of the agonist/antagonist components of a movement. It also helps coordinate movements that involve several joints and stores the memories of these movements so they are easily achieved the next time they are tried.

The cerebellum also participates in planning movements—integrating information about the nature of an intended movement with information about the surrounding space. The cerebellum then provides this as a feedforward (see Chapter 1) signal to the brain areas responsible for refining the motor program. Moreover, during the course of the movement, the cerebellum compares information about what the muscles *should* be doing with information about what they actually *are* doing. If a discrepancy develops between the intended movement and the actual one, the cerebellum sends an error signal to the motor cortex and subcortical centers to correct the ongoing program.

The importance of the cerebellum in programming movements can best be appreciated when observing its absence in individuals with ***cerebellar disease.*** They typically cannot perform limb or eye movements smoothly but move with a tremor—a so-called ***intention tremor*** that increases as a movement nears its final destination. This differs from patients with Parkinson's disease, who have a tremor while at rest. People with cerebellar disease also cannot combine the movements of several joints into a single, smooth, coordinated motion. The role of the cerebellum in the precision and timing of movements can be appreciated when you consider the complex tasks it helps us accomplish. For example, a tennis player sees a ball fly over the net, anticipates its flight path, runs along an intersecting path, and swings the racquet through an arc that will intercept the ball with the speed and force required to return it to the other side of the court. People with cerebellar damage cannot achieve this level of coordinated, precise, learned movement.

Unstable posture and awkward gait are two other symptoms characteristic of cerebellar disease. For example, people with cerebellar damage walk with their feet wide apart, and they have such difficulty maintaining balance that their gait is similar to that seen in people who are intoxicated by ethanol. Visual input helps compensate for some of the loss of motor coordination—patients can stand on one foot with eyes open but not closed. A final symptom involves difficulty in learning new motor skills. Individuals with cerebellar disease find it hard to modify movements in response to new situations. Unlike damage to areas of sensorimotor cortex, cerebellar damage is not usually associated with paralysis or weakness.

Descending Pathways

The influence exerted by the various brain regions on posture and movement occurs via descending pathways to the motor neurons and the interneurons that affect them. The pathways are of two types: the **corticospinal pathways,** which, as their name implies, originate in the cerebral cortex; and a second group we will refer to as the **brainstem pathways,** which originate in the brainstem.

Neurons from both types of descending pathways end at synapses on alpha and gamma motor neurons or on interneurons that affect them. Sometimes these are the same interneurons that function in local reflex arcs, thereby ensuring that the descending signals are fully integrated with local information before the activity of the motor neurons is altered. In other cases, the interneurons are part of neural networks involved in posture or locomotion. The ultimate effect of the descending pathways on the alpha motor neurons may be excitatory or inhibitory.

Importantly, some of the descending fibers affect *afferent* systems. They do this via (1) presynaptic synapses on the terminals of afferent neurons as these fibers enter the central nervous system, or (2) synapses on interneurons in the ascending pathways. The overall effect of this descending input to afferent systems is to regulate their influence on either the local or brain motor control areas, thereby altering the importance of a particular bit of afferent information or sharpening its focus. For example, when performing an exceptionally delicate or complicated task, like a doctor performing surgery, descending inputs might facilitate signaling in afferent pathways carrying proprioceptive inputs monitoring hand and finger movements. This descending (motor) control over ascending (sensory) information provides another example to show that there is no real functional separation between the motor and sensory systems.

Corticospinal Pathway The nerve fibers of the corticospinal pathways have their cell bodies in the sensorimotor cortex and terminate in the spinal cord. The corticospinal pathways are also called the **pyramidal tracts** or **pyramidal system** because of their triangular shape as they pass along the ventral surface of the medulla oblongata. In the medulla oblongata near the junction of the spinal

cord and brainstem, most of the corticospinal fibers cross (known as decussation) to descend on the opposite side (**Figure 10.11**). The skeletal muscles on the left side of the body are therefore controlled largely by neurons in the right half of the brain, and vice versa.

As the corticospinal fibers descend through the brain from the cerebral cortex, they are accompanied by fibers of the **corticobulbar pathway** (*bulbar* means "pertaining to the brainstem"), a pathway that begins in the sensorimotor cortex and ends in the brainstem. The corticobulbar fibers control, directly or indirectly via interneurons, the motor neurons that innervate muscles of the eye, face, tongue, and throat. These fibers provide the main source of control for voluntary movement of the muscles of the head and neck, whereas the corticospinal fibers provide control of voluntary movements of the distal extremeties. For convenience, we will include the corticobulbar pathway in the general term *corticospinal pathways.*

Convergence and divergence are hallmarks of the corticospinal pathway. For example, a great number of different neuronal sources converge on neurons of the sensorimotor cortex, which is not surprising when you consider the many factors that can affect motor behavior. As for the descending pathways, neurons from wide areas of the sensorimotor cortex converge onto single motor neurons at the local level so that multiple brain areas usually control single muscles. Also, axons of single corticospinal neurons diverge markedly to synapse with a number of different motor neuron populations at various levels of the spinal cord, thereby ensuring that the motor cortex can coordinate many different components of a movement.

This apparent "blurriness" of control is surprising when you think of the delicacy with which you can move a fingertip, because the corticospinal pathways control rapid, fine movements of the distal extremities, such as those you make when you manipulate an object with your fingers. After damage occurs to the corticospinal pathways, movements are slower and weaker, individual finger movements are absent, and it is difficult to release a grip.

