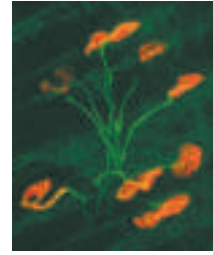


# Chapter 15



## Lower Motor Neuron Circuits and Motor Control

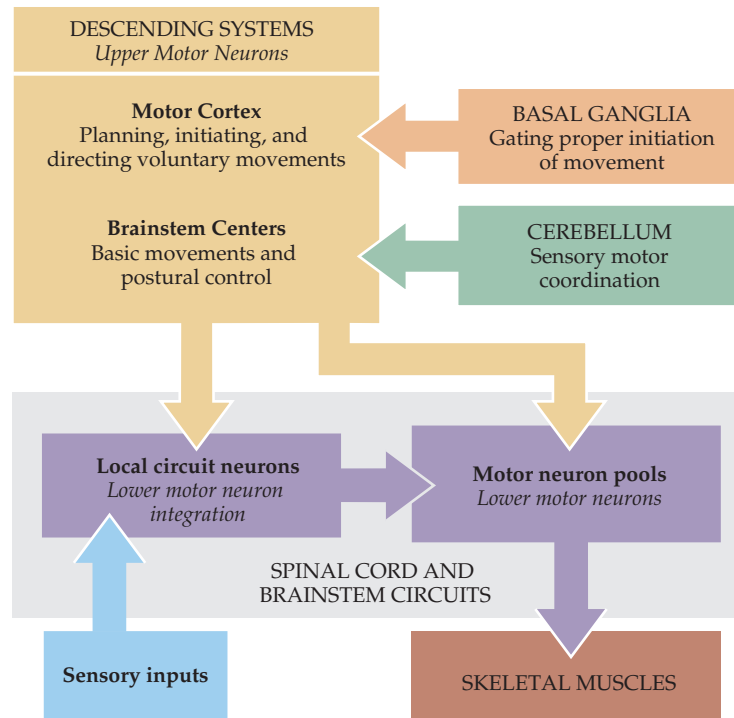
### Overview

Skeletal (striated) muscle contraction is initiated by “lower” motor neurons in the spinal cord and brainstem. The cell bodies of the lower neurons are located in the ventral horn of the spinal cord gray matter and in the motor nuclei of the cranial nerves in the brainstem. These neurons (also called  $\alpha$  motor neurons) send axons directly to skeletal muscles via the ventral roots and spinal peripheral nerves, or via cranial nerves in the case of the brainstem nuclei. The spatial and temporal patterns of activation of lower motor neurons are determined primarily by local circuits located within the spinal cord and brainstem. Descending pathways from higher centers comprise the axons of “upper” motor neurons and modulate the activity of lower motor neurons by influencing this local circuitry. The cell bodies of upper motor neurons are located either in the cortex or in brainstem centers, such as the vestibular nucleus, the superior colliculus, and the reticular formation. The axons of the upper motor neurons typically contact the local circuit neurons in the brainstem and spinal cord, which, via relatively short axons, contact in turn the appropriate combinations of lower motor neurons. The local circuit neurons also receive direct input from sensory neurons, thus mediating important sensory motor reflexes that operate at the level of the brainstem and spinal cord. Lower motor neurons, therefore, are the final common pathway for transmitting neural information from a variety of sources to the skeletal muscles.

### Neural Centers Responsible for Movement

The neural circuits responsible for the control of movement can be divided into four distinct but highly interactive subsystems, each of which makes a unique contribution to motor control (Figure 15.1). The first of these subsystems is the local circuitry within the gray matter of the spinal cord and the analogous circuitry in the brainstem. The relevant cells include the **lower motor neurons** (which send their axons out of the brainstem and spinal cord to innervate the skeletal muscles of the head and body, respectively) and the **local circuit neurons** (which are the major source of synaptic input to the lower motor neurons). All commands for movement, whether reflexive or voluntary, are ultimately conveyed to the muscles by the activity of the lower motor neurons; thus these neurons comprise, in the words of the great British neurophysiologist Charles Sherrington, the “final common path” for movement. The local circuit neurons receive sensory inputs as well as descending projections from higher centers. Thus, the circuits they form provide much of the coordination between different muscle groups that is

**Figure 15.1** Overall organization of neural structures involved in the control of movement. Four systems—local spinal cord and brainstem circuits, descending modulatory pathways, the cerebellum, and the basal ganglia—make essential and distinct contributions to motor control.



essential for organized movement. Even after the spinal cord is disconnected from the brain in an experimental animal such as a cat, appropriate stimulation of local spinal circuits elicits involuntary but highly coordinated limb movements that resemble walking.

The second motor subsystem consists of the **upper motor neurons** whose cell bodies lie in the brainstem or cerebral cortex and whose axons descend to synapse with the local circuit neurons or, more rarely, with the lower motor neurons directly. The upper motor neuron pathways that arise in the cortex are essential for the initiation of voluntary movements and for complex spatiotemporal sequences of skilled movements. In particular, descending projections from cortical areas in the frontal lobe, including Brodmann's area 4 (the **primary motor cortex**), the lateral part of area 6 (the **lateral premotor cortex**), and the medial part of area 6 (the **medial premotor cortex**) are essential for planning, initiating, and directing sequences of voluntary movements. Upper motor neurons originating in the brainstem are responsible for regulating muscle tone and for orienting the eyes, head, and body with respect to vestibular, somatic, auditory, and visual sensory information. Their contributions are thus critical for basic navigational movements, and for the control of posture.

The third and fourth subsystems are complex circuits with output pathways that have no direct access to either the local circuit neurons or the lower motor neurons; instead, they control movement by regulating the activity of the upper motor neurons. The third and larger of these subsystems, the **cerebellum**, is located on the dorsal surface of the pons (see Chapter 1). The cerebellum acts via its efferent pathways to the upper motor neurons as a servomechanism, detecting the difference, or "motor error," between an intended movement and the movement actually performed (see Chapter 19). The cerebellum uses this information about discrepancies to

mediate both real-time and long-term reductions in these motor errors (the latter being a form of motor learning). As might be expected from this account, patients with cerebellar damage exhibit persistent errors in movement. The fourth subsystem, embedded in the depths of the forebrain, consists of a group of structures collectively referred to as the **basal ganglia** (see Chapter 1). The basal ganglia suppress unwanted movements and prepare (or “prime”) upper motor neuron circuits for the initiation of movements. The problems associated with disorders of basal ganglia, such as Parkinson’s disease and Huntington’s disease, attest to the importance of this complex in the initiation of voluntary movements (see Chapter 17).

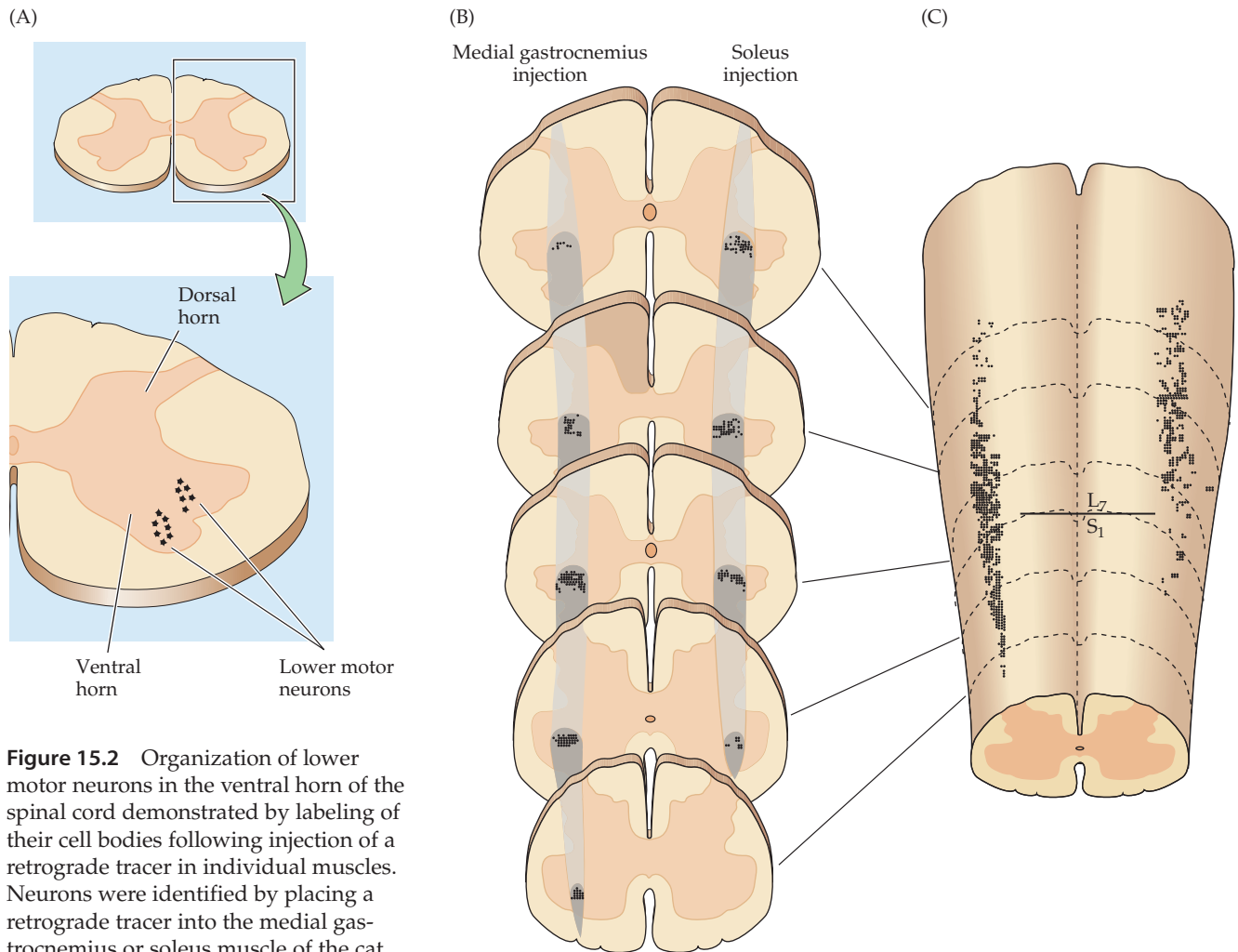
Despite much effort, the sequence of events that leads from volitional thought to movement is still poorly understood. The picture is clearest, however, at the level of control of the muscles themselves. It therefore makes sense to begin an account of motor behavior by considering the anatomical and physiological relationships between lower motor neurons and the muscle fibers they innervate.

### Motor Neuron–Muscle Relationships

By injecting individual muscle groups with visible tracers that are transported by the axons of the lower motor neurons back to their cell bodies, the lower motor neurons that innervate each of the body’s skeletal muscles can be seen in histological sections of the ventral horns of the spinal cord. Each lower motor neuron innervates muscle fibers within a single muscle, and all the motor neurons innervating a single muscle (called the **motor neuron pool** for that muscle) are grouped together into rod-shaped clusters that run parallel to the long axis of the cord for one or more spinal cord segments (Figure 15.2).

An orderly relationship between the location of the motor neuron pools and the muscles they innervate is evident both along the length of the spinal cord and across the mediolateral dimension of the cord, an arrangement that in effect provides a spatial map of the body’s musculature. For example, the motor neuron pools that innervate the arm are located in the cervical enlargement of the cord and those that innervate the leg in the lumbar enlargement (see Chapter 1). The mapping, or topography, of motor neuron pools in the mediolateral dimension can be appreciated in a cross section through the cervical enlargement (the level illustrated in Figure 15.3). Thus, neurons that innervate the axial musculature (i.e., the postural muscles of the trunk) are located medially in the cord. Lateral to these cell groups are motor neuron pools innervating muscles located progressively more laterally in the body. Neurons that innervate the muscles of the shoulders (or pelvis, if one were to look at a similar section in the lumbar enlargement; see Figure 15.2) are the next most lateral group, whereas those that innervate the proximal muscles of the arm (or leg) are located laterally to these. The motor neuron pools that innervate the distal parts of the extremities, the fingers or toes, lie farthest from the midline. This spatial organization provides clues about the functions of the descending upper motor neuron pathways described in the following chapter; some of these pathways terminate primarily in the medial region of the spinal cord, which is concerned with postural muscles, whereas other pathways terminate more laterally, where they have access to the lower motor neurons that control movements of the distal parts of the limbs, such as, the toes and the fingers.

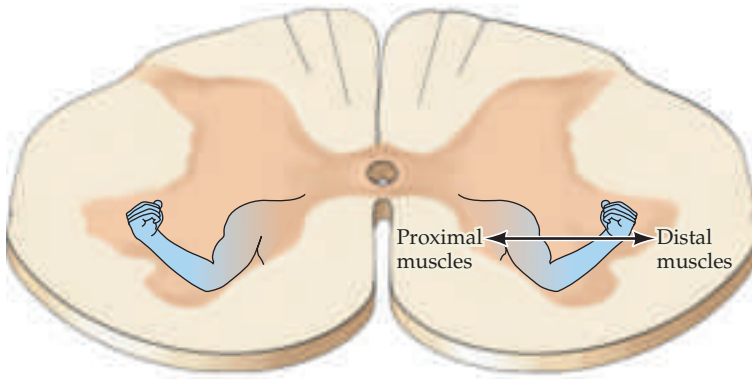
Two types of lower motor neuron are found in these neuronal pools. Small  $\gamma$  **motor neurons** innervate specialized muscle fibers that, in combina-



**Figure 15.2** Organization of lower motor neurons in the ventral horn of the spinal cord demonstrated by labeling of their cell bodies following injection of a retrograde tracer in individual muscles. Neurons were identified by placing a retrograde tracer into the medial gastrocnemius or soleus muscle of the cat. (A) Section through the lumbar level of the spinal cord showing the distribution of labeled cell bodies. Lower motor neurons form distinct clusters (motor pools) in the ventral horn. Spinal cord cross sections (B) and a reconstruction seen from the dorsal surface (C) illustrate the distribution of motor neurons innervating individual skeletal muscles in both axes of the cord. The cylindrical shape and distinct distribution of different pools are especially evident in the dorsal view of the reconstructed cord. The dashed lines in (C) represent individual lumbar and sacral spinal cord segments. (After Burke et al., 1977.)

tion with the nerve fibers that innervate them, are actually sensory receptors called muscle spindles (see Chapter 8). The muscle spindles are embedded within connective tissue capsules in the muscle, and are thus referred to as *intrafusal* muscle fibers (*fusal* means capsular). The *intrafusal* muscle fibers are also innervated by sensory axons that send information to the brain and spinal cord about the length and tension of the muscle. The function of the  $\gamma$  motor neurons is to regulate this sensory input by setting the *intrafusal* muscle fibers to an appropriate length (see the next section). The second type of lower motor neuron, called  **$\alpha$  motor neurons**, innervates the *extrafusal* muscle fibers, which are the striated muscle fibers that actually generate the forces needed for posture and movement.

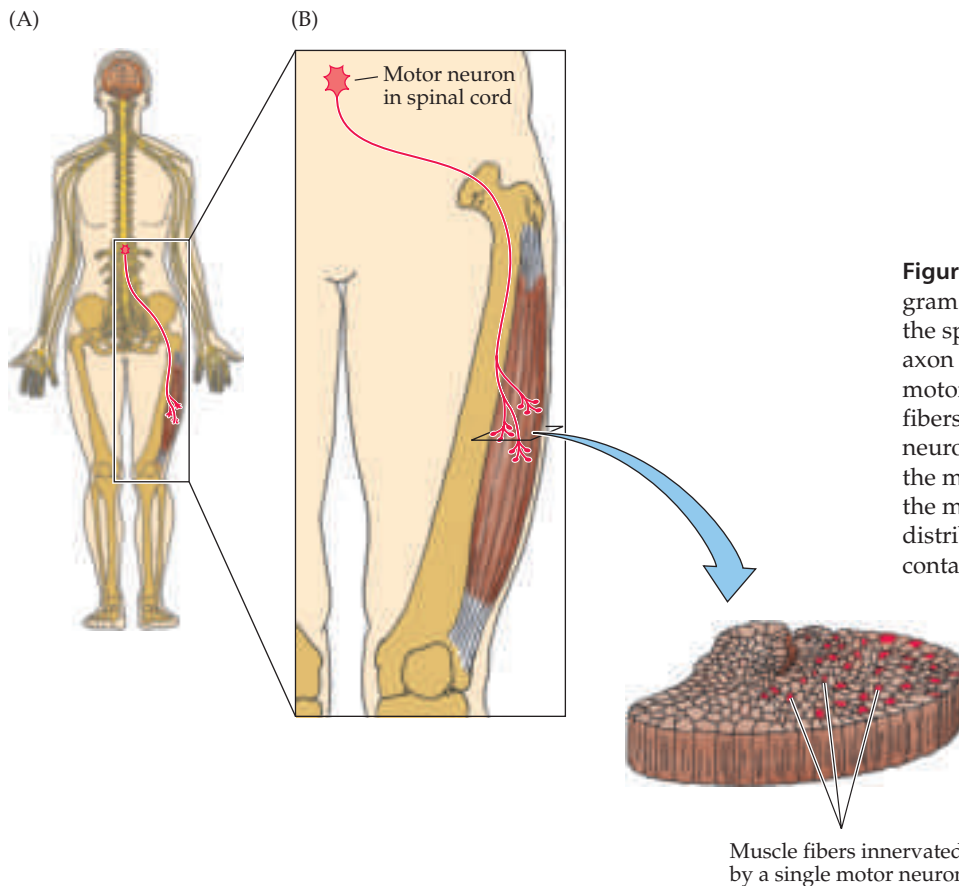
Although the following discussion focuses on the lower motor neurons in the spinal cord, comparable sets of motor neurons responsible for the control of muscles in the head and neck are located in the brainstem. The latter neurons are distributed in the eight motor nuclei of the cranial nerves in the medulla, pons, and midbrain (see Appendix A). Somewhat confusingly, but quite appropriately, these motor neurons in the brainstem are also called lower motor neurons.



**Figure 15.3** Somatotopic organization of lower motor neurons in a cross section of the ventral horn at the cervical level of the spinal cord. Motor neurons innervating axial musculature are located medially, whereas those innervating the distal musculature are located more laterally.