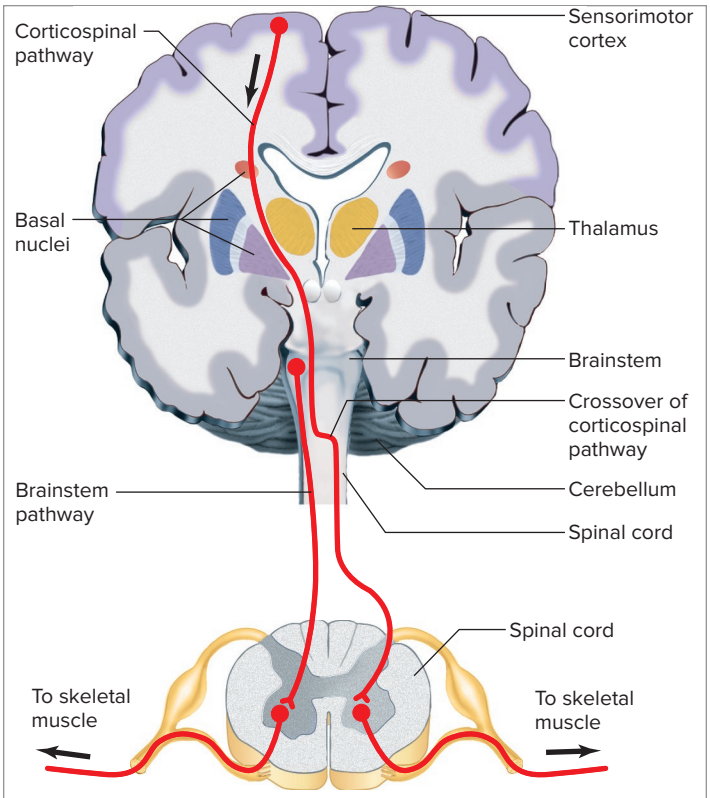
Brainstem Pathways Axons from neurons in the brainstem also form pathways that descend into the spinal cord to influence motor neurons. These pathways are sometimes referred to as the **extrapyramidal system,** or indirect pathways, to distinguish them from the corticospinal (pyramidal) pathways. However, no general term is widely accepted for these pathways; for convenience, we will refer to them collectively as the brainstem pathways.

Axons of most of the brainstem pathways remain uncrossed and affect muscles on the same side of the body (see Figure 10.11), although a few do cross over to influence contralateral muscles. In the spinal cord, the fibers of the brainstem pathways descend as distinct clusters, named according to their sites of origin. For example, the vestibulospinal pathway descends to the spinal cord from the vestibular nuclei in the brainstem, whereas the reticulospinal pathway descends from neurons in the brainstem reticular formation.

As stated previously, the corticospinal neurons generally have their greatest influence over motor neurons that control muscles involved in fine, isolated movements, particularly those of the fingers and hands. The brainstem descending pathways, in contrast, are involved more with coordination of the large muscle groups of the trunk and proximal portions of the limbs used in the maintenance of upright posture, in locomotion, and in head and body movements when turning toward a specific stimulus.

There is, however, much interaction between the descending pathways. For example, some fibers of the corticospinal pathway end on interneurons that have important functions in posture, whereas fibers of the brainstem descending pathways sometimes end directly on the alpha motor neurons to control discrete muscle movements. Because of this redundancy, one system may compensate for loss of function resulting from damage to the other system, although the compensation is generally not complete.

The distinctions between the corticospinal and brainstem descending pathways are not clear-cut. All movements, whether automatic or voluntary, require the continuous coordinated interaction of both types of pathways.



AP|R **Figure 10.11** The corticospinal and brainstem pathways. Most of the corticospinal fibers cross in the brainstem to descend in the opposite side of the spinal cord, but the brainstem pathways are mostly uncrossed. For simplicity, the descending neurons are shown synapsing directly onto motor neurons in the spinal cord, but they commonly synapse onto local interneurons.

PHYSIOLOGICAL INQUIRY

- If a blood clot blocked a cerebral blood vessel supplying a small region of the right cerebral cortex just in front of the central sulcus in the deep groove between the hemispheres, what symptoms might result? (*Hint:* See also Figure 10.10.)

Answer can be found at end of chapter.

10.4 Muscle Tone

Even when a skeletal muscle is relaxed, there is a slight and uniform resistance when it is stretched by an external force. This resistance is known as **muscle tone,** and it can be an important diagnostic tool for clinicians assessing a patient's neuromuscular function.

Intrinsic muscle tone in smooth muscle is due to a baseline level of Ca^{2+} in the cytosol that causes low-level activity of tension-generating cross-bridges. By contrast, muscle tone in skeletal muscles is due both to the passive elastic properties of the muscles and joints and to the degree of ongoing alpha motor neuron activity. When a person is very relaxed, the alpha motor neuron activity does not make a significant contribution to the resistance to stretch. As the person becomes increasingly alert, however, more activation of the alpha motor neurons occurs and muscle tone increases.

Abnormal Muscle Tone

Abnormally high muscle tone, called ***hypertonia,*** accompanies a number of diseases and is seen very clearly when a joint is moved passively at high speeds. The increased resistance is due to an increased level of alpha motor neuron activity, which keeps a muscle contracted despite the attempt to relax it. Hypertonia usually occurs with disorders of the descending pathways that normally inhibit the motor neurons.

Clinically, the descending pathways and neurons of the motor cortex are often referred to as the **upper motor neurons** (a confusing misnomer because they are not really motor neurons). Abnormalities due to their dysfunction are classified, therefore, as ***upper motor neuron disorders*****.** Thus, hypertonia usually indicates an upper motor neuron disorder. In this clinical classification, the alpha motor neurons—the true motor neurons—are termed **lower motor neurons.**

Spasticity is a form of hypertonia in which the muscles do not develop increased tone until they are stretched a bit; after a brief increase in tone, the contraction subsides for a short time. The period of "give" occurring after a time of resistance is called the ***clasp-knife phenomenon.*** (When an examiner bends the limb of a patient with this condition, it is like folding a pocketknife—at first, the spring resists the bending motion, but once bending begins, it closes easily.) Spasticity may be accompanied by increased responses of motor reflexes such as the knee jerk and by decreased coordination and strength of voluntary actions. ***Rigidity*** is a form of hypertonia in which the increased muscle contraction is continual and the resistance to passive stretch is constant (as occurs in the disease tetanus, which is described in detail in the Clinical Case Study at the end of this chapter). Two other forms of hypertonia that can occur suddenly in individual or multiple muscles may originate as problems either in muscle cells or neuronal pathways: Muscle ***spasms*** are brief, involuntary contractions that may or may not be painful, and muscle ***cramps*** are prolonged, involuntary, and painful contractions (see Chapter 9).

Hypotonia is a condition of abnormally low muscle tone accompanied by weakness, atrophy (a decrease in muscle bulk), and decreased or absent reflex responses. Dexterity and coordination are generally preserved unless profound weakness is present. Although hypotonia may develop after cerebellar disease, it more frequently accompanies disorders of the alpha motor neurons (lower motor neurons), neuromuscular junctions, or the muscles themselves. The term ***flaccid,*** which means "weak" or "soft," is often used to describe hypotonic muscles.

Amyotrophic Lateral Sclerosis ***Amyotrophic lateral sclerosis (ALS)*** is a lower motor neuron condition in which progressive degeneration of alpha motor neurons causes hypotonia and atrophy of skeletal muscles. It is often first detected as a weakness of limb and trunk muscles, but involvement of muscles used in respiration and swallowing is generally what makes the condition fatal. Typically diagnosed in middle age, its progression is usually rapid, with the average lifespan following diagnosis being 3–5 years. This was the case for a famous baseball player who suffered from ALS, and for whom the disease is also referred to as ***Lou Gehrig's disease***. The condition is more common in men than in women, and about 5600 new cases occur each year in the United States. In most cases the causes are not known, but may include viruses, neurotoxins, heavy metals, immune system abnormalities, or enzyme abnormalities. Approximately 5% to 10% of cases are inherited, with about half of them being caused by a defect in a gene coding for an enzyme that protects neurons from free radicals generated during oxidative stress (see Chapter 2). There is currently no cure for ALS; treatment consists of medications and respiratory, occupational, and physical therapies that provide relief from symptoms and maintain comfort and independence as long as possible.

10.5 Maintenance of Upright Posture and Balance

The skeleton supporting the body is a system of long bones and a many-jointed spine that cannot stand erect against the forces of gravity without the support provided through coordinated muscle activity. The muscles that maintain upright posture—that is, support the body's weight against gravity—are controlled by the brain and by reflex mechanisms "wired into" the neural networks of the brainstem and spinal cord. Many of the reflex pathways previously introduced (for example, the stretch and crossed-extensor reflexes) are active in posture control.

Added to the problem of maintaining upright posture is that of maintaining balance. A human being is a tall structure balanced on a relatively small base, with the center of gravity quite high, just above the pelvis. For stability, the center of gravity must be kept within the base of support the feet provide (**Figure 10.12**). Once the center of gravity has moved beyond this base, the body will fall unless one foot is shifted to broaden the base of support. Yet, people can operate under conditions of unstable equilibrium because complex interacting **postural reflexes** maintain their balance.

The afferent pathways of the postural reflexes come from three sources: the eyes, the vestibular apparatus, and the receptors involved in proprioception (joint, muscle, and touch receptors, for example). The efferent pathways are the alpha motor neurons to the skeletal muscles, and the integrating centers are neuron networks in the brainstem and spinal cord.

In addition to these integrating centers, there are centers in the brain that form an internal model of the body's geometry, its support conditions, and its orientation with respect to vertical. This internal representation serves two purposes: (1) It provides a reference framework for the perception of the body's position and orientation in space and for planning actions, and (2) it contributes to stability via the motor controls involved in maintaining upright posture.