### The Motor Unit

Most mature extrafusal skeletal muscle fibers in mammals are innervated by only a single  $\alpha$  motor neuron. Since there are by far more muscle fibers than motor neurons, individual motor axons branch within muscles to synapse on many different fibers that are typically distributed over a relatively wide area within the muscle, presumably to ensure that the contractile force of the motor unit is spread evenly (Figure 15.4). In addition, this arrangement reduces the chance that damage to one or a few  $\alpha$  motor neurons will significantly alter a muscle's action. Because an action potential generated by a



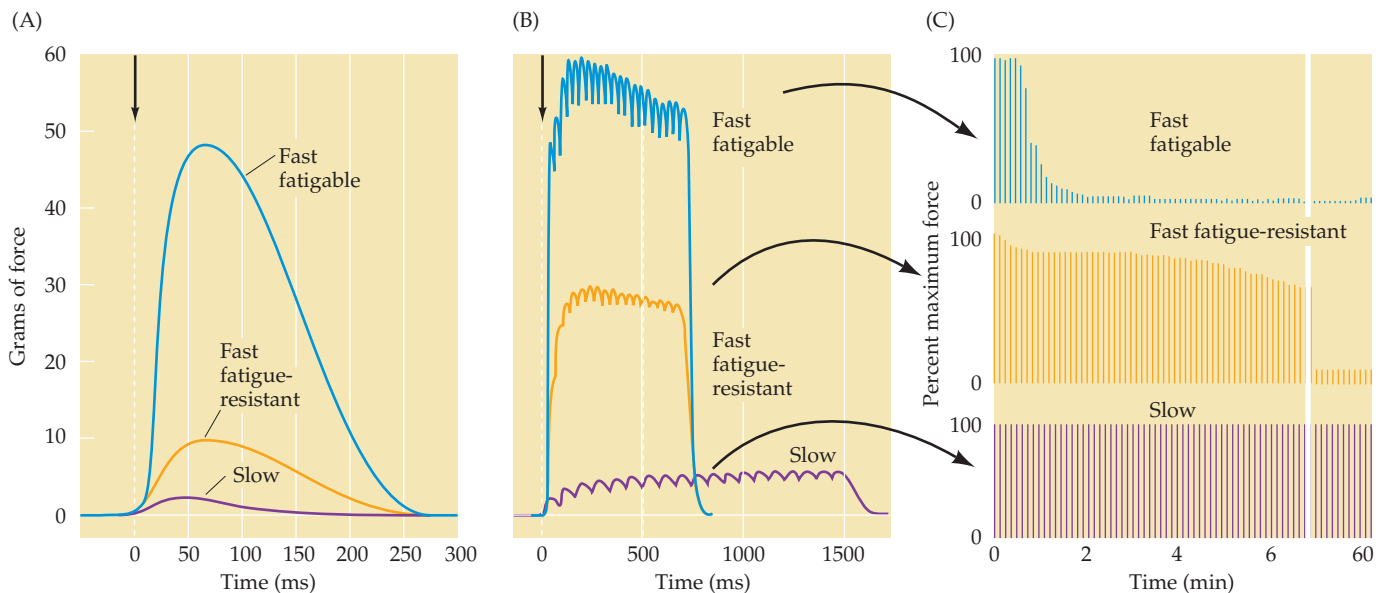
**Figure 15.4** The motor unit. (A) Diagram showing a lower motor neuron in the spinal cord and the course of its axon to its target muscle. (B) Each motor neuron synapses with multiple fibers within the muscle. The motor neuron and the fibers it contacts define the motor unit. Cross section through the muscle shows the relatively diffuse distribution of muscle fibers (red dots) contacted by the motor neuron.

motor neuron normally brings to threshold all of the muscle fibers it contacts, a single  $\alpha$  motor neuron and its associated muscle fibers together constitute the smallest unit of force that can be activated to produce movement. Sherrington was again the first to recognize this fundamental relationship between an  $\alpha$  motor neuron and the muscle fibers it innervates, for which he coined the term **motor unit**.

Both motor units and the  $\alpha$  motor neurons themselves vary in size. Small  $\alpha$  motor neurons innervate relatively few muscle fibers and form motor units that generate small forces, whereas large motor neurons innervate larger, more powerful motor units. Motor units also differ in the types of muscle fibers that they innervate. In most skeletal muscles, the smaller motor units comprise small “red” muscle fibers that contract slowly and generate relatively small forces; but, because of their rich myoglobin content, plentiful mitochondria, and rich capillary beds, such small red fibers are resistant to fatigue (these units are also innervated by relatively small  $\alpha$  motor neurons). These small units are called **slow (S) motor units** and are especially important for activities that require sustained muscular contraction, such as the maintenance of an upright posture. Larger  $\alpha$  motor neurons innervate larger, pale muscle fibers that generate more force; however, these fibers have sparse mitochondria and are therefore easily fatigued. These units are called **fast fatigable (FF) motor units** and are especially important for brief exertions that require large forces, such as running or jumping. A third class of motor units has properties that lie between those of the other two. These **fast fatigue-resistant (FR) motor units** are of intermediate size and are not quite as fast as FF units. They generate about twice the force of a slow motor unit and, as the name implies, are substantially more resistant to fatigue (Figure 15.5).

These distinctions among different types of motor units indicate how the nervous system produces movements appropriate for different circumstances. In most muscles, small, slow motor units have lower thresholds for activation than the larger units and are tonically active during motor acts that require sustained effort (standing, for instance). The thresholds for the large, fast motor units are reached only when rapid movements requiring great force are made, such as jumping. The functional distinctions between

**Figure 15.5** Comparison of the force and fatigability of the three different types of motor units. In each case, the response reflects stimulation of a single motor neuron. (A) Change in muscle tension in response to a single motor neuron action potential. (B) Tension in response to repetitive stimulation of the motor neurons. (C) Response to repeated stimulation at a level that evokes maximum tension. The ordinate represents the force generated by each stimulus. Note the strikingly different fatigue rates. (After Burke et al., 1974.)



the various classes of motor units also explain some structural differences among muscle groups. For example, a motor unit in the soleus (a muscle important for posture that comprises mostly small, slow units) has an average innervation ratio of 180 muscle fibers for each motor neuron. In contrast, the gastrocnemius, a muscle that comprises both small and larger units, has an innervation ratio of ~1000–2000 muscle fibers per motor neuron, and can generate forces needed for sudden changes in body position. More subtle variations are present in athletes on different training regimens. Thus, muscle biopsies show that sprinters have a larger proportion of powerful but rapidly fatiguing pale fibers in their leg muscles than do marathoners. Other differences are related to the highly specialized functions of particular muscles. For instance, the eyes require rapid, precise movements but little strength; in consequence, extraocular muscle motor units are extremely small (with an average innervation ratio of only 3!) and have a very high proportion of muscle fibers capable of contracting with maximal velocity.

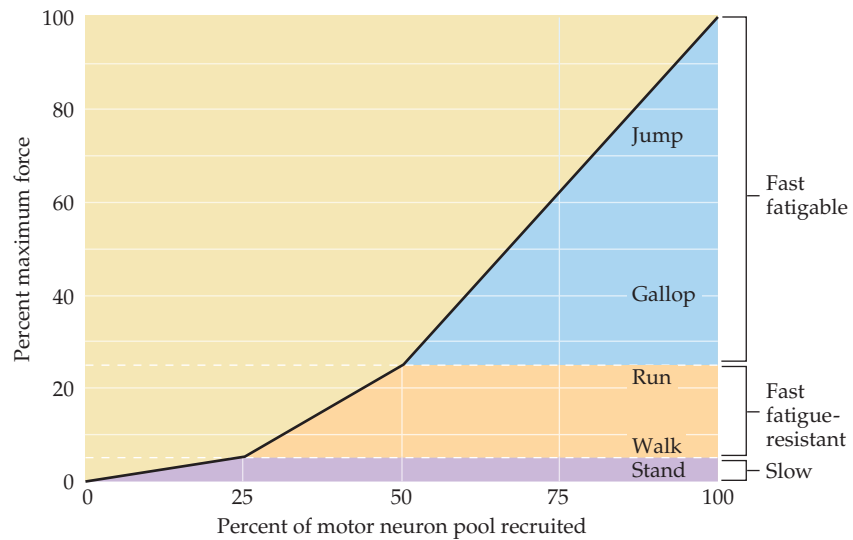
### The Regulation of Muscle Force

Increasing or decreasing the number of motor units active at any one time changes the amount of force produced by a muscle. In the 1960s, Elwood Henneman and his colleagues at Harvard Medical School found that progressive increases in muscle tension could be produced by progressively increasing the activity of axons that provide input to the relevant pool of lower motor neurons. This gradual increase in tension results from the recruitment of motor units in a fixed order according to their size. By stimulating either sensory nerves or upper motor pathways that project to a lower motor neuron pool while measuring the tension changes in the muscle, Henneman found that in experimental animals only the smallest motor units in the pool are activated by weak synaptic stimulation. When synaptic input increases, progressively larger motor units that generate larger forces are recruited: As the synaptic activity driving a motor neuron pool increases, low threshold S units are recruited first, then FR units, and finally, at the highest levels of activity, the FF units. Since these original experiments, evidence for the orderly recruitment of motor units has been found in a variety of voluntary and reflexive movements. As a result, this systematic relationship has come to be known as the **size principle**.

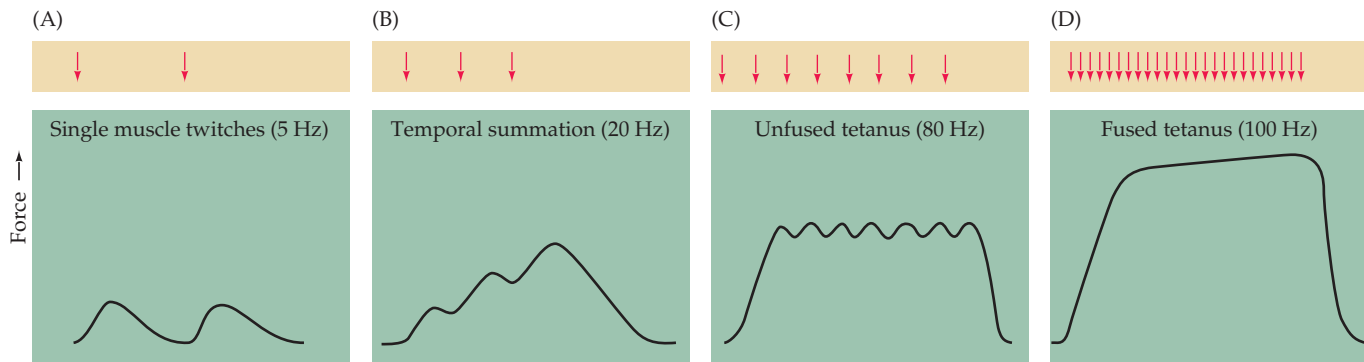
An illustration of how the size principle operates for the motor units of the medial gastrocnemius muscle in the cat is shown in Figure 15.6. When the animal is standing quietly, the force measured directly from the muscle tendon is only a small fraction (about 5%) of the total force that the muscle can generate. The force is provided by the S motor units, which make up about 25% of the motor units in this muscle. When the cat begins to walk, larger forces are necessary: locomotor activities that range from slow walking to fast running require up to 25% of the muscle's total force capacity. This additional need is met by the recruitment of FR units. Only movements such as galloping and jumping, which are performed infrequently and for short periods, require the full power of the muscle; such demands are met by the recruitment of the FF units. Thus, the size principle provides a simple solution to the problem of grading muscle force: The combination of motor units activated by such orderly recruitment optimally matches the physiological properties of different motor unit types with the range of forces required to perform different motor tasks.

The frequency of the action potentials generated by motor neurons also contributes to the regulation of muscle tension. The increase in force that

**Figure 15.6** The recruitment of motor neurons in the cat medial gastrocnemius muscle under different behavioral conditions. Slow (S) motor units provide the tension required for standing. Fast fatigue-resistant (FR) units provide the additional force needed for walking and running. Fast fatigable (FF) units are recruited for the most strenuous activities, such as jumping. (After Walmsley et al., 1978.)

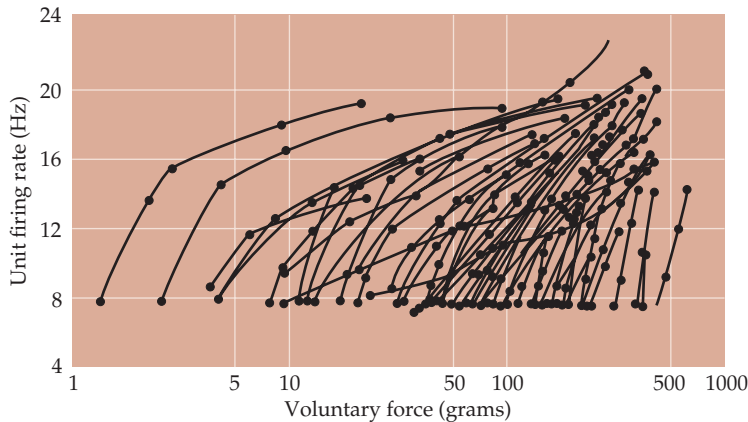


**Figure 15.7** The effect of stimulation rate on muscle tension. (A) At low frequencies of stimulation, each action potential in the motor neuron results in a single twitch of the related muscle fibers. (B) At higher frequencies, the twitches sum to produce a force greater than that produced by single twitches. (C) At a still higher frequency of stimulation, the force produced is greater, but individual twitches are still apparent. This response is referred to as unfused tetanus. (D) At the highest rates of motor neuron activation, individual twitches are no longer apparent (a condition called fused tetanus).



occurs with increased firing rate reflects the summation of successive muscle contractions: The muscle fibers are activated by the next action potential before they have had time to completely relax, and the forces generated by the temporally overlapping contractions are summed (Figure 15.7). The lowest firing rates during a voluntary movement are on the order of 8 per second (Figure 15.8). As the firing rate of individual units rises to a maximum of about 20–25 per second in the muscle being studied here, the amount of force produced increases. At the highest firing rates, individual muscle fibers are in a state of “fused tetanus”—that is, the tension produced in individual motor units no longer has peaks and troughs that correspond to the individual twitches evoked by the motor neuron’s action potentials. Under normal conditions, the maximum firing rate of motor neurons is less than that required for fused tetanus (see Figure 15.8). However, the asynchronous firing of different lower motor neurons provides a steady level of input to the muscle, which causes the contraction of a relatively constant number of motor units and averages out the changes in tension due to contractions and relaxations of individual motor units. All this allows the resulting movements to be executed smoothly.





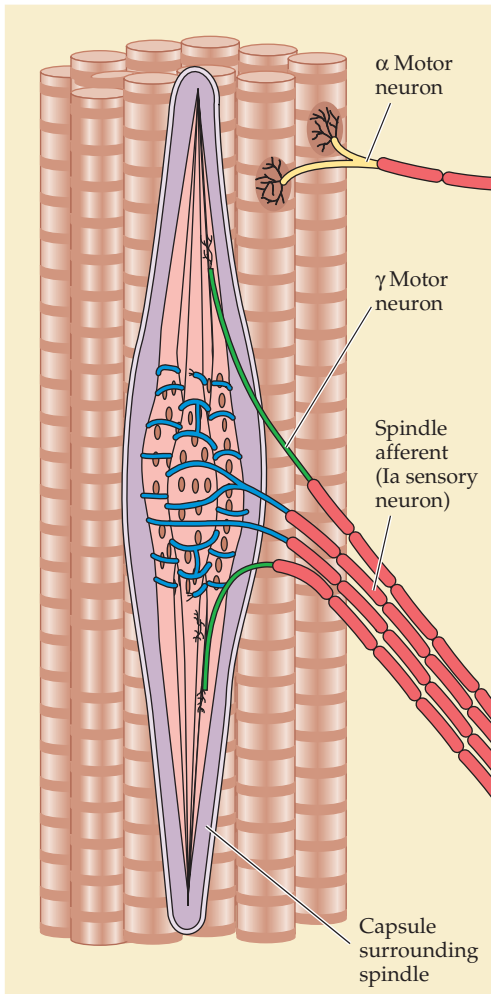
**Figure 15.8** Motor units recorded transcutaneously in a muscle of the human hand as the amount of voluntary force produced is progressively increased. Motor units (represented by the lines between the dots) are initially recruited at a low frequency of firing (8 Hz); the rate of firing for each unit increases as the subject generates more and more force. (After Monster and Chan, 1977.)