There are many familiar examples of using reflexes to maintain upright posture; one is the crossed-extensor reflex. As one leg is flexed and lifted off the ground, the other is extended more strongly to support the weight of the body, and the positions of various parts of the body are shifted to move the center of

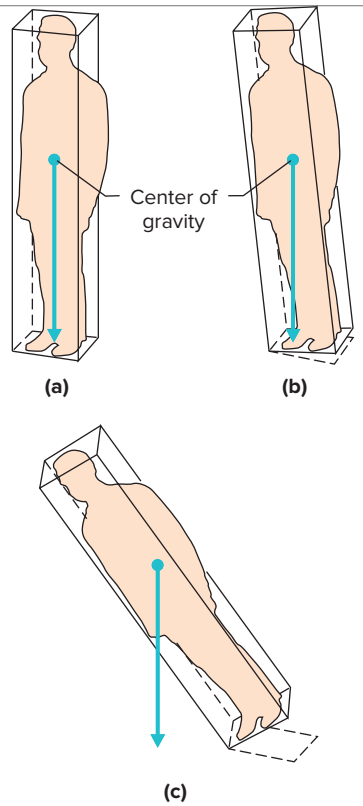


Figure 10.12 The center of gravity is the point in an object at which, if a string were attached and pulled up, all the downward force due to gravity would be exactly balanced. (a) The center of gravity must remain within the upward vertical projections of the object's base (the tall box outlined in the drawing) if stability is to be maintained. (b) Stable conditions. The box tilts a bit, but the center of gravity remains within the base area—the dashed rectangle on the floor—so the box returns to its upright position. (c) Unstable conditions. The box tilts so far that its center of gravity is not above any part of the object's base and the object will fall.

PHYSIOLOGICAL INQUIRY

- The effect of gravity on stable posture reflects the general principle of physiology that physiological processes are dictated by the laws of chemistry and physics. List other ways you can imagine in which gravity influences physiological functions, including but not limited to motor function.

Answer can be found at end of chapter.

gravity over the single, weight-bearing leg. This shift in the center of gravity, as **Figure 10.13** demonstrates, is an important component in the stepping mechanism of locomotion.

As previously described, afferent inputs from the eyes, vestibular apparatus, and somatic receptors of proprioception are integrated for optimal postural adjustments. However, the loss of vision or vestibular inputs alone does not cause a person to topple over. Blind people maintain their balance quite well with only a slight loss of precision, and people whose vestibular mechanisms have been destroyed can, with extensive rehabilitation, have very little disability in everyday life as long as their visual system and somatic receptors are functioning. On the other hand, loss of afferent proprioceptive inputs, as occurs in a condition

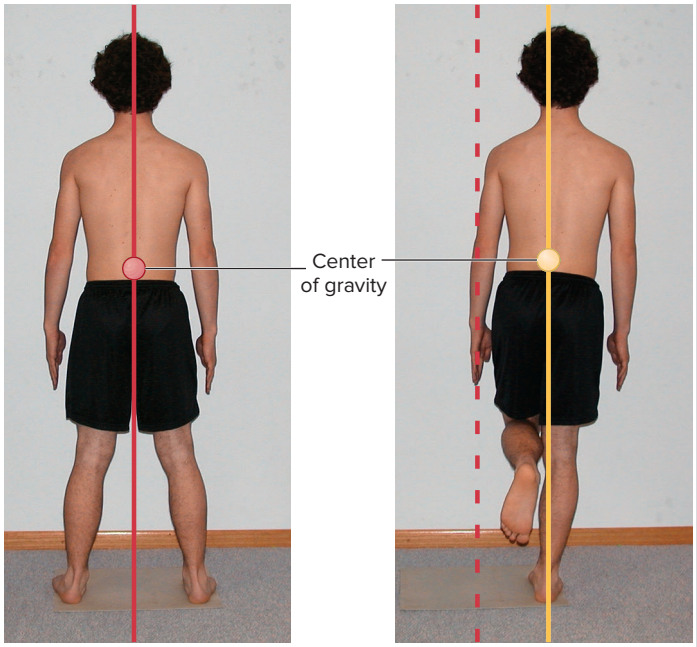


Figure 10.13 Postural changes with stepping. (a) Normal standing posture. The center of gravity falls directly between the two feet. (b) As the left foot is raised, the whole body leans to the right so that the center of gravity shifts over the right foot. The dashed line in part (b) indicates the location of the center of gravity when the subject was standing on both feet. ©Kevin Strang

PHYSIOLOGICAL INQUIRY

- How might the posture shown in part (b) influence contractions of this individual's shoulder muscles?

Answer can be found at end of chapter.

called ***large fiber sensory neuropathy***, has extremely debilitating effects on posture and balance. Individuals with this disorder have to visually monitor the location of body parts in space at all times in order to maintain their posture and balance.

10.6 Walking

Walking requires the coordination of many muscles, each activated to a precise degree at a precise time. We initiate walking by allowing the body to fall forward to an unstable position and then moving one leg forward to provide support. When the extensor muscles are activated on the supported side of the body to bear the body's weight, the contralateral extensors are inhibited to allow the nonsupporting limb to flex and swing forward. The cyclical, alternating movements of walking are brought about largely by central pattern-generating networks of interneurons in the spinal cord at the local level. The interneuron networks coordinate the output of the various motor neuron pools that control the appropriate muscles of the arms, shoulders, trunk, hips, legs, and feet.

The network neurons rely on both plasma membrane spontaneous pacemaker properties and patterned synaptic activity to establish their rhythms. At the same time, however, the networks

are remarkably adaptable and a single network can generate many different patterns of neural activity, depending upon its inputs. These inputs come from other local interneurons, afferent fibers, and descending pathways.

These complex spinal cord neural networks can even produce the rhythmic movement of limbs in the absence of command inputs from descending pathways or sensory feedback. This was demonstrated in classical experiments involving animals with their cerebrums surgically separated from their spinal cords just above the brainstem. Though voluntary movement was completely absent, normal walking and running actions could be initiated by activating pattern-generating circuits and reflex pathways in the spinal cord. This demonstrates that afferent inputs and local spinal cord neural networks contribute substantially to the coordination of locomotion.