### The Spinal Cord Circuitry Underlying Muscle Stretch Reflexes

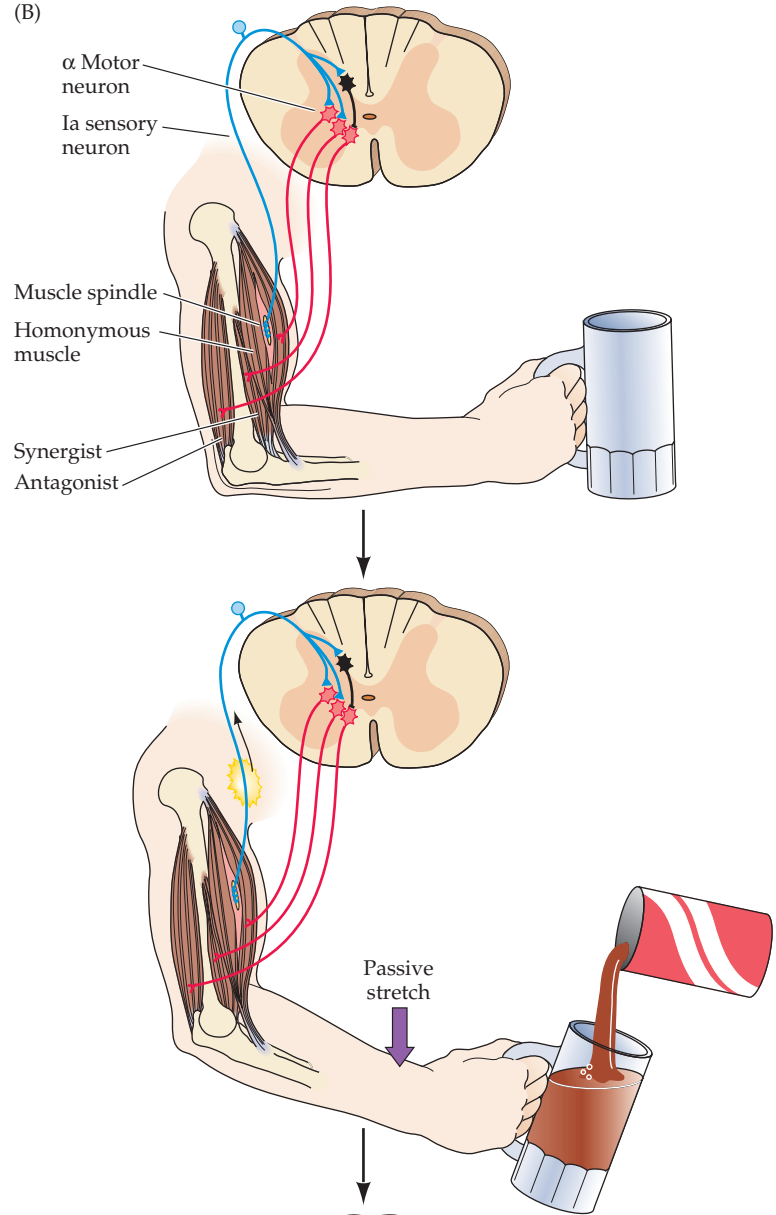
The local circuitry within the spinal cord mediates a number of sensory motor reflex actions. The simplest of these reflex arcs entails a sensory response to muscle stretch, which provides direct excitatory feedback to the motor neurons innervating the muscle that has been stretched (Figure 15.9). As already mentioned, the sensory signal for the **stretch reflex** originates in **muscle spindles**, the sensory receptors embedded within most muscles (see the previous section and Chapter 8). The spindles comprise 8–10 intrafusal fibers arranged in parallel with the extrafusal fibers that make up the bulk of the muscle (Figure 15.9A). Large-diameter sensory fibers, called Ia afferents, are coiled around the central part of the spindle. These afferents are the largest axons in peripheral nerves and, since action potential conduction velocity is a direct function of axon diameter (see Chapters 2 and 3), they mediate very rapid reflex adjustments when the muscle is stretched. The stretch imposed on the muscle deforms the intrafusal muscle fibers, which in turn initiate action potentials by activating mechanically gated ion channels in the afferent axons coiled around the spindle. The centrally projecting branch of the sensory neuron forms monosynaptic excitatory connections with the  $\alpha$  motor neurons in the ventral horn of the spinal cord that innervate the same (homonymous) muscle and, via local circuit neurons, forms inhibitory connections with the  $\alpha$  motor neurons of antagonistic (heteronymous) muscles. This arrangement is an example of what is called *reciprocal innervation* and results in rapid contraction of the stretched muscle and simultaneous relaxation of the antagonist muscle. All of this leads to especially rapid and efficient responses to changes in the length or tension in the muscle (Figure 15.9B). The excitatory pathway from a spindle to the  $\alpha$  motor neurons innervating the same muscle is unusual in that it is a monosynaptic reflex; in most cases, sensory neurons from the periphery do not contact the lower motor neuron directly but exert their effects through local circuit neurons.

This monosynaptic reflex arc is variously referred to as the “stretch,” “deep tendon,” or “myotatic” reflex, and it is the basis of the knee, ankle, jaw, biceps, or triceps responses tested in a routine neurological examination. The tap of the reflex hammer on the tendon stretches the muscle and therefore excites an afferent volley of activity in the Ia sensory axons that innervate the muscle spindles. The afferent volley is relayed to the  $\alpha$  motor neurons in the brainstem or spinal cord, and an efferent volley returns to the muscle (see Figure 1.5). Since muscles are always under some degree of

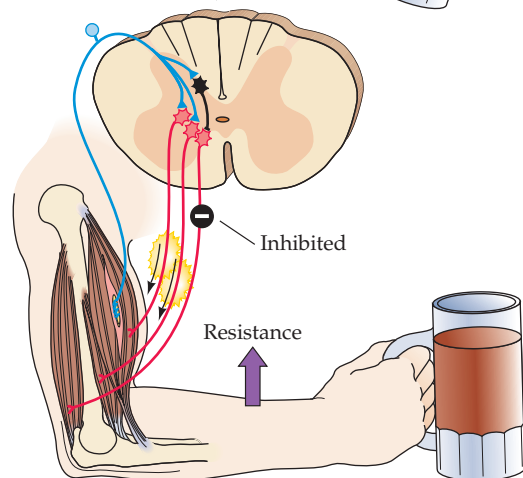
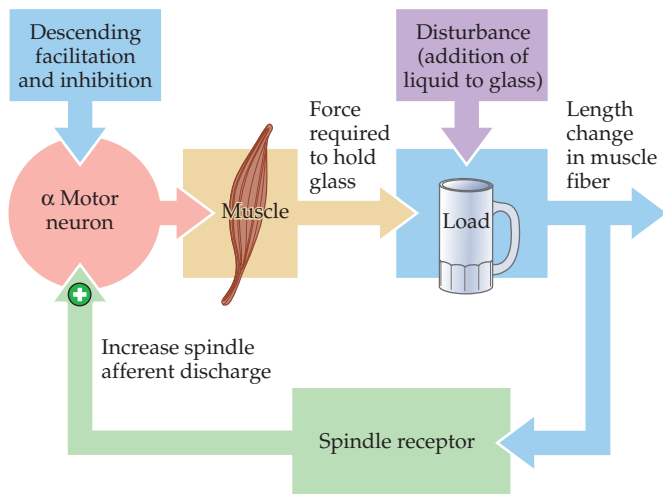
(A) Muscle spindle



(B)



(C)



◀ **Figure 15.9** Stretch reflex circuitry. (A) Diagram of muscle spindle, the sensory receptor that initiates the stretch reflex. (B) Stretching a muscle spindle leads to increased activity in Ia afferents and an increase in the activity of  $\alpha$  motor neurons that innervate the same muscle. Ia afferents also excite the motor neurons that innervate synergistic muscles, and inhibit the motor neurons that innervate antagonists (see also Figure 1.5). (C) The stretch reflex operates as a negative feedback loop to regulate muscle length.

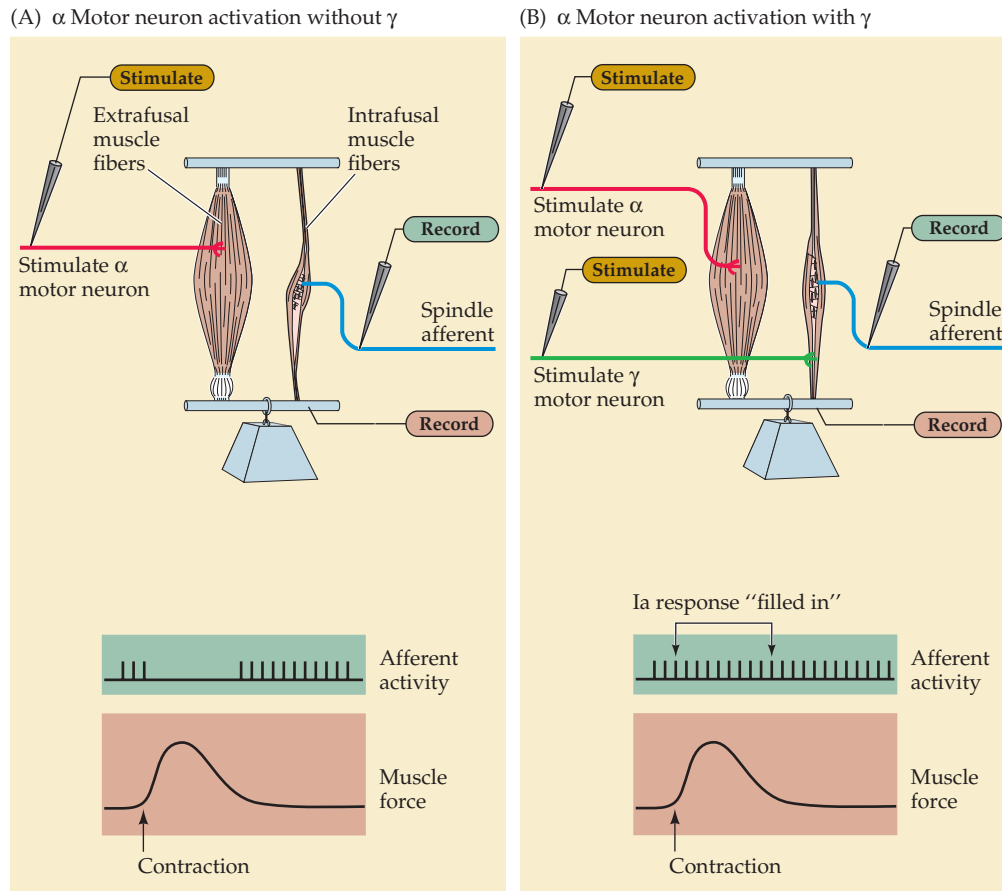
stretch, this reflex circuit is normally responsible for the steady level of tension in muscles called **muscle tone**. Changes in muscle tone occur in a variety of pathological conditions, and it is these changes that are assessed by examination of tendon reflexes.

In terms of engineering principles, the stretch reflex arc is a negative feedback loop used to maintain muscle length at a desired value (Figure 15.9C). The appropriate muscle length is specified by the activity of descending upper motor neuron pathways that influence the motor neuron pool. Deviations from the desired length are detected by the muscle spindles, since increases or decreases in the stretch of the intrafusal fibers alter the level of activity in the sensory axons that innervate the spindles. These changes lead in turn to adjustments in the activity of the  $\alpha$  motor neurons, returning the muscle to the desired length by contracting the stretched muscle and relaxing the opposed muscle group, and by restoring the level of spindle activity to what it was before.

The smaller  $\gamma$  motor neurons control the functional characteristics of the muscle spindles by modulating their level of excitability. As was described earlier, when the muscle is stretched, the spindle is also stretched and the rate of discharge in the afferent fibers increased. When the muscle shortens, however, the spindle is relieved of tension, or “unloaded,” and the sensory axons that innervate the spindle might therefore be expected to fall silent during contraction. However, they remain active. The  $\gamma$  motor neurons terminate on the contractile poles of the intrafusal fibers, and the activation of these neurons causes intrafusal fiber contraction—in this way maintaining the tension on the middle (or equatorial region) of the intrafusal fibers where the sensory axons terminate. Thus, co-activation of the  $\alpha$  and  $\gamma$  motor neurons allows spindles to function (i.e., send information centrally) at all muscle lengths during movements and postural adjustments.

### The Influence of Sensory Activity on Motor Behavior

The level of  $\gamma$  motor neuron activity often is referred to as  $\gamma$  bias, or gain, and can be adjusted by upper motor neuron pathways as well as by local reflex circuitry. The larger the gain of the stretch reflex, the greater the change in muscle force that results from a given amount of stretch applied to the intrafusal fibers. If the gain of the reflex is high, then a small amount of stretch applied to the intrafusal fibers will produce a large increase in the number of  $\alpha$  motor neurons recruited and a large increase in their firing rates; this in turn leads to a large increase in the amount of tension produced by the extrafusal fibers. If the gain is low, a greater stretch is required to generate the same amount of tension in the extrafusal muscle fibers. In fact, the gain of the stretch reflex is continuously adjusted to meet different functional requirements. For example, while standing in a moving bus, the gain of the stretch reflex can be modulated by upper motor neuron pathways to com-



**Figure 15.10** The role of  $\gamma$  motor neuron activity in regulating the responses of muscle spindles. (A) When  $\alpha$  motor neurons are stimulated without activation of  $\gamma$  motor neurons, the response of the Ia fiber decreases as the muscle contracts. (B) When both  $\alpha$  and  $\gamma$  motor neurons are activated, there is no decrease in Ia firing during muscle shortening. Thus, the  $\gamma$  motor neurons can regulate the gain of muscle spindles so they can operate efficiently at any length of the parent muscle. (After Hunt and Kuffler, 1951.)

compensate for the variable changes that occur as the bus stops and starts or progresses relatively smoothly. During voluntary movements,  $\alpha$  and  $\gamma$  motor neurons are often co-activated by higher centers to prevent muscle spindles from being unloaded (Figure 15.10).

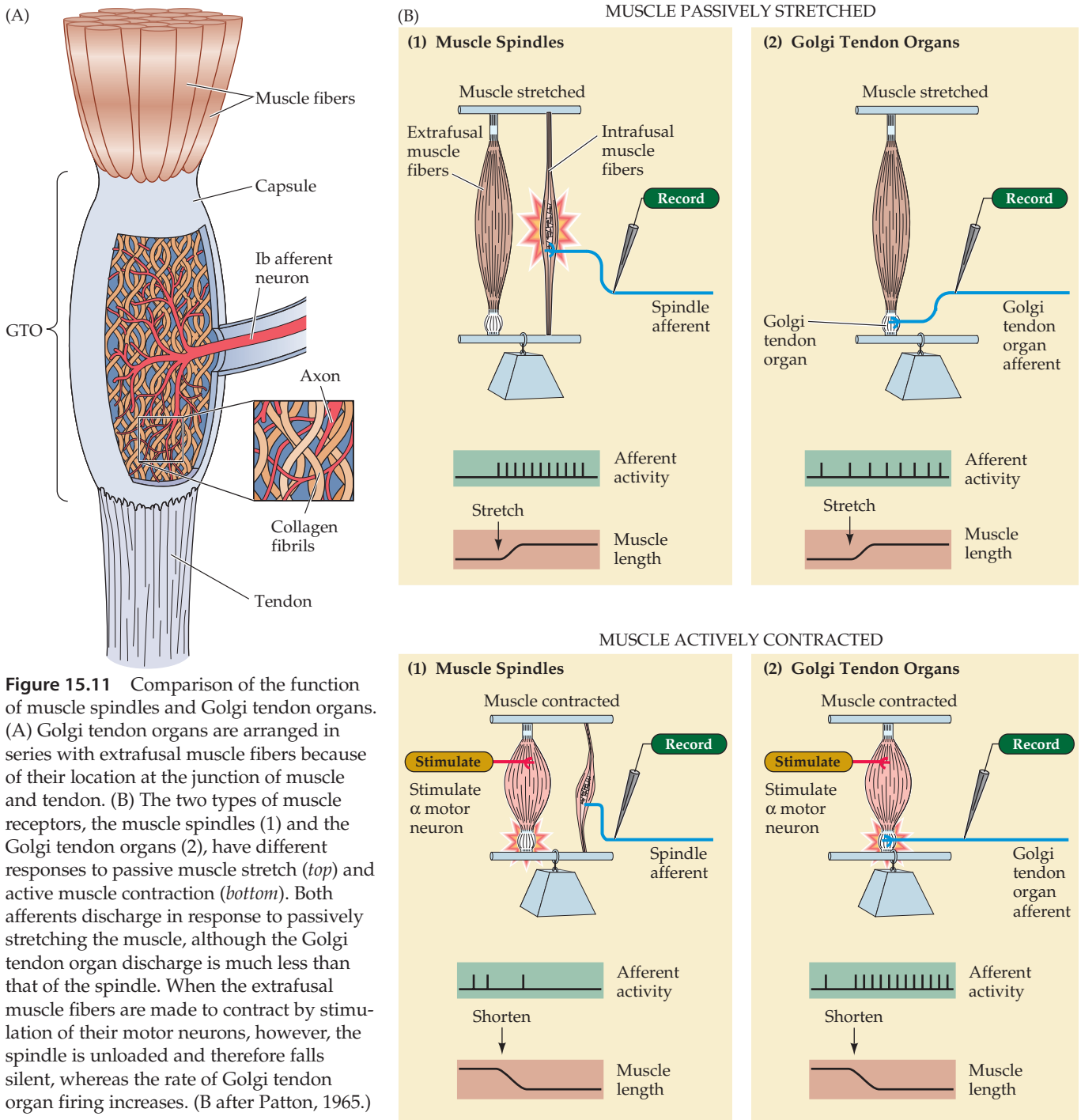
In addition, the level of  $\gamma$  motor neuron activity can be modulated independently of  $\alpha$  activity if the context of a movement requires it. In general, the baseline activity level of  $\gamma$  motor neurons is high if a movement is relatively difficult and demands rapid and precise execution. For example, recordings from cat hindlimb muscles show that  $\gamma$  activity is high when the animal has to perform a difficult movement such as walking across a narrow beam. Unpredictable conditions, as when the animal is picked up or handled, also lead to marked increases in  $\gamma$  activity and greatly increased spindle responsiveness.

Gamma motor neuron activity, however, is not the only factor that sets the gain of the stretch reflex. The gain also depends on the level of excitability of the  $\alpha$  motor neurons that serve as the efferent side of this reflex loop. Thus, in addition to the influence of descending upper motor neuron projections, other local circuits in the spinal cord can change the gain of the stretch reflex by excitation or inhibition of either  $\alpha$  or  $\gamma$  motor neurons.

### Other Sensory Feedback That Affects Motor Performance

Another sensory receptor that is important in the reflex regulation of motor unit activity is the **Golgi tendon organ**. Golgi tendon organs are encapsu-

lated afferent nerve endings located at the junction of a muscle and tendon (Figure 15.11A; see also Table 9.1). Each tendon organ is innervated by a single group Ib sensory axon (the Ib axons are slightly smaller than the Ia axons that innervate the muscle spindles). In contrast to the parallel arrangement of extrafusal muscle fibers and spindles, Golgi tendon organs are in series with the extrafusal muscle fibers. When a muscle is passively stretched, most of the change in length occurs in the muscle fibers, since they are more elas-



**Figure 15.11** Comparison of the function of muscle spindles and Golgi tendon organs. (A) Golgi tendon organs are arranged in series with extrafusal muscle fibers because of their location at the junction of muscle and tendon. (B) The two types of muscle receptors, the muscle spindles (1) and the Golgi tendon organs (2), have different responses to passive muscle stretch (*top*) and active muscle contraction (*bottom*). Both afferents discharge in response to passively stretching the muscle, although the Golgi tendon organ discharge is much less than that of the spindle. When the extrafusal muscle fibers are made to contract by stimulation of their motor neurons, however, the spindle is unloaded and therefore falls silent, whereas the rate of Golgi tendon organ firing increases. (B after Patton, 1965.)

## Box A

### Locomotion in the Leech and the Lamprey

All animals must coordinate body movements so they can navigate successfully in their environment. All vertebrates, including mammals, use local circuits in the spinal cord (central pattern generators) to control the coordinated movements associated with locomotion. The cellular basis of organized locomotor activity, however, has been most thoroughly studied in an invertebrate, the leech, and a simple vertebrate, the lamprey.

Both the leech and the lamprey lack peripheral appendages for locomotion possessed by many vertebrates (limbs, flippers, fins, or their equivalent). Furthermore, their bodies comprise repeating muscle segments (as well as repeating skeletal elements in the lamprey). Thus, in order to move through the water, both animals must coordinate the movement of each segment. They do this by orchestrating a sinusoidal displace-

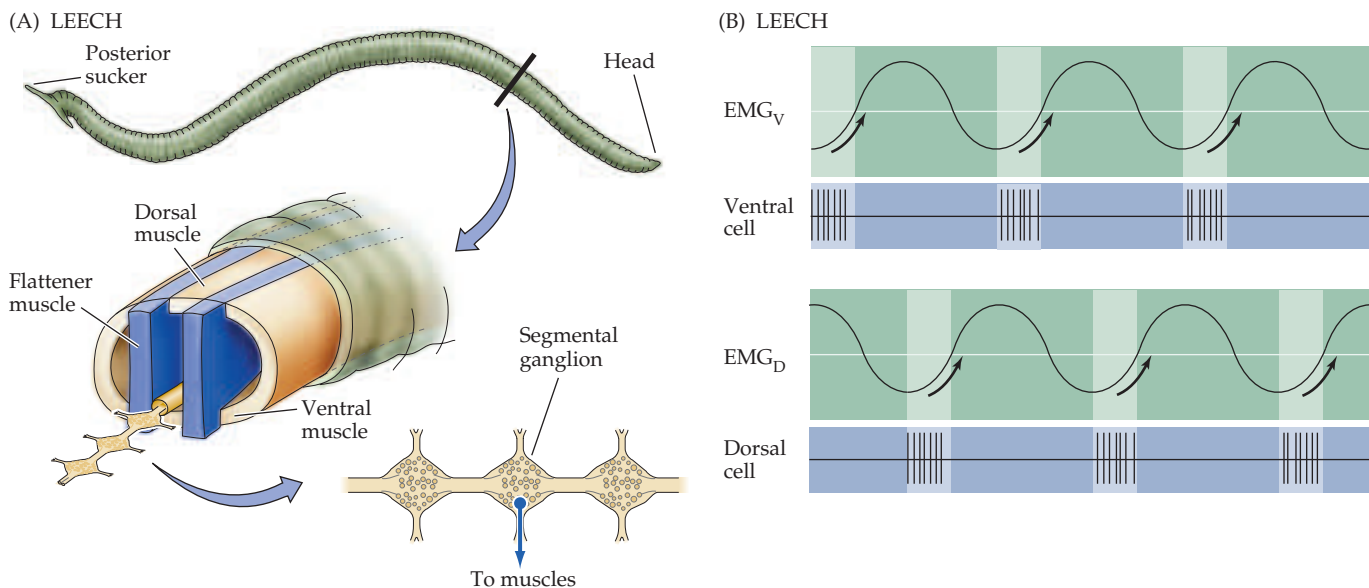
ment of each body segment in sequence, so that the animal is propelled forward through the water.

The leech is particularly well-suited for studying the circuit basis of coordinated movement. The nervous system in the leech consists of a series of interconnected segmental ganglia, each with motor neurons that innervate the corresponding segmental muscles (Figure A). These segmental ganglia facilitate electrophysiological studies, because there is a limited number of neurons in each and each neuron has a distinct identity. The neurons can thus be recognized and studied from animal to animal, and their electrical activity correlated with the sinusoidal swimming movements.

A central pattern generator circuit coordinates this undulating motion. In the leech, the relevant neural circuit is an ensemble of sensory neurons, interneurons, and motor neurons repeated in each

segmental ganglion that controls the local sequence of contraction and relaxation in each segment of the body wall musculature (Figure B). The sensory neurons detect the stretching and contraction of the body wall associated with the sequential swimming movements. Dorsal and ventral motor neurons in the circuit provide innervation to dorsal and ventral muscles, whose phasic contractions propel the leech forward. Sensory information and motor neuron signals are coordinated by interneurons that fire rhythmically, setting up phasic patterns of activity in the dorsal and ventral cells that lead to sinusoidal movement. The intrinsic swimming rhythm is established by a variety of membrane conductances that mediate periodic bursts of supra-threshold action potentials followed by well-defined periods of hyperpolarization.

The lamprey, one of the simplest vertebrates, is distinguished by its clearly



(A) The leech propels itself through the water by sequential contraction and relaxation of the body wall musculature of each segment. The segmental ganglia in the ventral midline coordinate swimming, each ganglion containing a population of identified neurons. (B) Electrical recordings from the ventral (EMG<sub>V</sub>) and dorsal (EMG<sub>D</sub>) muscles in the leech and the corresponding motor neurons show a reciprocal pattern of excitation for the dorsal and ventral muscles of a given segment.

segmented musculature and by its lack of bilateral fins or other appendages. In order to move through the water, the lamprey contracts and relaxes each muscle segment in sequence (Figure C), which produces a sinusoidal motion, much like that of the leech. Again, a central pattern generator coordinates this sinusoidal movement.

Unlike the leech with its segmental ganglia, the lamprey has a continuous spinal cord that innervates its muscle segments. The lamprey spinal cord is simpler than that of other vertebrates, and several classes of identified neurons occupy stereotyped positions. This orderly arrangement again facilitates the identification and analysis of neurons that constitute the central pattern generator circuit.

In the lamprey spinal cord, the intrinsic firing pattern of a set of interconnected sensory neurons, interneurons and motor neurons establishes the pattern of undulating muscle contractions that underlie swimming (Figure D). The patterns of connectivity between neurons, the neurotransmitters used by each class of cell, and the physiological properties of the elements in the lamprey pattern generator are now known. Different neurons in the circuit fire with distinct rhythmicity, thus controlling specific aspects of the swim cycle (Figure E). Particularly important are reciprocal inhibitory connections across the midline that coordinate the pattern generating circuitry on each side of the spinal cord. This circuitry in the lamprey thus provides a basis for understanding the cir-

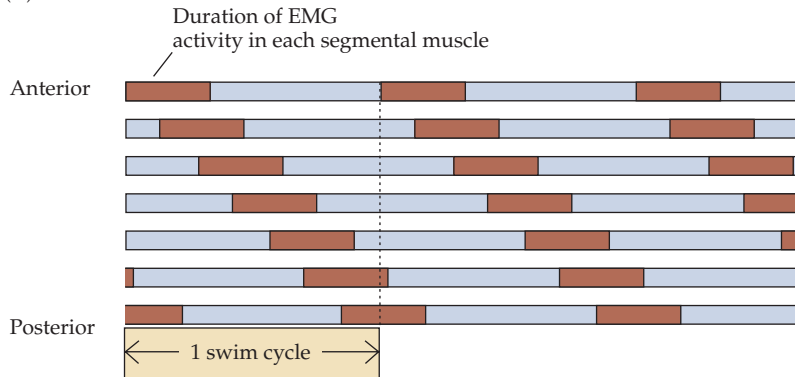
cuits that control locomotion in more complex vertebrates.

These observations on pattern generating circuits for locomotion in relatively simple animals have stimulated parallel studies of terrestrial mammals in which central pattern generators in the spinal cord also coordinate locomotion. Although different in detail, terrestrial locomotion ultimately relies on the sequential movements similar to those that propel the leech and the lamprey through aquatic environments, and intrinsic physiological properties of spinal cord neurons that establish rhythmicity for coordinated movement.

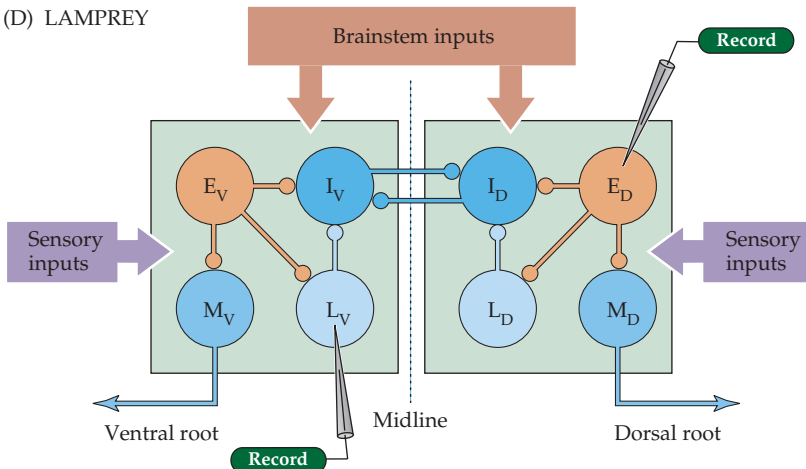
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(C) LAMPREY

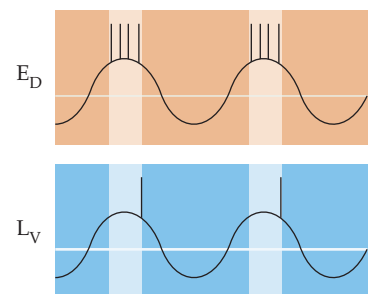


(D) LAMPREY



(C) In the lamprey, as this diagram indicates, the pattern of activity across segments is also highly coordinated. (D) The elements of the central pattern generator in the lamprey have been worked out in detail, providing a guide to understanding homologous circuitry in more complex spinal cords. (E) As in the leech, different patterns of electrical activity in lamprey spinal neurons (neurons  $E_D$  and  $L_V$  in this example) correspond to distinct periods in the sequence of muscle contractions related to the swim cycle.

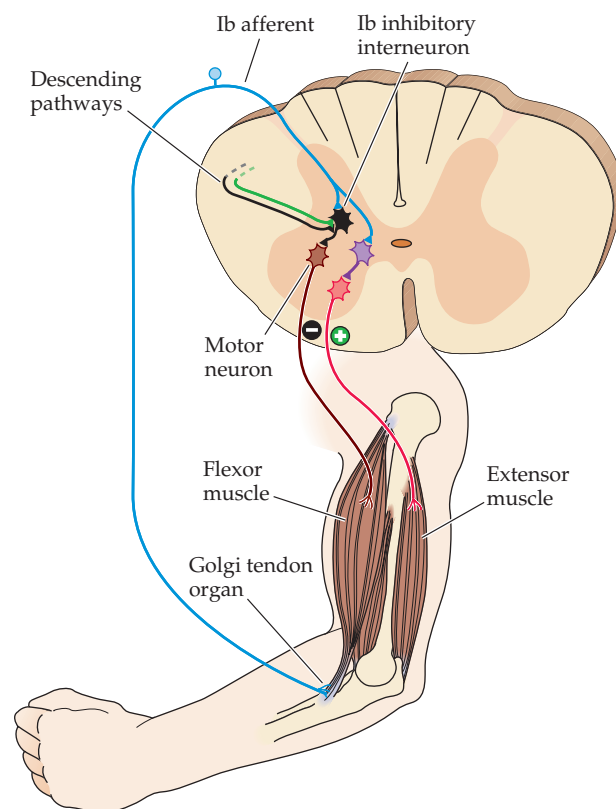
(E) LAMPREY



tic than the fibrils of the tendon. When a muscle actively contracts, however, the force acts directly on the tendon, leading to an increase in the tension of the collagen fibrils in the tendon organ and compression of the intertwined sensory receptors. As a result, Golgi tendon organs are exquisitely sensitive to increases in muscle *tension* that arise from muscle contraction but, unlike spindles, are relatively insensitive to *passive stretch* (Figure 15.11B).

The Ib axons from Golgi tendon organs contact inhibitory local circuit neurons in the spinal cord (called Ib inhibitory interneurons) that synapse, in turn, with the  $\alpha$  motor neurons that innervate the same muscle. The Golgi tendon circuit is thus a negative feedback system that regulates muscle tension; it decreases the activation of a muscle when exceptionally large forces are generated and this way protects the muscle. This reflex circuit also operates at reduced levels of muscle force, counteracting small changes in muscle tension by increasing or decreasing the inhibition of  $\alpha$  motor neurons. Under these conditions, the Golgi tendon system tends to maintain a steady level of force, counteracting effects that diminish muscle force (such as fatigue). In short, the muscle spindle system is a feedback system that monitors and maintains muscle *length*, and the Golgi tendon system is a feedback system that monitors and maintains muscle *force*.

Like the muscle spindle system, the Golgi tendon organ system is not a closed loop. The Ib inhibitory interneurons also receive synaptic inputs from a variety of other sources, including cutaneous receptors, joint receptors, muscle spindles, and descending upper motor neuron pathways (Figure 15.12). Acting in concert, these inputs regulate the responsiveness of Ib interneurons to activity arising in Golgi tendon organs.



**Figure 15.12** Negative feedback regulation of muscle tension by Golgi tendon organs. The Ib afferents from tendon organs contact inhibitory interneurons that decrease the activity of  $\alpha$  motor neurons innervating the same muscle. The Ib inhibitory interneurons also receive input from other sensory fibers, as well as from descending pathways. This arrangement prevents muscles from generating excessive tension.



## Flexion Reflex Pathways

So far, the discussion has focused on reflexes driven by sensory receptors located within muscles or tendons. Other reflex circuitry mediates the withdrawal of a limb from a painful stimulus, such as a pinprick or the heat of a flame. Contrary to what might be imagined given the speed with which we are able to withdraw from a painful stimulus, this **flexion reflex** involves several synaptic links (Figure 15.13). As a result of activity in this circuitry, stimulation of nociceptive sensory fibers leads to withdrawal of the limb from the source of pain by excitation of ipsilateral flexor muscles and reciprocal inhibition of ipsilateral extensor muscles. Flexion of the stimulated limb is also accompanied by an opposite reaction in the contralateral limb (i.e., the contralateral extensor muscles are excited while flexor muscles are inhibited). This **crossed extension reflex** provides postural support during withdrawal of the affected limb from the painful stimulus.

Like the other reflex pathways, local circuit neurons in the flexion reflex pathway receive converging inputs from several different sources, including other spinal cord interneurons and upper motor neuron pathways. Although the functional significance of this complex pattern of connectivity is unclear, changes in the character of the reflex following damage to descending pathways provides some insight. Under normal conditions, a noxious stimulus is required to evoke the flexion reflex; following damage to descending pathways, however, other types of stimulation, such as squeezing a limb, can sometimes produce the same response. This observation suggests that the descending projections to the spinal cord modulate the responsiveness of the local circuitry to a variety of sensory inputs.

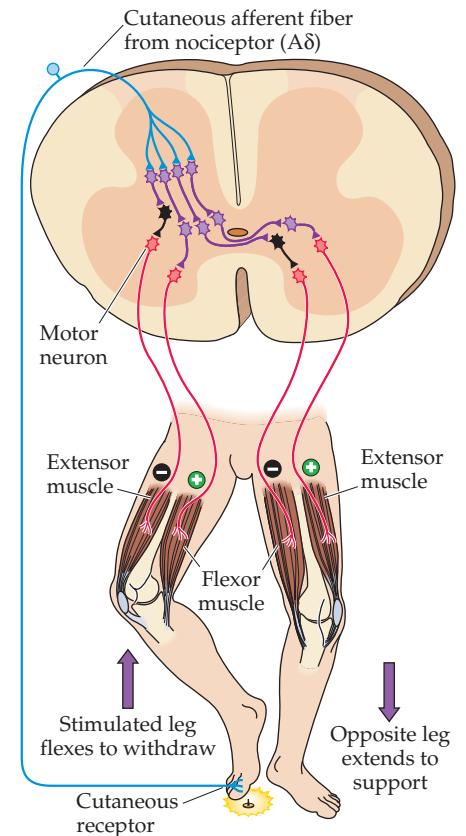
## Spinal Cord Circuitry and Locomotion

The contribution of local circuitry to motor control is not, of course, limited to reflexive responses to sensory inputs. Studies of rhythmic movements such as locomotion and swimming in animal models (Box A) have demonstrated that local circuits in the spinal cord called **central pattern generators** are fully capable of controlling the timing and coordination of such complex patterns of movement, and of adjusting them in response to altered circumstances (Box B).

A good example is locomotion (walking, running, etc.). The movement of a single limb during locomotion can be thought of as a cycle consisting of two phases: a *stance phase*, during which the limb is extended and placed in contact with the ground to propel humans or other bipeds forward; and a *swing phase*, during which the limb is flexed to leave the ground and then brought forward to begin the next stance phase (Figure 15.14A). Increases in the speed of locomotion reduce the amount of time it takes to complete a cycle, and most of the change in cycle time is due to shortening the stance phase; the swing phase remains relatively constant over a wide range of locomotor speeds.

In quadrupeds, changes in locomotor speed are also accompanied by changes in the sequence of limb movements. At low speeds, for example, there is a back-to-front progression of leg movements, first on one side and then on the other. As the speed increases to a trot, the movements of the right forelimb and left hindlimb are synchronized (as are the movements of the left forelimb and right hindlimb). At the highest speeds (gallop), the movements of the two front legs are synchronized, as are the movements of the two hindlimbs (Figure 15.14B).

Given the precise timing of the movement of individual limbs and the coordination among limbs that are required in this process, it is natural to



**Figure 15.13** Spinal cord circuitry responsible for the flexion reflex. Stimulation of cutaneous receptors in the foot (by stepping on a tack in this example) leads to activation of spinal cord local circuits that withdraw (flex) the stimulated extremity and extend the other extremity to provide compensatory support.