Under normal conditions, neural activation occurs in the cerebral cortex, cerebellum, and brainstem, as well as in the spinal cord during locomotion. Moreover, middle and higher levels of the motor control hierarchy are necessary for postural control, voluntary override commands (like breaking stride to jump over a puddle), and adaptations to the environment (like walking across a stream on unevenly spaced stepping stones). Damage to even small areas of the sensorimotor cortex can cause marked disturbances in gait, which demonstrates its importance in locomotor control. ■

SUMMARY

Skeletal muscles are controlled by their motor neurons. All the motor neurons that control a given muscle form a motor neuron pool.

Motor Control Hierarchy

I. The neural systems that control body movements can be conceptualized as being arranged in a motor control hierarchy.
   a. The highest level determines the general intention of an action.
   b. The middle level establishes a motor program and specifies the postures and movements needed to carry out the intended action, taking into account sensory information that indicates the body's position.
   c. The local level ultimately determines which motor neurons will be activated.
   d. As the movement progresses, information about what the muscles are doing feeds back to the motor control centers, which make program corrections.
   e. Almost all actions have voluntary and involuntary components.

Local Control of Motor Neurons

I. Most direct input to motor neurons comes from local interneurons, which themselves receive input from peripheral receptors, descending pathways, and other interneurons.
II. Muscle-spindle stretch receptors monitor muscle length and the velocity of changes in length.
   a. Activation of these receptors initiates the stretch reflex, which inhibits motor neurons of ipsilateral antagonists and activates those of the stretched muscle and its synergists. This provides negative feedback control of muscle length.
   b. Tension on the stretch receptors is maintained during muscle contraction by activation of gamma motor neurons to the spindle muscle fibers.
   c. Alpha and gamma motor neurons are generally coactivated.
III. Golgi tendon organs monitor muscle tension. Through interneurons, they help to coordinate limb position and stiffness during complex movements like walking and running, and also supply ascending information for conscious perception of muscle force.
IV. The withdrawal reflex excites the ipsilateral flexor muscles and inhibits the ipsilateral extensors. The crossed-extensor reflex excites the contralateral extensor muscles and inhibits the contralateral flexor muscles.

The Brain Motor Centers and the Descending Pathways They Control

I. Neurons in the motor cortex are anatomically arranged in a somatotopic map.
II. Different areas of sensorimotor cortex have different functions but much overlap in activity.
III. The basal nuclei form a link in a circuit that originates in and returns to sensorimotor cortex. These subcortical nuclei facilitate some motor behaviors and inhibit others.
IV. The cerebellum coordinates posture and movement and participates in motor learning.
V. The corticospinal pathways pass directly from the sensorimotor cortex to motor neurons in the spinal cord (or brainstem, in the case of the corticobulbar pathways) or, more commonly, to interneurons near the motor neurons.
   a. In general, neurons on one side of the brain control muscles on the other side of the body.
   b. Corticospinal pathways control predominantly fine, precise movements of the distal extremities.
   c. Some corticospinal fibers affect the transmission of information in afferent pathways.
VI. Other descending pathways arise in the brainstem, control muscles on the same side of the body, and are involved mainly in the coordination of large groups of muscles used in posture and locomotion.
VII. There is significant interaction between the two descending pathways.

Muscle Tone

I. Hypertonia, as seen in spasticity and rigidity, usually occurs with disorders of neurons in CNS integrating and descending pathways, generically referred to as upper motor neuron disorders.
II. Hypotonia can be seen with cerebellar disease or, more commonly, with disease of the alpha motor neurons or muscle.

Maintenance of Upright Posture and Balance

I. Maintenance of posture and balance depends upon inputs from the eyes, vestibular apparatus, and somatic proprioceptors.
II. To maintain balance, the body's center of gravity must be maintained over the body's base.
III. The crossed-extensor reflex is a postural reflex.

Walking

I. The activity of central pattern generating networks in the spinal cord brings about the cyclical, alternating movements of locomotion.
II. These pattern generators are controlled by corticospinal and brainstem descending pathways and affected by feedback and motor programs.

REVIEW QUESTIONS

1. Describe motor control in terms of the conceptual motor control hierarchy. Use the following terms: *highest, middle,* and *local levels; motor program; descending pathways;* and *motor neuron.*
2. List the characteristics of voluntary actions.
3. Picking up a book, for example, has both voluntary and involuntary components. List the components of this action and indicate whether each is voluntary or involuntary.
4. List the inputs that can converge on the interneurons active in local motor control.
5. Draw a muscle spindle within a muscle, labeling the spindle, intrafusal and extrafusal muscle fibers, stretch receptors, afferent fibers, and alpha and gamma efferent fibers.

6. Describe the components of the knee-jerk reflex (stimulus, receptor, afferent pathway, integrating center, efferent pathway, effector, and response).
7. Describe the major function of alpha–gamma coactivation.
8. Distinguish among the following areas of the cerebral cortex: sensorimotor, primary motor, premotor, and supplementary motor.
9. Contrast the two major types of descending motor pathways in terms of structure and function.
10. Describe the functions that the basal nuclei and cerebellum have in motor control.
11. Explain how hypertonia may result from disease of the descending pathways.
12. Explain how hypotonia may result from lower motor neuron disease.
13. Explain the function of the crossed-extensor reflex in postural stability.
14. Explain the function of the interneuronal networks in walking, incorporating in your discussion the following terms: *interneuron, reciprocal innervation, synergistic muscle, antagonist,* and *feedback*.