## Box B

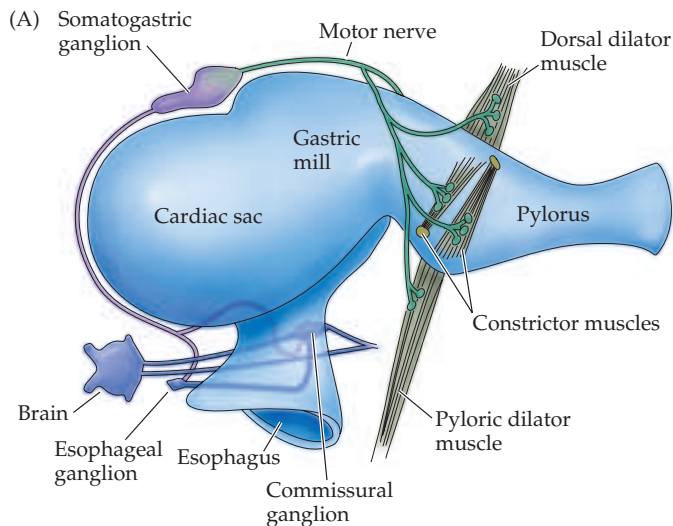
### The Autonomy of Central Pattern Generators: Evidence from the Lobster Stomatogastric Ganglion

A principle that has emerged from studies of central pattern generators is that rhythmic patterns of firing elicit complex motor responses without need of ongoing sensory stimulation. A good example is the behavior mediated by a small group of nerve cells in lobsters and other crustaceans called the stomatogastric ganglion (STG) that controls the muscles of the gut (Figure A). This ensemble of 30 motor neurons and interneurons in the lobster is perhaps the most completely characterized neural circuit known. Of the 30 cells, defined subsets are essential for two distinct rhythmic movements: gastric mill movements that mediate grinding of food by “teeth” in the lobster’s foregut, and pyloric movements that propel food into the hindgut. Phasic firing patterns of the motor neurons and interneurons of the STG are directly correlated with these two rhythmic move-

ments. Each of the relevant cells has now been identified based on its position in the ganglion, and its electrophysiological and neuropharmacological properties characterized (Figures B and C).

Patterned activity in the motor neurons and interneurons of the ganglion begins only if the appropriate neuromodulatory input is provided by sensory axons that originate in other ganglia. Depending upon the activity of the sensory axons, neuronal ensembles in the STG produce one of several characteristic rhythmic firing patterns. Once activated, however, the intrinsic membrane properties of identified cells within the ensemble sustain the rhythmicity of the circuit in the absence of further sensory input.

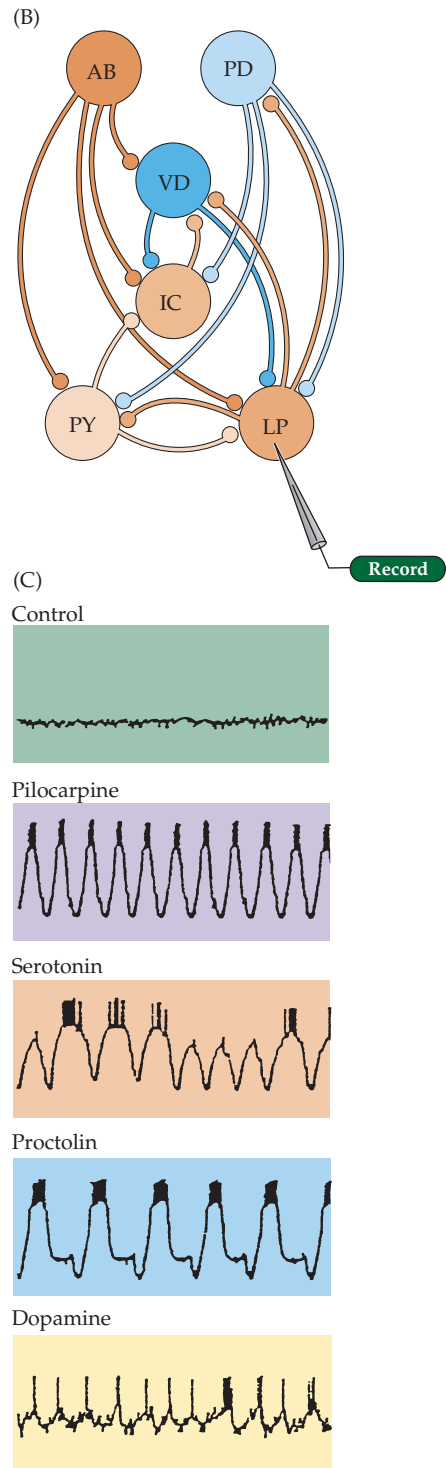
Another key fact that has emerged from this work is that the same neurons can participate in different programmed motor activities, as circumstances



(A) Location of the lobster stomatogastric ganglion in relation to the gut.

(B) Subset of identified neurons in the stomatogastric ganglion that generates gastric mill and pyloric activity. The abbreviations indicate individual identified neurons, all of which project to different pyloric muscles (except the AB neuron, which is an interneuron).

(C) Recording from one of the neurons, the lateral pyloric or LP neuron, in this circuit showing the different patterns of activity elicited by several neuromodulators known to be involved in the normal synaptic interactions in this ganglion.



demand. For example, the subset of neurons producing gastric mill activity overlaps the subset that generates pyloric activity. This economic use of neuronal subsets has not yet been described in the central pattern generators of mammals, but seems likely to be a feature of all such circuits.

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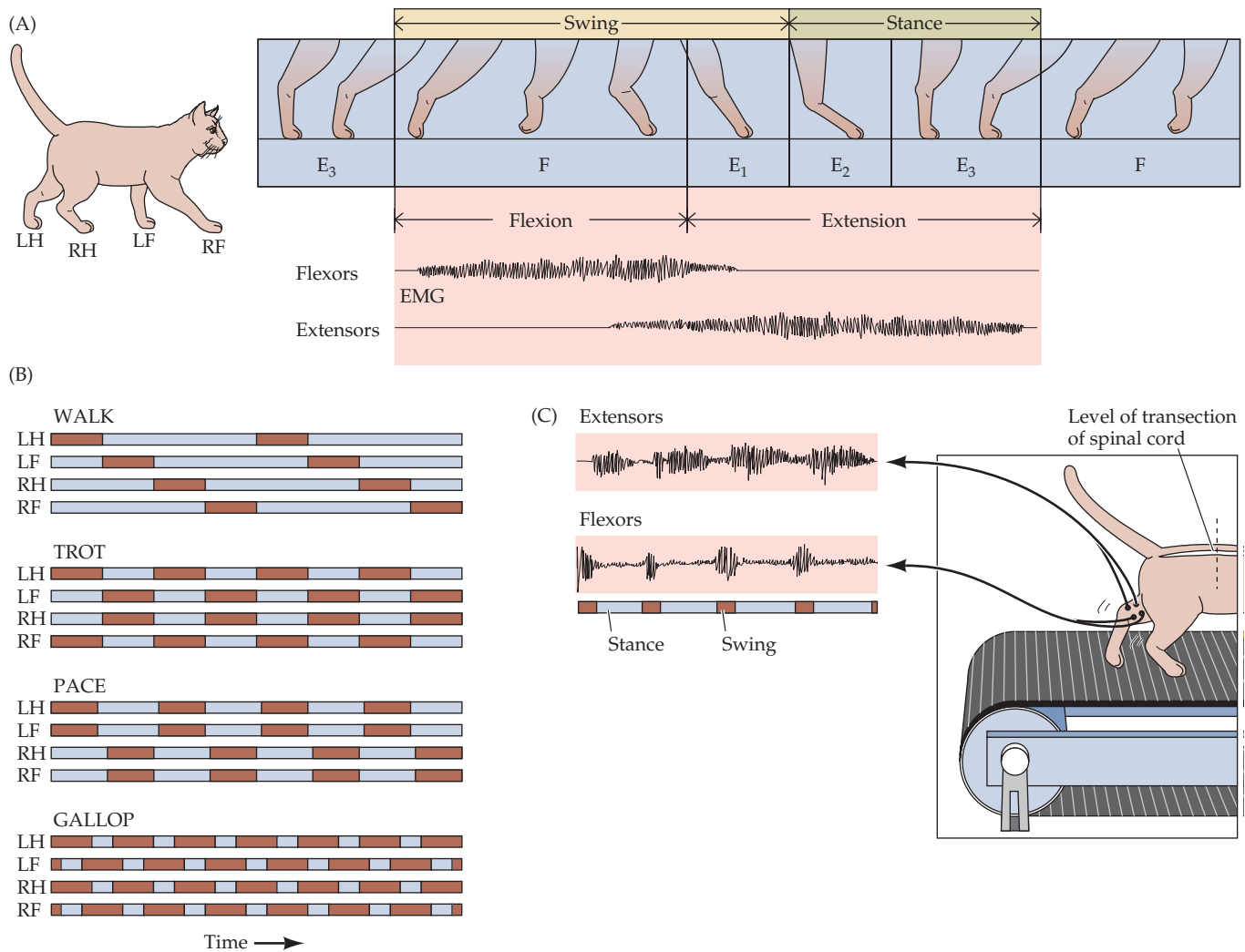
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assume that locomotion is accomplished by higher centers that organize the spatial and temporal activity patterns of the individual limbs. However, following transection of the spinal cord at the thoracic level, a cat's hindlimbs will still make coordinated locomotor movements if the animal is supported and placed on a moving treadmill (Figure 15.14C). Under these conditions, the speed of locomotor movements is determined by the speed of the treadmill, suggesting that the movement is nothing more than a reflexive response to stretching the limb muscles. This possibility is ruled out, however, by experiments in which the dorsal roots are also sectioned. Although the speed of walking is slowed and the movements are less coordinated than under normal conditions, appropriate locomotor movements are still observed. These and other observations in experimental animals show that the basic rhythmic patterns of limb movement during locomotion are not dependent on sensory input; nor are they dependent on input from descending projections from higher centers. Rather, each limb appears to have its own central pattern generator responsible for the alternating flexion and extension of the limb during locomotion (see Box B). Under normal conditions, the central pattern generators for the limbs are variably coupled to each other by additional local circuits in order to achieve the different sequences of movements that occur at different speeds.

Although some locomotor movements can also be elicited in humans following damage to descending pathways, these are considerably less effective than the movements seen in the cat. The reduced ability of the transected spinal cord to mediate rhythmic stepping movements in humans presumably reflects an increased dependence of local circuitry on upper motor neuron pathways. Perhaps bipedal locomotion carries with it requirements for postural control greater than can be accommodated by spinal cord circuitry alone. Whatever the explanation, the basic oscillatory circuits that control such rhythmic behaviors as flying, walking, and swimming in many animals also play an important part in human locomotion.

## The Lower Motor Neuron Syndrome

The complex of signs and symptoms that arise from damage to the lower motor neurons of the brainstem and spinal cord is referred to as the "lower motor neuron syndrome." In clinical neurology, this constellation of problems must be distinguished from the "upper motor neuron syndrome" that results from damage to the descending upper motor neuron pathways (see Chapter 16 for a discussion of the signs and symptoms associated with damage to upper motor neurons).



**Figure 15.14** The cycle of locomotion for terrestrial mammals (a cat in this instance) is organized by central pattern generators. (A) The step cycle, showing leg flexion (F) and extension (E) and their relation to the swing and stance phases of locomotion. EMG indicates electromyographic recordings. (B) Comparison of the stepping movements for different gaits. Brown bars, foot lifted (swing phase); gray bars, foot planted (stance phase). (C) Transection of the spinal cord at the thoracic level isolates the hindlimb segments of the cord. The hindlimbs are still able to walk on a treadmill after recovery from surgery, and reciprocal bursts of electrical activity can be recorded from flexors during the swing phase and from extensors during the stance phase of walking. (After Pearson, 1976.)

Damage to lower motor neuron cell bodies or their peripheral axons results in paralysis (loss of movement) or paresis (weakness) of the affected muscles, depending on the extent of the damage. In addition to paralysis and/or paresis, the lower motor neuron syndrome includes a loss of reflexes (areflexia) due to interruption of the efferent (motor) limb of the sensory motor reflex arcs. Damage to lower motor neurons also entails a loss of muscle tone, since tone is in part dependent on the monosynaptic reflex arc that links the muscle spindles to the lower motor neurons (see also Box D in Chapter 16). A somewhat later effect is atrophy of the affected muscles due to denervation and disuse. The muscles involved may also exhibit fibrillations and fasciculations, which are spontaneous twitches characteristic of single denervated muscle fibers or motor units, respectively. These phenomena arise from changes in the excitability of denervated muscle fibers in the case of fibrillation, and from abnormal activity of injured  $\alpha$  motor neurons in the case of fasciculations. These spontaneous contractions can be readily recognized in an electromyogram, providing an especially helpful clinical tool in diagnosing lower motor neuron disorders (Box C).

## Box C

### Amyotrophic Lateral Sclerosis

Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disease that affects an estimated 0.05% of the population in the United States. It is also called Lou Gehrig's disease, after the New York Yankees baseball player who died of the disorder in 1936. ALS is characterized by the slow but inexorable degeneration of  $\alpha$  motor neurons in the ventral horn of the spinal cord and brainstem (*lower* motor neurons), and of neurons in the motor cortex (*upper* motor neurons). Affected individuals show progressive weakness due to upper and/or lower motor neuron involvement, wasting of skeletal muscles due to lower motor neuron involvement, and usually die within 5 years of onset. Sadly, these patients are condemned to watch their own demise, since the intellect remains intact. No available therapy effectively prevents the inexorable progression of this disease.

Approximately 10% of ALS cases are familial, and several distinct familial forms have been identified. An autosomal dominant form of familial ALS (FALS) is caused by mutations of the

gene that encodes the cytosolic antioxidant enzyme copper/zinc superoxide dismutase (SOD1). Mutations of *SOD1* account for roughly 20% of families with FALS. A rare autosomal recessive, juvenile-onset form is caused by mutations in a protein called alsin, a putative GTPase regulator. Another rare type of FALS consists of a slowly progressive, autosomal dominant, lower motor neuron disease without sensory symptoms, with onset in early adulthood; this form is caused by mutations of a protein named dynactin.

How these mutant genes lead to the phenotype of motor neuron disease is uncertain. Defects of axonal transport have long been hypothesized to cause ALS. Evidence for this cause is that transgenic mice with mutant SOD1 exhibit defects in slow axonal transport early in the course of the disease, and that dynactin binds to microtubules and thus that mutant dynactin may modify axonal transport along microtubules. However, whether defective axonal transport is the cellular mechanism by which these

mutant proteins lead to motor neuron disease remains to be clearly established. Despite these uncertainties, demonstration that mutations of each of these three genes can cause familial ALS has given scientists valuable clues about the molecular pathogenesis of at least some forms of this tragic disorder.

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#### Summary

Four distinct but highly interactive motor subsystems—local circuits in the spinal cord and brainstem, descending upper motor neuron pathways that control these circuits, the basal ganglia, and the cerebellum—all make essential contributions to motor control. Alpha motor neurons located in the spinal cord and in the cranial nerve nuclei in the brainstem directly link the nervous system and muscles, with each motor neuron and its associated muscle fibers constituting a functional entity called the motor unit. Motor units vary in size, amount of tension produced, speed of contraction, and degree of fatigability. Graded increases in muscle tension are mediated by both the orderly recruitment of different types of motor units and an increase in motor neuron firing frequency. Local circuitry involving sensory inputs, local circuit neurons, and  $\alpha$  and  $\gamma$  motor neurons are especially important in the reflexive control of muscle activity. The stretch reflex is a monosynaptic circuit with connections between sensory fibers arising from muscle spindles and the  $\alpha$  motor neurons that innervate the same or syner-

gistic muscles. Gamma motor neurons regulate the gain of the stretch reflex by adjusting the level of tension in the intrafusal muscle fibers of the muscle spindle. This mechanism sets the baseline level of activity in  $\alpha$  motor neurons and helps to regulate muscle length and tone. Other reflex circuits provide feedback control of muscle tension and mediate essential functions such as the rapid withdrawal of limbs from painful stimuli. Much of the spatial coordination and timing of muscle activation required for complex rhythmic movements such as locomotion are provided by specialized local circuits called central pattern generators. Because of their essential role in all of these circuits, damage to lower motor neurons leads to paralysis of the associated muscle and to other changes, including the loss of reflex activity, the loss of muscle tone, and eventually muscle atrophy.

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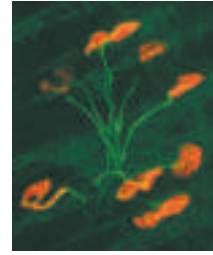
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# Chapter 19

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## Eye Movements and Sensory Motor Integration

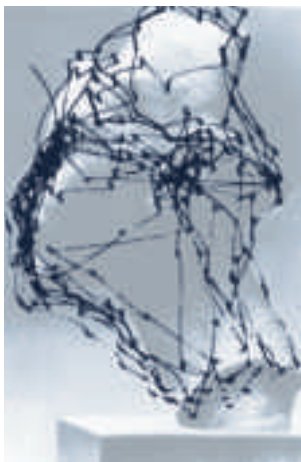
### Overview

Eye movements are, in many ways, easier to study than movements of other parts of the body. This fact arises from the relative simplicity of muscle actions on the eyeball. There are only six extraocular muscles, each of which has a specific role in adjusting eye position. Moreover, there are only four stereotyped kinds of eye movements, each with its own control circuitry. Eye movements have therefore been a useful model for understanding the mechanisms of motor control. Indeed, much of what is known about the regulation of movements by the cerebellum, basal ganglia, and vestibular system has come from the study of eye movements (see Chapters 13, 17, and 18). Here the major features of eye movement control are used to illustrate the principles of sensory motor integration that also apply to more complex motor behaviors.

### What Eye Movements Accomplish

Eye movements are important in humans because high visual acuity is restricted to the fovea, the small circular region (about 1.5 mm in diameter) in the central retina that is densely packed with cone photoreceptors (see Chapter 10). Eye movements can direct the fovea to new objects of interest (a process called “foveation”) or compensate for disturbances that cause the fovea to be displaced from a target already being attended to.

As demonstrated several decades ago by the Russian physiologist Alfred Yarbus, eye movements reveal a good deal about the strategies used to inspect a scene. Yarbus used contact lenses with small mirrors on them (see Box A) to document (by the position of a reflected beam) the pattern of eye movements made while subjects examined a variety of objects and scenes. Figure 19.1 shows the direction of a subject’s gaze while viewing a picture of Queen Nefertiti. The thin, straight lines represent the quick, ballistic eye movements (saccades) used to align the foveas with particular parts of the scene; the denser spots along these lines represent points of fixation where the observer paused for a variable period to take in visual information (little or no visual perception occurs during a saccade, which occupies only a few tens of milliseconds). The results obtained by Yarbus, and subsequently many others, showed that vision is an active process in which eye movements typically shift the view several times each second to selected parts of the scene to examine especially interesting features. The spatial distribution of the fixation points indicates that much more time is spent scrutinizing Nefertiti’s eye, nose, mouth, and ear than examining the middle of her cheek or neck. Thus, eye movements allow us to scan the visual field, pausing to focus attention on the portions of the scene that convey the most significant



**Figure 19.1** The eye movements of a subject viewing a picture of Queen Nefertiti. The bust at the top is what the subject saw; the diagram on the bottom shows the subject's eye movements over a 2-minute viewing period. (From Yarbus, 1967.)

information. As is apparent in Figure 19.1, tracking eye movements can be used to determine what aspects of a scene are particularly arresting. Advertisers now use modern versions of Yarbus' method to determine which pictures and scene arrangements will best sell their product.

The importance of eye movements for visual perception has also been demonstrated by experiments in which a visual image is stabilized on the retina, either by paralyzing the extraocular eye muscles or by moving a scene in exact register with eye movements so that the different features of the image always fall on exactly the same parts of the retina (Box A). Stabilized visual images rapidly disappear, for reasons that remain poorly understood. Nonetheless, these observations on motionless images make it plain that eye movements are also essential for normal visual perception.

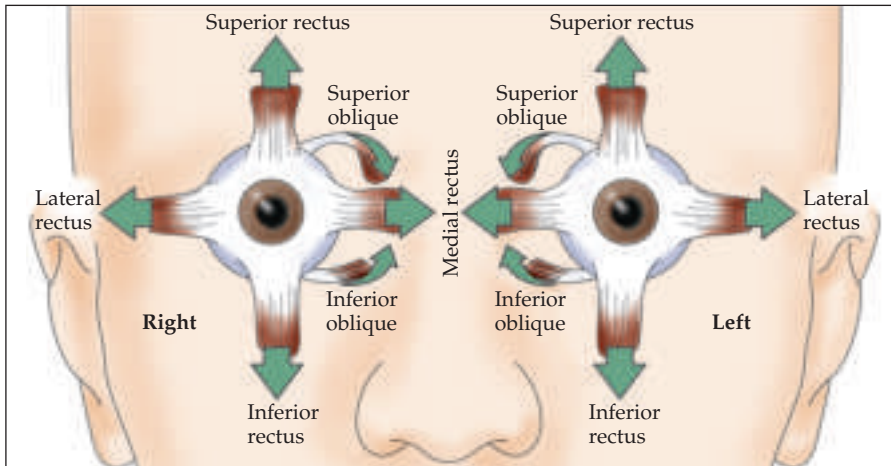
### The Actions and Innervation of Extraocular Muscles

Three antagonistic pairs of muscles control eye movements: the **lateral** and **medial rectus muscles**, the **superior** and **inferior rectus muscles**, and the **superior** and **inferior oblique muscles**. These muscles are responsible for movements of the eye along three different axes: *horizontal*, either toward the nose (adduction) or away from the nose (abduction); *vertical*, either elevation or depression; and *torsional*, movements that bring the top of the eye toward the nose (intorsion) or away from the nose (extorsion). Horizontal movements are controlled entirely by the medial and lateral rectus muscles; the medial rectus muscle is responsible for adduction, the lateral rectus muscle for abduction. Vertical movements require the coordinated action of the superior and inferior rectus muscles, as well as the oblique muscles. The relative contribution of the rectus and oblique groups depends on the horizontal position of the eye (Figure 19.2). In the primary position (eyes straight ahead), both of these groups contribute to vertical movements. Elevation is due to the action of the superior rectus and inferior oblique muscles, while depression is due to the action of the inferior rectus and superior oblique muscles. When the eye is abducted, the rectus muscles are the prime vertical movers. Elevation is due to the action of the superior rectus, and depression is due to the action of the inferior rectus. When the eye is adducted, the oblique muscles are the prime vertical movers. Elevation is due to the action of the inferior oblique muscle, while depression is due to the action of the superior oblique muscle. The oblique muscles are also primarily responsible for torsional movements.

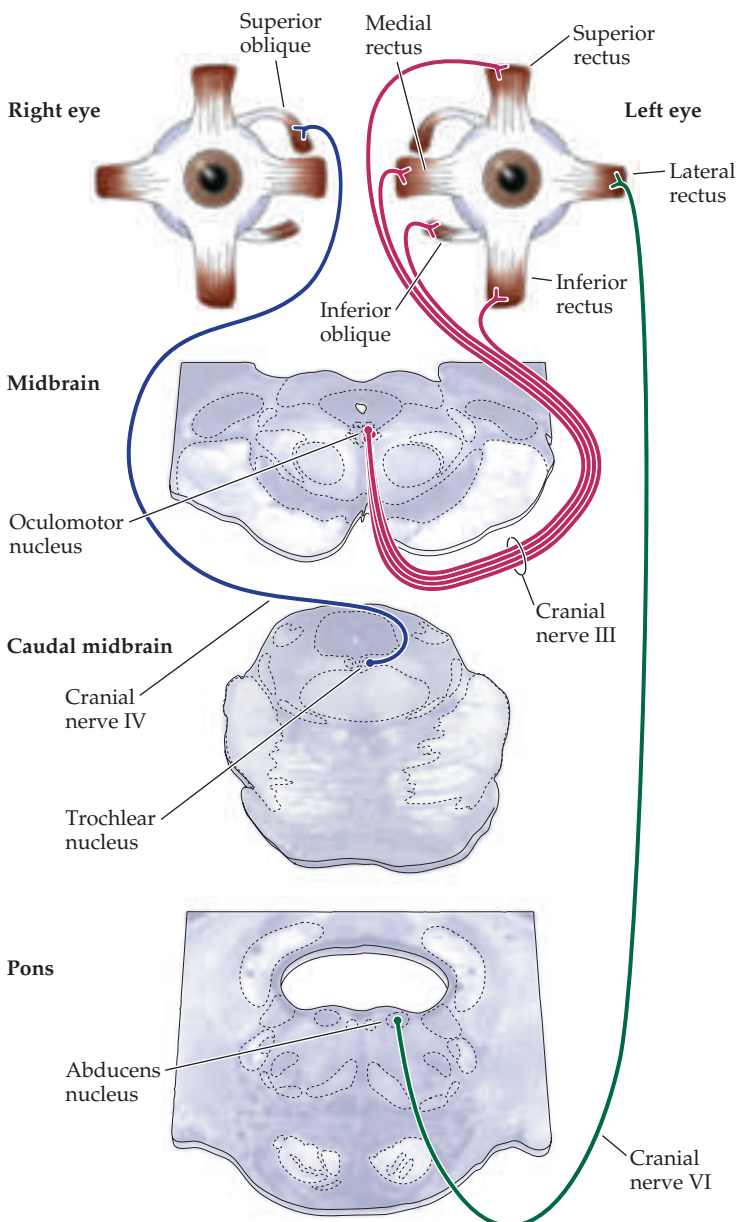
The extraocular muscles are innervated by lower motor neurons that form three cranial nerves: the abducens, the trochlear, and the oculomotor (Figure 19.3). The **abducens nerve** (cranial nerve VI) exits the brainstem from the pons–medullary junction and innervates the lateral rectus muscle. The **trochlear nerve** (cranial nerve IV) exits from the caudal portion of the midbrain and supplies the superior oblique muscle. In distinction to all other cranial nerves, the trochlear nerve exits from the dorsal surface of the brainstem and crosses the midline to innervate the superior oblique muscle on the contralateral side. The **oculomotor nerve** (cranial nerve III), which exits from the rostral midbrain near the cerebral peduncle, supplies all the rest of the extraocular muscles. Although the oculomotor nerve governs several different muscles, each receives its innervation from a separate group of lower motor neurons within the third nerve nucleus.

In addition to supplying the extraocular muscles, a distinct cell group within the oculomotor nucleus innervates the levator muscles of the eyelid; the axons from these neurons also travel in the third nerve. Finally, the third





**Figure 19.2** The contributions of the six extraocular muscles to vertical and horizontal eye movements. Horizontal movements are mediated by the medial and lateral rectus muscles, while vertical movements are mediated by the superior and inferior rectus and the superior and inferior oblique muscle groups.



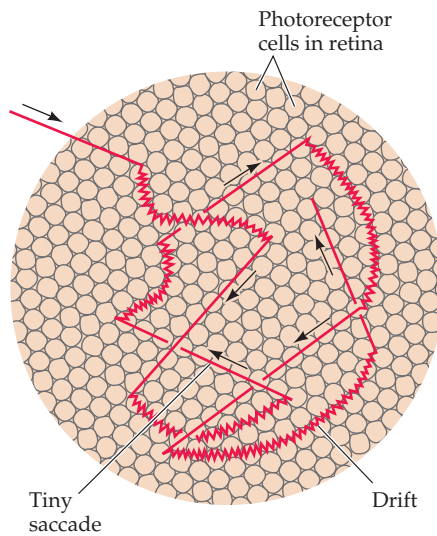
**Figure 19.3** Organization of the cranial nerve nuclei that govern eye movements, showing their innervation of the extraocular muscles. The abducens nucleus innervates the lateral rectus muscle; the trochlear nucleus innervates the superior oblique muscle; and the oculomotor nucleus innervates all the rest of the extraocular muscles (the medial rectus, inferior rectus, superior rectus, and inferior oblique).

## Box A

### The Perception of Stabilized Retinal Images

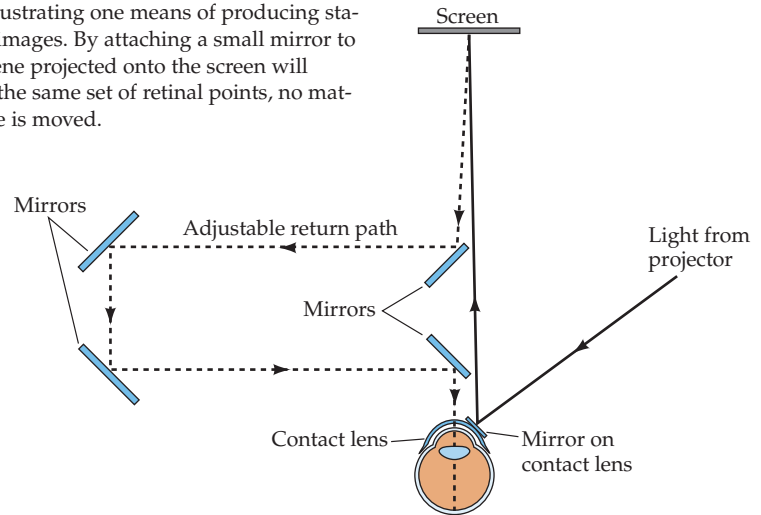
Visual perception depends critically on frequent changes of scene. Normally, our view of the world is changed by saccades, and tiny saccades that continue to move the eyes abruptly over a fraction of a degree of visual arc occur even when the observer stares intently at an object of interest. Moreover, continual drift of the eyes during fixation progressively shifts the image onto a nearby but different set of photoreceptors. As a consequence of these several sorts of eye movements (Figure A), our point of view changes more or less continually.

The importance of a continually changing scene for normal vision is dramatically revealed when the retinal image is stabilized. If a small mirror is



A) Diagram of the types of eye movements that continually change the retinal stimulus during fixation. The straight lines indicate microsaccades and the curved lines drift; the structures in the background are photoreceptors drawn approximately to scale. The normal scanning movements of the eyes (saccades) are much too large to be shown here, but obviously contribute to the changes of view that we continually experience, as do slow tracking eye movements (although the fovea tracks a particular object, the scene nonetheless changes). (After Pritchard, 1961.)

(B) Diagram illustrating one means of producing stabilized retinal images. By attaching a small mirror to the eye, the scene projected onto the screen will always fall on the same set of retinal points, no matter how the eye is moved.



attached to the eye by means of a contact lens and an image reflected off the mirror onto a screen, then the subject necessarily sees the same thing, whatever the position of the eye: Every time the eye moves, the projected image moves exactly the same amount (Figure B). Under these circumstances, the stabilized image actually disappears from perception within a few seconds!

A simple way to demonstrate the rapid disappearance of a stabilized retinal image is to visualize one's own retinal blood vessels. The blood vessels, which lie in front of the photoreceptor layer, cast a shadow on the underlying receptors. Although normally invisible, the vascular shadows can be seen by moving a source of light across the eye, a phenomenon first noted by J. E. Purkinje more than 150 years ago. This perception can be elicited with an ordinary penlight pressed gently against the lateral side of the closed eyelid. When the light is wiggled vigorously, a rich network of black blood vessel shadows appears against an orange background. (The vessels appear black because they are shadows.) By starting and stopping the movement, it is readily apparent that the image of the

blood vessel shadows disappears within a fraction of a second after the light source is stilled.

The conventional interpretation of the rapid disappearance of stabilized images is retinal adaptation. In fact, the phenomenon is at least partly of central origin. Stabilizing the retinal image in one eye, for example, diminishes perception through the other eye, an effect known as interocular transfer. Although the explanation of these remarkable effects is not entirely clear, they emphasize the point that the visual system is designed to deal with novelty.

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nerve carries axons that are responsible for pupillary constriction (see Chapter 11) from the nearby Edinger-Westphal nucleus. Thus, damage to the third nerve results in three characteristic deficits: impairment of eye movements, drooping of the eyelid (ptosis), and pupillary dilation.

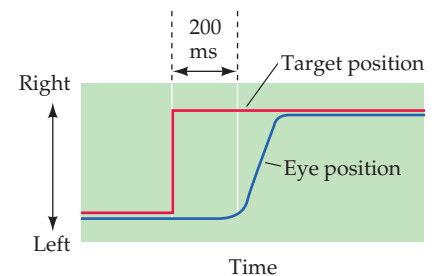
## Types of Eye Movements and Their Functions

There are four basic types of eye movements: saccades, smooth pursuit movements, vergence movements, and vestibulo-ocular movements. The functions of each type of eye movement are introduced here; in subsequent sections, the neural circuitry responsible for three of these types of movements is presented in more detail (see Chapters 13 and 18 for further discussion of neural circuitry underlying vestibulo-ocular movements).

**Saccades** are rapid, ballistic movements of the eyes that abruptly change the point of fixation. They range in amplitude from the small movements made while reading, for example, to the much larger movements made while gazing around a room. Saccades can be elicited voluntarily, but occur reflexively whenever the eyes are open, even when fixated on a target (see Box A). The rapid eye movements that occur during an important phase of sleep (see Chapter 27) are also saccades. The time course of a saccadic eye movement is shown in Figure 19.4. After the onset of a target for a saccade (in this example, the stimulus was the movement of an already fixated target), it takes about 200 milliseconds for eye movement to begin. During this delay, the position of the target with respect to the fovea is computed (that is, how far the eye has to move), and the difference between the initial and intended position, or “motor error” (see Chapter 18), is converted into a motor command that activates the extraocular muscles to move the eyes the correct distance in the appropriate direction. Saccadic eye movements are said to be ballistic because the saccade-generating system cannot respond to subsequent changes in the position of the target during the course of the eye movement. If the target moves again during this time (which is on the order of 15–100 ms), the saccade will miss the target, and a second saccade must be made to correct the error.

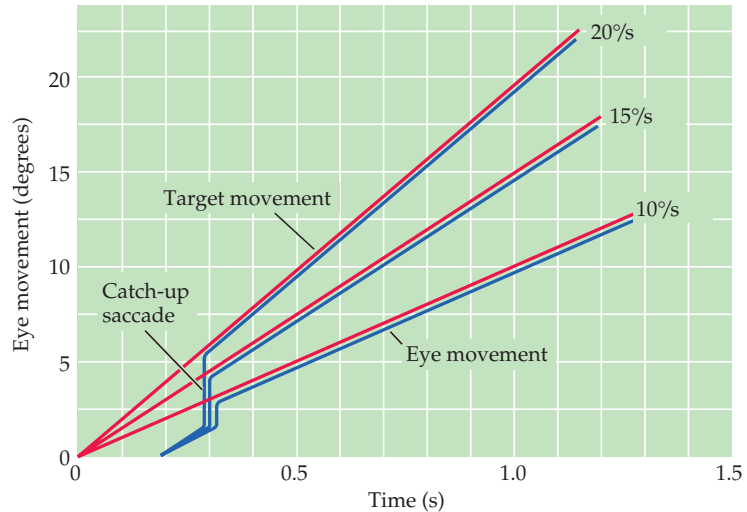
**Smooth pursuit movements** are much slower tracking movements of the eyes designed to keep a moving stimulus on the fovea. Such movements are under voluntary control in the sense that the observer can choose whether or not to track a moving stimulus (Figure 19.5). (Saccades can also be voluntary, but are also made unconsciously.) Surprisingly, however, only highly trained observers can make a smooth pursuit movement in the *absence* of a moving target. Most people who try to move their eyes in a smooth fashion without a moving target simply make a saccade.

The smooth pursuit system can be tested by placing a subject inside a rotating cylinder with vertical stripes. (In practice, the subject is more often seated in front of a screen on which a series of horizontally moving vertical bars is presented to conduct this “optokinetic test.”) The eyes automatically follow a stripe until they reach the end of their excursion. There is then a quick saccade in the direction opposite to the movement, followed once again by smooth pursuit of a stripe. This alternating slow and fast movement of the eyes in response to such stimuli is called **optokinetic nystagmus**. Optokinetic nystagmus is a normal reflexive response of the eyes in response to large-scale movements of the visual scene and should not be confused with the pathological nystagmus that can result from certain kinds of brain injury (for example, damage to the vestibular system or the cerebellum; see Chapters 13 and 18).



**Figure 19.4** The metrics of a saccadic eye movement. The red line indicates the position of a fixation target and the blue line the position of the fovea. When the target moves suddenly to the right, there is a delay of about 200 ms before the eye begins to move to the new target position. (After Fuchs, 1967.)