KEY TERMS

motor neuron pool

10.1 Motor Control Hierarchy

descending pathways
motor program
proprioception
sensorimotor cortex
voluntary movement

10.2 Local Control of Motor Neurons

alpha–gamma coactivation
alpha motor neurons
crossed-extensor reflex
extrafusal fibers
gamma motor neurons
Golgi tendon organs
intrafusal fibers
knee-jerk reflex
monosynaptic reflex
muscle spindle
muscle-spindle stretch receptors
polysynaptic
reciprocal innervation
stretch reflex
synergistic muscles
withdrawal reflex

10.3 The Brain Motor Centers and the Descending Pathways They Control

basal nuclei
brainstem pathways
corticobulbar pathway
corticospinal pathways
extrapyramidal system
motor cortex
parietal-lobe association cortex
premotor area
primary motor cortex
pyramidal system
pyramidal tracts
somatosensory cortex
somatotopic map
substantia nigra
supplementary motor cortex

10.4 Muscle Tone

lower motor neurons
muscle tone
upper motor neurons

10.5 Maintenance of Upright Posture and Balance

postural reflexes

CLINICAL TERMS

10.3 The Brain Motor Centers and the Descending Pathways They Control

akinesia
bradykinesia
cerebellar disease
deep brain stimulation
intention tremor
Levodopa (L-dopa)
MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine)
Parkinson's disease

10.4 Muscle Tone

amyotrophic lateral sclerosis (ALS)
clasp-knife phenomenon
cramps
flaccid
hypertonia
hypotonia
Lou Gehrig's disease
rigidity
spasms
spasticity
upper motor neuron disorders

10.5 Maintenance of Upright Posture and Balance

large fiber sensory neuropathy
postural reflexes

CHAPTER 10

Clinical Case Study: A Woman Develops Stiff Jaw Muscles After a Puncture Wound

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A 55-year-old woman with complaints of muscle pain was brought to an urgent-care clinic by her husband. The woman had trouble speaking, so her husband explained that over the previous 3 days, her back and jaw muscles had grown gradually stiffer and more painful. By the time of her visit, she could barely open her mouth wide enough to drink through a straw. Until that week, she had been extremely healthy, had no history of allergies or surgical procedures, and was not taking any regular medications. At the time of examination, her blood pressure was 122/70 mmHg and her temperature was 98.5°F. Other than a stiff jaw, findings from a head and neck exam were otherwise unremarkable, her lung sounds were clear, and her heart sounds were normal.

Evaluating her extremities, the physician noticed that her right leg was bandaged just below the knee. A little over a week prior to this visit, she had been working in her garden and had stumbled and fallen onto a rake, puncturing her shin. The wound had not bled a great deal, so she had washed and bandaged it herself. Removal of the bandage revealed a raised, 5-cm-wide erythematous (reddened) region, surrounding a 0.5 cm puncture wound that had scabbed over. The doctor then asked a key question, When had she received her most recent tetanus booster shot? It had been so long ago that neither the woman nor her husband could remember exactly when it was—more than 20 years, they guessed. This piece of information, along with her leg wound and symptoms, led the physician to conclude that the woman had developed tetanus. Because this is a potentially fatal condition, she was admitted to the hospital.

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Reflect and Review #1

- What are the two basic ways in which alpha motor neurons are controlled at the level of the spinal cord?

Tetanus is a neurological disorder that results from a decrease in the inhibitory input to alpha motor neurons. It occurs when spores of *Clostridium tetani,* a bacterium commonly found in manure-treated soils, invade a poorly oxygenated wound (**Figure 10.14**). Proliferation of the bacterium under anaerobic conditions induces it to secrete a neurotoxin called ***tetanospasmin*** (sometimes referred to as tetanus toxin or tetanus neurotoxin; see Chapter 6) that enters alpha motor neurons and is then transported backward (retrogradely) into the CNS. Once there, it is released onto inhibitory interneurons in the brainstem and spinal cord. The toxin blocks the release of inhibitory neurotransmitter from these interneurons. This allows the normal excitatory inputs to dominate control of the alpha motor neurons, and the result is high-frequency action potential firing that causes increased muscle tone and spasms.

Because the toxin attacks interneurons by traveling backward along the axons of alpha motor neurons, muscles with short motor neurons are affected first. Muscles of the head are in this category, in particular those that move the jaw. The jaw rigidly clamps shut, because the muscles that close it are much stronger than those that open it. Appearance of this symptom early in the disease process explains the common name of this condition, ***lockjaw.*** Untreated tetanus is fatal, as progressive spastic contraction of all of the skeletal muscles eventually affects those involved in respiration, and asphyxia occurs.

Treatment for tetanus includes (1) cleaning and sterilizing wounds; (2) administering antibiotics to kill the bacteria; (3) injecting antibodies known as ***tetanus immune globulin (TIG)*** that bind the toxin, (4) providing neuromuscular blocking drugs to relax and/or paralyze spastic muscles; and (5) mechanically ventilating the lungs to maintain airflow despite spastic or paralyzed respiratory muscles. Treated promptly, 80% to 90% of tetanus victims recover completely. It can take several months, however, because inhibitory axon terminals damaged by the toxin must be regrown.