**Figure 19.5** The metrics of smooth pursuit eye movements. These traces show eye movements (blue lines) tracking a stimulus moving at three different velocities (red lines). After a quick saccade to capture the target, the eye movement attains a velocity that matches the velocity of the target. (After Fuchs, 1967.)



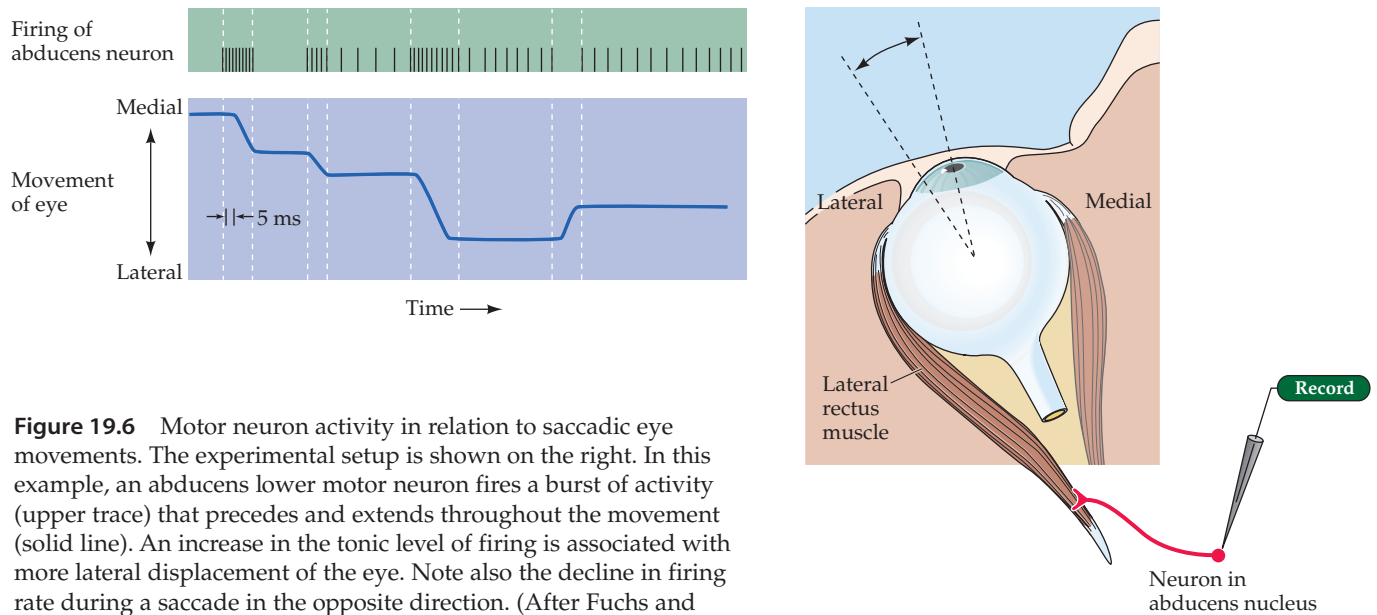
**Vergence movements** align the fovea of each eye with targets located at different distances from the observer. Unlike other types of eye movements in which the two eyes move in the same direction (**conjugate eye movements**), vergence movements are **disconjugate** (or **disjunctive**); they involve either a convergence or divergence of the lines of sight of each eye to see an object that is nearer or farther away. Convergence is one of the three reflexive visual responses elicited by interest in a near object. The other components of the so-called **near reflex triad** are accommodation of the lens, which brings the object into focus, and pupillary constriction, which increases the depth of field and sharpens the image on the retina (see Chapter 10).

**Vestibulo-ocular movements** stabilize the eyes relative to the external world, thus compensating for head movements. These reflex responses prevent visual images from “slipping” on the surface of the retina as head position varies. The action of vestibulo-ocular movements can be appreciated by fixating an object and moving the head from side to side; the eyes automatically compensate for the head movement by moving the same distance but in the opposite direction, thus keeping the image of the object at more or less the same place on the retina. The vestibular system detects brief, transient changes in head position and produces rapid corrective eye movements (see Chapter 13). Sensory information from the semicircular canals directs the eyes to move in a direction opposite to the head movement.

Although the vestibular system operates effectively to counteract rapid movements of the head, it is relatively insensitive to slow movements or to persistent rotation of the head. For example, if the vestibulo-ocular reflex is tested with continuous rotation and without visual cues about the movement of the image (i.e., with eyes closed or in the dark), the compensatory eye movements cease after only about 30 seconds of rotation. However, if the same test is performed with visual cues, eye movements persist. The compensatory eye movements in this case are due to the activation of the smooth pursuit system, which relies not on vestibular information but on visual cues indicating motion of the visual field.

### Neural Control of Saccadic Eye Movements

The problem of moving the eyes to fixate a new target in space (or indeed any other movement) entails two separate issues: controlling the *amplitude* of



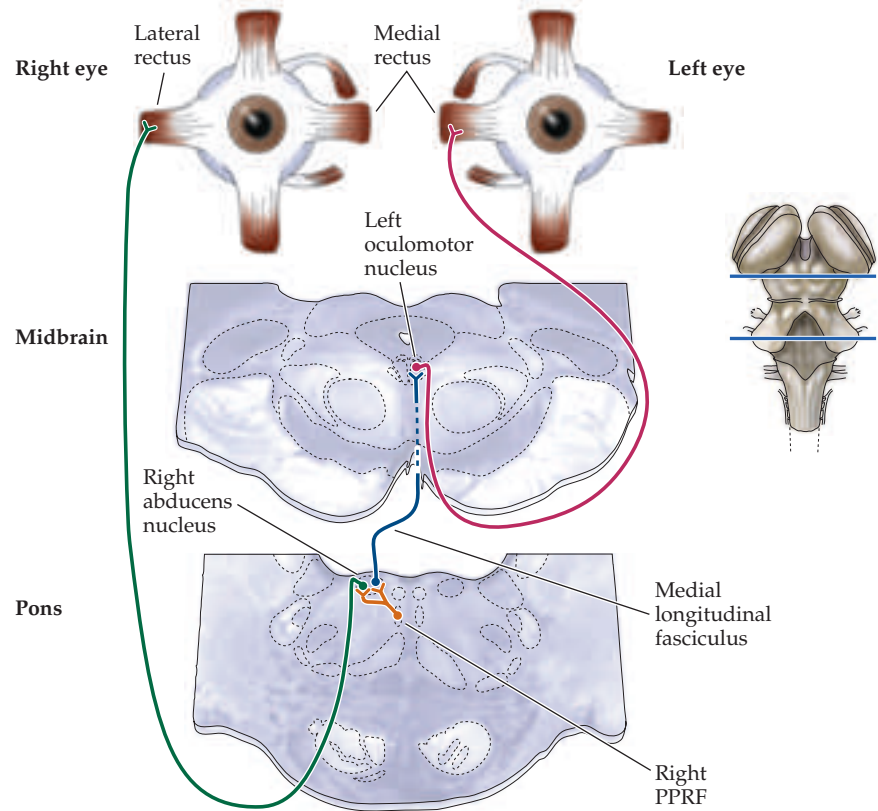
**Figure 19.6** Motor neuron activity in relation to saccadic eye movements. The experimental setup is shown on the right. In this example, an abducens lower motor neuron fires a burst of activity (upper trace) that precedes and extends throughout the movement (solid line). An increase in the tonic level of firing is associated with more lateral displacement of the eye. Note also the decline in firing rate during a saccade in the opposite direction. (After Fuchs and Luschei, 1970.)

movement (how far), and controlling the *direction* of the movement (which way). The amplitude of a saccadic eye movement is encoded by the duration of neuronal activity in the lower motor neurons of the oculomotor nuclei. As shown in Figure 19.6, for instance, neurons in the abducens nucleus fire a burst of action potentials prior to abducting the eye (by causing the lateral rectus muscle to contract) and are silent when the eye is adducted. The amplitude of the movement is correlated with the duration of the burst of action potentials in the abducens neuron. With each saccade, the abducens neurons reach a new baseline level of discharge that is correlated with the position of the eye in the orbit. The steady baseline level of firing holds the eye in its new position.

The direction of the movement is determined by which eye muscles are activated. Although in principle any given direction of movement could be specified by independently adjusting the activity of individual eye muscles, the complexity of the task would be overwhelming. Instead, the direction of eye movement is controlled by the local circuit neurons in two **gaze centers** in the reticular formation, each of which is responsible for generating movements along a particular axis. The **paramedian pontine reticular formation (PPRF)** or **horizontal gaze center** is a collection of local circuit neurons near the midline in the pons responsible for generating horizontal eye movements (Figure 19.7). The **rostral interstitial nucleus** or **vertical gaze center** is located in the rostral part of the midbrain reticular formation and is responsible for vertical movements. Activation of each gaze center separately results in movements of the eyes along a single axis, either horizontal or vertical. Activation of the gaze centers in concert results in oblique movements whose trajectories are specified by the relative contribution of each center.

An example of how the PPRF works with the abducens and oculomotor nuclei to generate a horizontal saccade to the right is shown in Figure 19.7. Neurons in the PPRF innervate cells in the abducens nucleus on the same side of the brain. There are, however, two types of neurons in the abducens nucleus. One type is a lower motor neuron that innervates the lateral rectus

**Figure 19.7** Simplified diagram of synaptic circuitry responsible for horizontal movements of the eyes to the right. Activation of local circuit neurons in the right horizontal gaze center (the PPRF; orange) leads to increased activity of lower motor neurons (red and green) and internuclear neurons (blue) in the right abducens nucleus. The lower motor neurons innervate the lateral rectus muscle of the right eye. The internuclear neurons innervate lower motor neurons in the contralateral oculomotor nucleus, which in turn innervate the medial rectus muscle of the left eye.



muscle on the same side. The other type, called internuclear neurons, send their axons across the midline and ascend in a fiber tract called the **medial longitudinal fasciculus**, terminating in the portion of the oculomotor nucleus that contains lower motor neurons innervating the medial rectus muscle. As a result of this arrangement, activation of PPRF neurons on the right side of the brainstem causes horizontal movements of both eyes to the right; the converse is of course true for the PPRF neurons in the left half of the brainstem.

Neurons in the PPRF also send axons to the medullary reticular formation, where they contact inhibitory local circuit neurons. These local circuit neurons, in turn, project to the contralateral abducens nucleus, where they terminate on lower motor neurons and internuclear neurons. In consequence, activation of neurons in the PPRF on the right results in a reduction in the activity of the lower motor neurons whose muscles would oppose movements of the eyes to the right. This inhibition of antagonists resembles the strategy used by local circuit neurons in the spinal cord to control limb muscle antagonists (see Chapter 15).

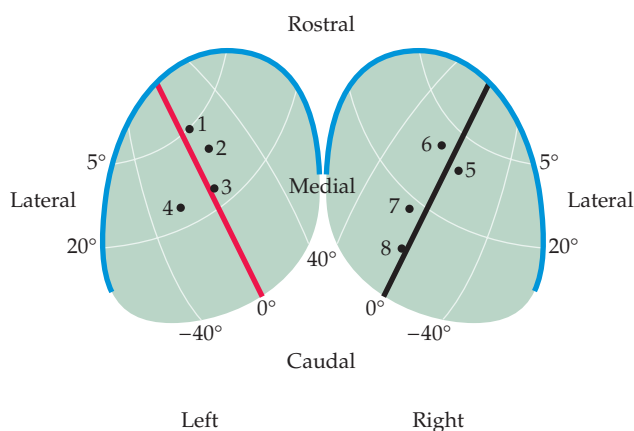
Although saccades can occur in complete darkness, they are often elicited when something attracts attention and the observer directs the foveas toward the stimulus. How then is sensory information about the location of a target in space transformed into an appropriate pattern of activity in the horizontal and vertical gaze centers? Two structures that project to the gaze centers are demonstrably important for the initiation and accurate targeting of saccadic eye movements: the **superior colliculus** of the midbrain, and a region of the frontal lobe that lies just rostral to premotor cortex, known as

the **frontal eye field (Brodmann's area 8)**. Upper motor neurons in both of these structures, each of which contains a topographical motor map, discharge immediately prior to saccades. Thus, activation of a particular site in the superior colliculus or in the frontal eye field produces saccadic eye movements in a specified direction and for a specified distance that is independent of the initial position of the eyes in the orbit. The direction and distance are always the same for a given stimulation site, changing systematically when different sites are activated.

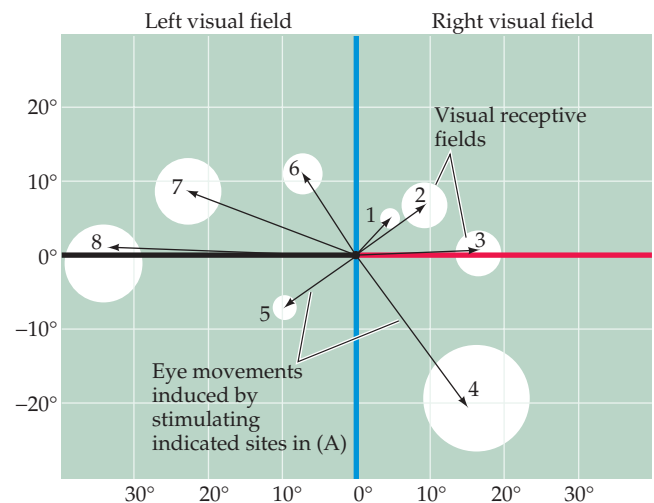
Both the superior colliculus and the frontal eye field also contain cells that respond to visual stimuli; however, the relation between the sensory and motor responses of individual cells is better understood for the superior colliculus. An orderly map of visual space is established by the termination of retinal axons within the superior colliculus (see Chapter 11), and this sensory map is in register with the motor map that generates eye movements. Thus, neurons in a particular region of the superior colliculus are activated by the presentation of visual stimuli in a limited region of visual space. This activation leads to the generation of a saccade that moves the eye by an amount just sufficient to align the foveas with the region of visual space that provided the stimulation (Figure 19.8).

Neurons in the superior colliculus also respond to auditory and somatic stimuli. Indeed, the location in space for these other modalities also is mapped in register with the motor map in the colliculus. Topographically organized maps of auditory space and of the body surface in the superior colliculus can therefore orient the eyes (and the head) in response to a variety of different sensory stimuli. This registration of the sensory and motor maps in the colliculus illustrates an important principle of topographical maps in the motor system, namely to provide an efficient mechanism for sensory motor transformations (Box B).

(A) Superior colliculus



(B) Visual space



**Figure 19.8** Evidence for sensory motor transformation obtained from electrical recording and stimulation in the superior colliculus. (A) Surface views of the superior colliculus illustrating the location of eight separate electrode recording and stimulation sites. (B) Map of visual space showing the receptive field location of the sites in (A) (white circles), and the amplitude and direction of the eye movements elicited by stimulating these sites electrically (arrows). In each case, electrical stimulation results in eye movements that align the fovea with a region of visual space that corresponds to the visual receptive field of the site. (After Schiller and Stryker, 1972.)

## Box B

### Sensory Motor Integration in the Superior Colliculus

The superior colliculus is a laminated structure in which the differences between the layers provide clues about how sensory and motor maps interact to produce appropriate movements. As discussed in the text, the superficial or “visual” layer of the colliculus receives input from retinal axons that form a topographic map. Thus, each site in the superficial layer is activated maximally by the presence of a stimulus at a particular point of visual space. In contrast, neurons in the deeper or “motor” layers generate bursts of action potentials that command saccades, effectively generating a motor map; thus, activation of different sites generates saccades having different vectors. The visual and motor maps are *in register*, so that visual cells responding to a stimulus in a specific region of visual space are located directly above the motor cells that command eye movements toward that same region (see Figure 19.8).

The registration of the visual and motor maps suggests a simple strategy for how the eyes might be guided toward an object of interest in the visual field. When an object appears at a particular location in the visual field, it will activate neurons in the corresponding part of the visual map. As a result, bursts of action potentials are generated by the subjacent motor cells to command a saccade that rotates the two eyes just the right amount to direct the foveas toward that same location in the

visual field. This behavior is called “visual grasp” because successful sensory motor integration results in the accurate foveation of a visual target.

This seemingly simple model, formulated in the early 1970s when the collicular maps were first found, assumes point to point connections between the visual and motor maps. In practice, however, these connections have been difficult to demonstrate. Neither the anatomical nor the physiological methods available at the time were sufficiently precise to establish these postulated synaptic connections. At about the same time, motor neurons were found to command saccades to nonvisual stimuli; moreover, spontaneous saccades occur in the dark. Thus, it was clear that visual layer activity is not always necessary for saccades. To confuse matters further, animals could be trained *not* to make a saccade when an object appeared in the visual field, showing that the activation of visual neurons is sometimes insufficient to command saccades. The fact that activity of neurons in the visual map is *neither necessary nor sufficient* for eliciting saccades led investigators away from the simple model of direct connections between corresponding regions of the two maps, toward models that linked the layers indirectly through pathways that detoured through the cortex.

Eventually, however, new and better methods resolved this uncertainty.

Techniques for filling single cells with axonal tracers showed an overlap between descending visual layer axons and ascending motor layer dendrites, in accord with direct anatomical connections between corresponding regions of the maps. At the same time, *in vitro* whole-cell patch clamp recording (see Box A in Chapter 4) permitted more discriminating functional studies that distinguished excitatory and inhibitory inputs to the motor cells. These experiments showed that the visual and motor layers do indeed have the functional connections required to initiate the command for a visually guided saccadic eye movement. A single brief electrical stimulus delivered to the superficial layer generates a prolonged burst of action potentials that resembles the command bursts that normally occur just before a saccade (see figure).

These direct connections presumably provide the substrate for the very short latency reflex-like “express saccades” that are unaffected by destruction of the frontal eye fields. Other visual and nonvisual inputs to the deep layers probably explain why activation of the retina is neither necessary nor sufficient for the production of saccades.