The patient in this case was fortunate to have had partial immunity from vaccinations received earlier in her life and to have received prompt treatment. Her disease was relatively mild as a result and did not require weeks of hospitalization with drug-induced paralysis and ventilation, as is necessary in more serious cases. She was immediately given intramuscular injections of TIG and a combination of strong antibiotics to be taken for the next 10 days. The leg wound was surgically opened, thoroughly cleaned, and monitored closely over the next week as the redness and swelling gradually subsided. Within 2 days, her jaw and back muscles had relaxed. She was released from the hospital with orders to continue the complete course of antibiotics and return immediately if any muscular symptoms returned. At the time of discharge, she was also vaccinated to stimulate production of her own antibodies against the tetanus toxin and was advised to receive booster shots against tetanus at least every 10 years.

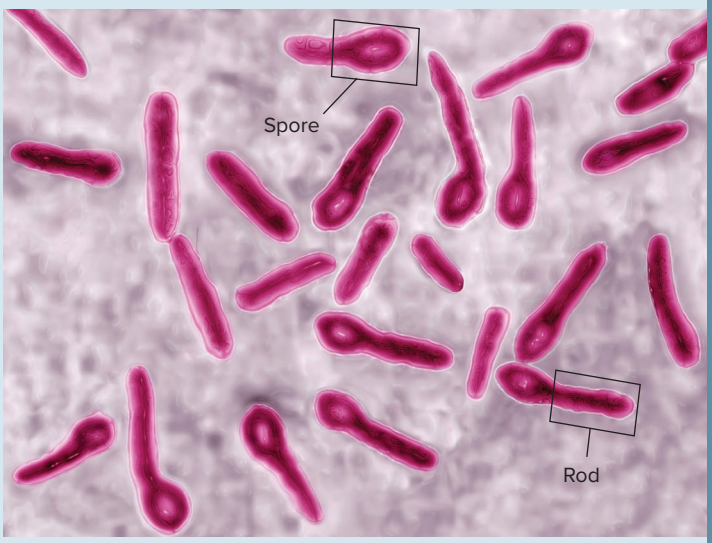

Figure 10.14 *Clostridium tetani* (magnification approximately 1000x). The mature bacteria contain a rod-like region and a spore region that contains the DNA and that is extremely resistant to heat and other environmental challenges. ©BSIP/UIG/Getty Images

Clinical terms: lockjaw, tetanospasmin, tetanus, tetanus immune globulin (TIG)

See Chapter 19 for complete, integrative case studies.

CHAPTER 10 TEST QUESTIONS *Recall and Comprehend*

Answers appear in Appendix A.

These questions test your recall of important details covered in this chapter. They also help prepare you for the type of questions encountered in standardized exams. Many additional questions of this type are available on Connect and LearnSmart.

1. Which is a correct statement regarding the hierarchical organization of motor control?
   a. Skeletal muscle contraction can only be initiated by neurons in the cerebral cortex.
   b. The basal nuclei participate in the creation of a motor program that specifies the pattern of neural activity required for a voluntary movement.
   c. Neurons in the cerebellum have long axons that synapse directly on alpha motor neurons in the ventral horn of the spinal cord.
   d. The cell bodies of alpha motor neurons are found in the primary motor region of the cerebral cortex.
   e. Neurons with cell bodies in the basal nuclei can form either excitatory or inhibitory synapses onto skeletal muscle cells.

2. In the stretch reflex,
   a. Golgi tendon organs activate contraction in extrafusal muscle fibers connected to that tendon.
   b. lengthening of muscle-spindle receptors in a muscle leads to contraction in an antagonist muscle.
   c. action potentials from muscle-spindle receptors in a muscle form monosynaptic excitatory synapses on motor neurons to extrafusal fibers within the same muscles.
   d. slackening of intrafusal fibers within a muscle activates gamma motor neurons that form excitatory synapses with extrafusal fibers within that same muscle.
   e. afferent neurons to the sensorimotor cortex stimulate the agonist muscle to contract and the antagonist muscle to be inhibited.

3. Which would result in reflex contraction of the extensor muscles of the right leg?
   a. stepping on a tack with the left foot
   b. stretching the flexor muscles in the right leg
   c. dropping a hammer on the right big toe
   d. action potentials from nociceptors of the right leg
   e. action potentials from muscle-spindle receptors in flexors of the right leg
4. If implanted electrodes were used to stimulate action potentials in gamma motor neurons to flexors of the left arm, which would be the most likely result?
   a. inhibition of the flexors of the left arm
   b. a decrease in action potentials from muscle-spindle receptors in the left arm
   c. a decrease in action potentials from Golgi tendon organs in the left arm
   d. an increase in action potentials along alpha motor neurons to flexors in the left arm
   e. contraction of flexor muscles in the right arm
5. Where is the primary motor cortex found?
   a. in the cerebellum
   b. in the occipital lobe of the cerebrum
   c. between the somatosensory cortex and the premotor area of the cerebrum
   d. in the ventral horn of the spinal cord
   e. just posterior to the parietal lobe association cortex

True or False

6. Neurons in the primary motor cortex of the right cerebral hemisphere mainly control muscles on the left side of the body.
7. Patients with upper motor neuron disorders generally have reduced muscle tone and flaccid paralysis.
8. Neurons descending in the corticospinal pathway control mainly trunk musculature and postural reflexes, whereas neurons of the brainstem pathways control fine motor movements of the distal extremities.
9. In patients with Parkinson's disease, an excess of dopamine from neurons of the substantia nigra causes intention tremors when the person performs voluntary movements.
10. The disease tetanus results when a bacterial toxin blocks the release of inhibitory neurotransmitter.