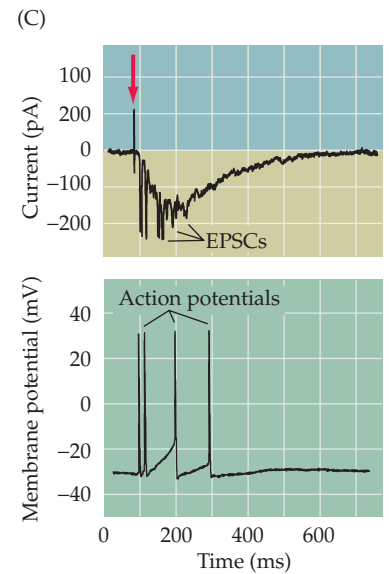
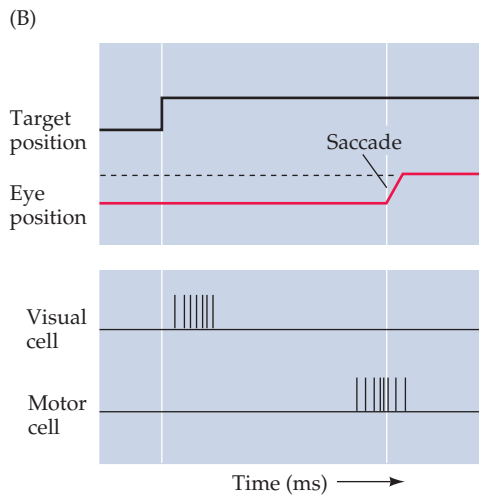
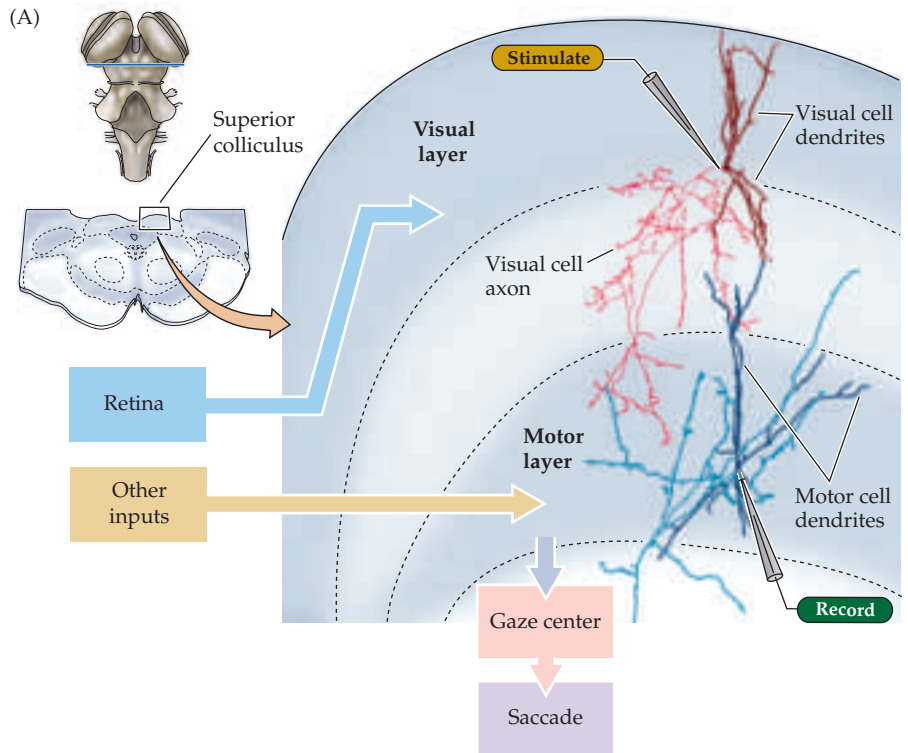
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The functional relationship between the frontal eye field and the superior colliculus in controlling eye movements is similar to that between the motor cortex and the red nucleus in the control of limb movements (see Chapter 16). The frontal eye field projects to the superior colliculus, and the superior colliculus projects to the PPRF on the contralateral side (Figure 19.9). (It also projects to the vertical gaze center, but for simplicity the discussion here is



(A) The superior colliculus receives visual input from the retina and sends a command signal to the gaze centers to initiate a saccade (see text). In the experiment illustrated here, a stimulating electrode activates cells in the visual layer and a patch clamp pipette records the response evoked in a neuron in the subjacent motor layer. The cells in the visual and motor layers were subsequently labeled with a tracer called biocytin. This experiment demonstrates that the terminals of the visual neuron are located in the same region as the dendrites of the motor neuron. (B) The onset of a target in the visual field (top trace) is followed after a short interval by a saccade to foveate the target (second trace). In the superior colliculus, the visual cell responds shortly after the onset of the target, while the motor cell responds later, just before the onset of the saccade. (C) Bursts of excitatory postsynaptic currents (EPSCs) recorded from a motor layer neuron in response to a brief (0.5 ms) current stimulus applied via a steel wire electrode in the visual layer (top; see arrow). These synaptic currents generate bursts of action potentials in the same cell (bottom).



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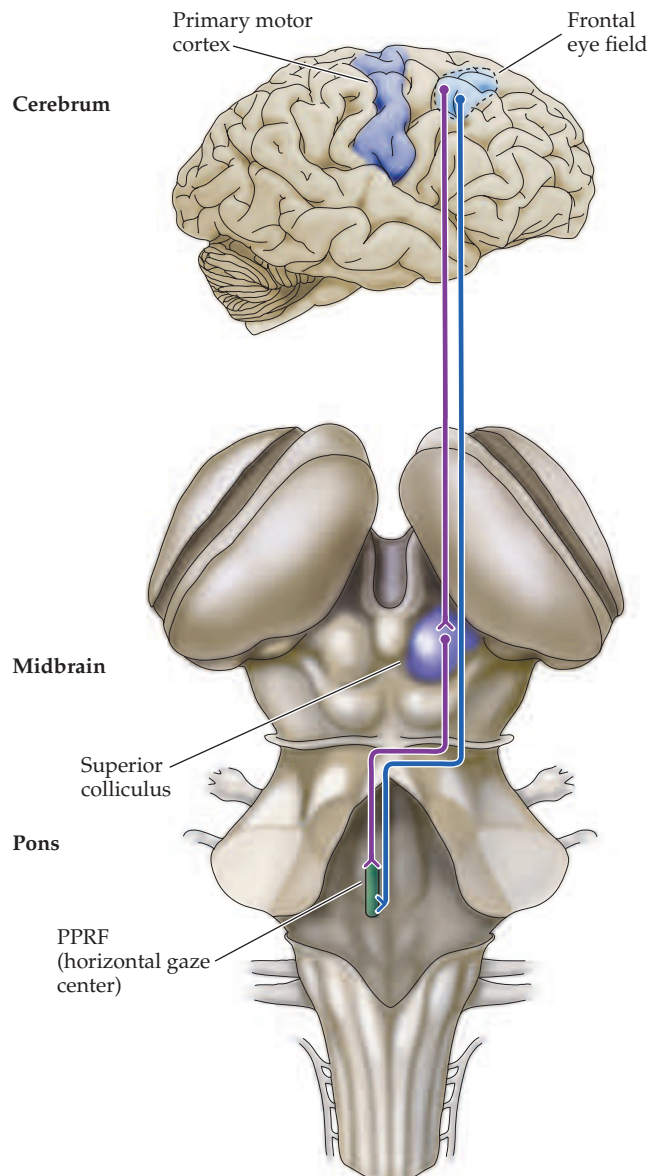
SCHILLER, P. H. AND M. STRYKER (1972) Single-unit recording and stimulation in superior colliculus of the alert rhesus monkey. *J. Neurophysiol.* 35: 915–924.

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limited to the PPRF.) The frontal eye field can thus control eye movements by activating selected populations of superior colliculus neurons. This cortical area also projects directly to the contralateral PPRF; as a result, the frontal eye field can also control eye movements independently of the superior colliculus. The parallel inputs to the PPRF from the frontal eye field and superior colliculus are reflected in the deficits that result from damage to these

**Figure 19.9** The relationship of the frontal eye field in the right cerebral hemisphere (Brodmann's area 8) to the superior colliculus and the horizontal gaze center (PPRF). There are two routes by which the frontal eye field can influence eye movements in humans: indirectly by projections to the superior colliculus, which in turn projects to the contralateral PPRF, and directly by projections to the contralateral PPRF.



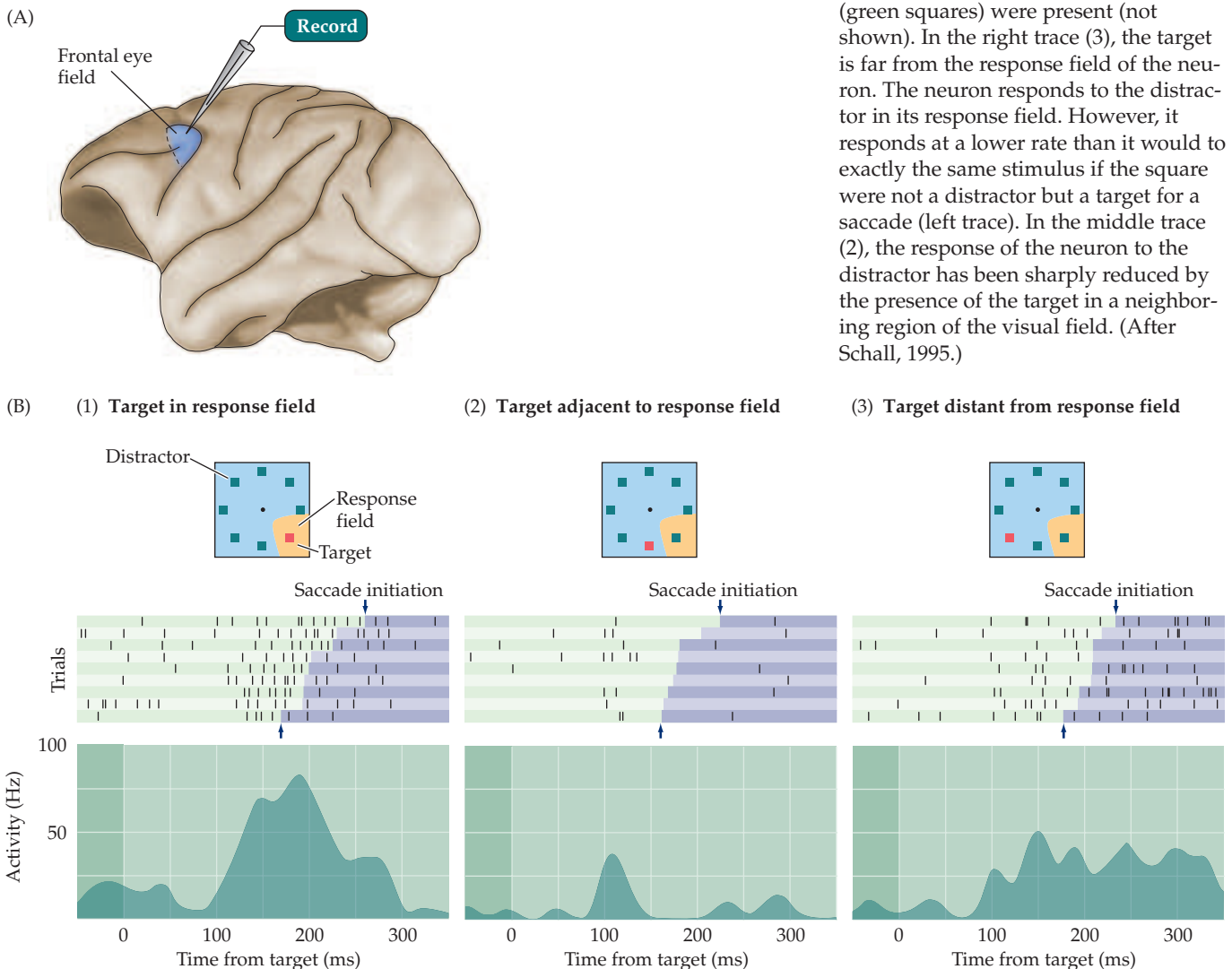
structures. Injury to the frontal eye field results in an inability to make saccades to the contralateral side and a deviation of the eyes to the side of the lesion. These effects are transient, however; in monkeys with experimentally induced lesions of this cortical region, recovery is virtually complete in two to four weeks. Lesions of the superior colliculus change the accuracy, frequency, and velocity of saccades; yet saccades still occur, and the deficits also improve with time. These results suggest that the frontal eye fields and the superior colliculus provide complementary pathways for the control of saccades. Moreover, one of these structures appears to be able to compensate (at least partially) for the loss of the other. In support of this interpretation, combined lesions of the frontal eye field and the superior colliculus produce a dramatic and permanent loss in the ability to make saccadic eye movements.

These observations do not, however, imply that the frontal eye fields and the superior colliculus have the same functions. Superior colliculus lesions

produce a permanent deficit in the ability to perform very short latency reflex-like eye movements called “express saccades.” The express saccades are evidently mediated by direct pathways to the superior colliculus from the retina or visual cortex that can access the upper motor neurons in the colliculus without extensive, and more time-consuming, processing in the frontal cortex (see Box B). In contrast, frontal eye field lesions produce permanent deficits in the ability to make saccades that are not guided by an external target. For example, patients (or monkeys) with a lesion in the frontal eye fields cannot voluntarily direct their eyes *away* from a stimulus in the visual field, a type of eye movement called an “antisaccade.” Such lesions also eliminate the ability to make a saccade to the remembered location of a target that is no longer visible.

Finally, the frontal eye fields are essential for systematically scanning the visual field to locate an object of interest within an array of distracting objects (see Figure 19.1). Figure 19.10 shows the responses of a frontal eye field neuron during a visual task in which a monkey was required to foveate a target located within an array of distracting objects. This frontal eye field

**Figure 19.10** Responses of neurons in the frontal eye fields. (A) Locus of the left frontal eye field on a lateral view of the rhesus monkey brain. (B) Activation of a frontal eye field neuron during visual search for a target. The vertical tickmarks represent action potentials, and each row of tick marks is a different trial. The graphs below show the average frequency of action potentials as a function of time. The change in color from green to purple in each row indicates the time of onset of a saccade toward the target. In the left trace (1), the target (red square) is in the part of the visual field “seen” by the neuron, and the response to the target is similar to the response that would be generated by the neuron even if no distractors (green squares) were present (not shown). In the right trace (3), the target is far from the response field of the neuron. The neuron responds to the distractor in its response field. However, it responds at a lower rate than it would to exactly the same stimulus if the square were not a distractor but a target for a saccade (left trace). In the middle trace (2), the response of the neuron to the distractor has been sharply reduced by the presence of the target in a neighboring region of the visual field. (After Schall, 1995.)



neuron discharges at different levels to the same stimulus, depending on whether the stimulus is the target of the saccade or a “distractor,” and on the location of the distractor relative to the actual target. For example, the differences between the middle and the left and right traces in Figure 19.10 demonstrate that the response to the distractor is much reduced if it is located close to the target in the visual field. Results such as these suggest that lateral interactions within the frontal eye fields enhance the neuronal responses to stimuli that will be selected as saccade targets, and that such interactions suppress the responses to uninteresting and potentially distracting stimuli. These sorts of interactions presumably reduce the occurrence of unwanted saccades to distracting stimuli in the visual field.

### Neural Control of Smooth Pursuit Movements

Smooth pursuit movements are also mediated by neurons in the PPRF, but are under the influence of motor control centers other than the superior colliculus and frontal eye field. (The superior colliculus and frontal eye field are exclusively involved in the generation of saccades.) The exact route by which visual information reaches the PPRF to generate smooth pursuit movements is not known (a pathway through the cerebellum has been suggested). It is clear, however, that neurons in the striate and extrastriate visual areas provide sensory information that is essential for the initiation and accurate guidance of smooth pursuit movements. In monkeys, neurons in the middle temporal area (which is largely concerned with the perception of moving stimuli and a target of the magnocellular stream; see Chapter 11) respond selectively to targets moving in a specific direction. Moreover, damage to this area disrupts smooth pursuit movements. In humans, damage of comparable areas in the parietal and occipital lobes also results in abnormalities of smooth pursuit movements. Unlike the effects of lesions to the frontal eye field and the superior colliculus, the deficits are in eye movements made toward the side of the lesion. For example, a lesion of the left parieto-occipital region is likely to result in an inability to track an object moving from right to left.

### Neural Control of Vergence Movements

When a person wishes to look from one object to another object that are located at different distances from the eyes, a saccade is made that shifts the direction of gaze toward the new object, and the eyes either diverge or converge until the object falls on the fovea of each eye. The structures and pathways responsible for mediating the vergence movements are not well understood, but appear to include several extrastriate areas in the occipital lobe (see Chapter 11). Information about the location of retinal activity is relayed through the two lateral geniculate nuclei to the cortex, where the information from the two eyes is integrated. The appropriate command to diverge or converge the eyes, which is based largely on information from the two eyes about the amount of binocular disparity (see Chapter 11), is then sent via upper motor neurons from the occipital cortex to “vergence centers” in the brainstem. One such center is a population of local circuit neurons located in the midbrain near the oculomotor nucleus. These neurons generate a burst of action potentials. The onset of the burst is the command to generate a vergence movement, and the frequency of the burst determines its velocity.

There is a division of labor within the vergence center, so that some neurons command convergence movements while others command divergence movements. These neurons also coordinate vergence movements of the eyes with accommodation of the lens and pupillary constriction to produce the near reflex discussed in Chapter 10.

## Summary

Despite their specialized function, the systems that control eye movements have much in common with the motor systems that govern movements of other parts of the body. Just as the spinal cord provides the basic circuitry for coordinating the actions of muscles around a joint, the reticular formation of the pons and midbrain provides the basic circuitry that mediates movements of the eyes. Descending projections from higher-order centers in the superior colliculus and the frontal eye field innervate the brainstem gaze centers, providing a basis for integrating eye movements with a variety of sensory information that indicates the location of objects in space. The superior colliculus and the frontal eye field are organized in a parallel as well as a hierarchical fashion, enabling one of these structures to compensate for the loss of the other. Eye movements, like other movements, are also under the control of the basal ganglia and cerebellum (see Chapters 17 and 18); this control ensures the proper initiation and successful execution of these relatively simple motor behaviors, thus allowing observers to interact efficiently with the universe of things that can be seen.

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