CHAPTER 10 TEST QUESTIONS *Apply, Analyze, and Evaluate*

Answers appear in Appendix A.

These questions, which are designed to be challenging, require you to integrate concepts covered in the chapter to draw your own conclusions. See if you can first answer the questions without using the hints that are provided; then, if you are having difficulty, refer back to the figures or sections indicated in the hints.

1. What changes would occur in the knee-jerk reflex after destruction of the gamma motor neurons? *Hint:* Think about whether the intrafusal fibers are stretched or flaccid when this test is performed.
2. What changes would occur in the knee-jerk reflex after destruction of the alpha motor neurons? *Hint:* See Figure 10.5; what are the functions of alpha motor neurons?
3. Draw a cross section of the spinal cord and a portion of the thigh (similar to Figure 10.6) and "wire up" and activate the neurons so the leg becomes a stiff pillar, that is, so the knee does not bend. *Hint:* Remember to include both extensors and flexors.
4. Hypertonia is usually considered a sign of disease of the descending motor pathways. How might it also result from abnormal function of the alpha motor neurons? *Hint:* Think about inhibitory synapses.
5. What neurotransmitters/receptors might be effective targets for drugs used to prevent the muscle spasms characteristic of the disease tetanus? *Hint:* Think about the concept of agonists and antagonists first described in Chapter 6.

CHAPTER 10 TEST QUESTIONS *General Principles Assessment*

Answers appear in Appendix A.

These questions reinforce the key theme first introduced in Chapter 1, that general principles of physiology can be applied across all levels of organization and across all organ systems.

1. One of the general principles of physiology introduced in Chapter 1 states that *most physiological functions are controlled by multiple regulatory systems, often working in opposition.* However, skeletal muscle cells are only innervated by alpha motor neurons, which always release acetylcholine and always excite them to contract. By what mechanism are skeletal muscles induced to relax?
2. Another general principle of physiology is that *homeostasis is essential for health and survival.* How might the withdrawal reflex (see Figure 10.8) contribute to the maintenance of homeostasis?

CHAPTER 10 ANSWERS TO PHYSIOLOGICAL INQUIRY QUESTIONS

Figure 10.3 Recall that when chloride ion channels are opened, a neuron is inhibited from depolarizing to threshold (see Figures 6.29 and 6.30 and accompanying text). Thus, the neurons of the spinal cord that release glycine are inhibitory interneurons. By specifically blocking glycine receptors, strychnine shifts the balance of inputs to motor neurons in favor of excitatory interneurons, resulting in excessive excitation. Poisoning victims experience excessive and uncontrollable muscle contractions body-wide; when the respiratory muscles are affected, asphyxiation can occur. These symptoms are similar to those observed in the disease state tetanus, which is described in the Clinical Case Study at the end of this chapter.

Figure 10.6 Stimulation of gamma motor neurons to leg flexor muscles would stretch muscle-spindle receptors in those muscles. That would trigger a monosynaptic reflex that would cause contraction of the flexor muscles and, through an interneuron, the extensor muscles would be inhibited. As a result, there would be a reflexive bending of the leg—the opposite of what occurs in the typical knee-jerk reflex.

Figure 10.7 Although the contracting muscle results in the greatest stretch of the tendon, the muscle itself (and consequently the intrafusal fibers) are stretched the most under passive stretch conditions. Action potentials from muscle-spindle receptors would therefore have the greatest frequency during passive stretch.

Figure 10.8 When crawling, the crossed-extensor reflex will occur for the arms just like it does in the legs during walking. Afferent pain pathways will stimulate flexor muscles and inhibit extensor muscles in the right arm, while stimulating extensor muscles and inhibiting flexor muscles in the left arm. This withdraws the right hand from the painful stimulus while the left arm straightens to bear the child's weight.

Figure 10.10 Different regions of the primary motor cortex have evolved different numbers of neurons associated with the specific features of the movements of particular body parts. In this way, the structural organization of the primary motor cortex is correlated with the functional ability of different body parts. An example is the fine motor control necessary for the movement of fingers while playing a piano; such movements require many more motor neurons than does the ability to move one's toes.

Figure 10.11 When a region of the brain is deprived of oxygen and nutrients for even a short time, it often results in a stroke—neuronal cell death (see Chapter 6, Section D). Because the right primary motor cortex was damaged in this case, the patient would have impaired motor function on the left side of the body. Given the midline location of the lesion, the leg would be most affected (see Figure 10.10).

Figure 10.12 Gravity not only influences posture and balance but also places constraints on many types of motor behaviors, such as jumping or even walking. Simply lifting one's leg up to take a step requires energy to overcome gravity and to maintain a stable posture and gait. In addition, gravity influences the movement of fluids in the body, such as the flow of blood up to one's head while standing.

Figure 10.13 To stand on the right foot, the hip extensors on the right side are activated while the hip flexors on the left side are activated. This is similar to what occurs when a walking person lifts the left leg and pushes forward with the right foot. In adults, spinal cord interneurons form locomotor pattern generators that connect the arms and legs, typically activating them in reciprocal fashion. Therefore, while standing on the right foot, the right shoulder flexor muscles and the left shoulder extensor muscles will tend to be activated.

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