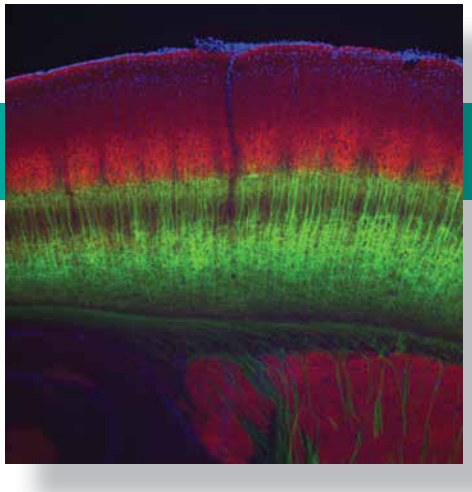
CHAPTER FOURTEEN



Brain Control of Movement

INTRODUCTION

In Chapter 13, we discussed the organization of the peripheral somatic motor system: the joints, skeletal muscles, and their sensory and motor innervation. We saw that the final common pathway for behavior is the alpha motor neuron, that the activity of this cell is under the control of sensory feedback and spinal interneurons, and that reflex movements reveal the complexity of this spinal control system. In this chapter, we'll explore how the brain influences the activity of the spinal cord to command voluntary movements.

The central motor system is arranged as a hierarchy of control levels, with the forebrain at the top and the spinal cord at the bottom. It is useful to think of this motor control hierarchy as having three levels (Table 14.1). The highest level, represented by the association areas of neocortex and basal ganglia of the forebrain, is concerned with *strategy*: the goal of the movement and the movement strategy that best achieves the goal. The middle level, represented by the motor cortex and cerebellum, is concerned with *tactics:* the sequences of muscle contractions, arranged in space and time, required to smoothly and accurately achieve the strategic goal. The lowest level, represented by the brain stem and spinal cord, is concerned with *execution*: activation of the motor neuron and interneuron pools that generate the goal-directed movement and make any necessary adjustments of posture.

To appreciate the different contributions of the three hierarchical levels to movement, consider the actions of a baseball pitcher preparing to pitch to a batter (Figure 14.1). The cerebral neocortex has information—based on vision, audition, somatic sensation, and proprioception—about precisely where the body is in space. Strategies must be devised to move the body from the current state to one in which a pitch is delivered and the desired outcome is attained (a swing and a miss by the batter). Several throwing options are available to the pitcher—a curve ball, a fast ball, a slider, and so on—and these alternatives are filtered through the basal ganglia and back to the cortex until a final decision is made, based in large part on past experience (e.g., "This batter hit a home run last time I threw a fast ball"). The motor areas of cortex and the cerebellum then make the tactical decision (to throw a curve ball) and issue instructions to the brain stem and spinal cord. Activation of neurons in the brain stem and spinal cord then causes the movement to be executed. The properly timed activation of motor neurons in the cervical spinal cord generates a coordinated movement of the shoulder, elbow, wrist, and fingers. Simultaneously, brain stem inputs to the thoracic and lumbar spinal cord command the appropriate leg movements along with postural adjustments that keep the pitcher from falling over during the throw. In addition, brain stem motor neurons are activated to keep the pitcher's eyes fixed on the catcher, his target, as his head and body move about.

According to the laws of physics, the movement of a thrown baseball through space is *ballistic*, referring to a trajectory that cannot be altered.

TABLE 14.1 **The Motor Control Hierarchy**

Level	Function	Structures
High	Strategy	Association areas of neocortex, basal ganglia
Middle	Tactics	Motor cortex, cerebellum
Low	Execution	Brain stem, spinal cord

◀ FIGURE 14.1
The contributions of the motor control hierarchy. As a baseball pitcher plans to pitch a ball to a batter, chooses which pitch to throw, and then throws the ball, he engages the three hierarchical levels of motor control.

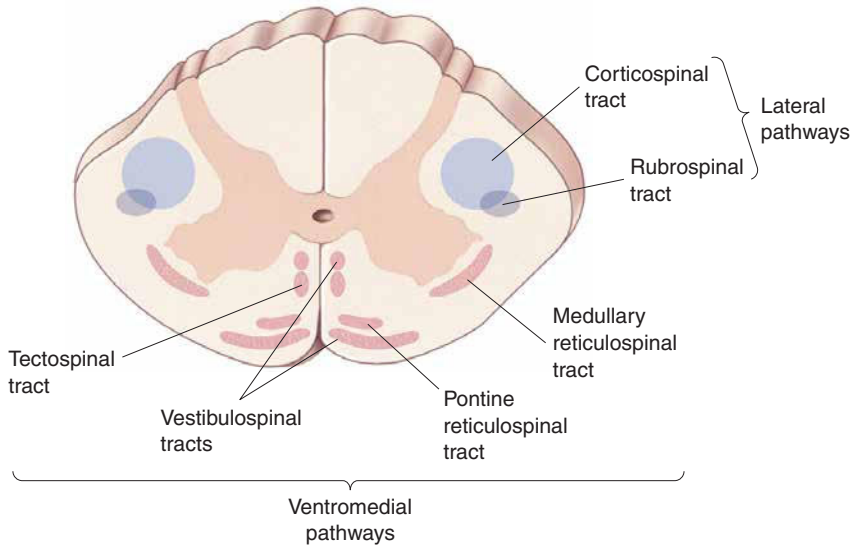
The movement of the pitcher's arm that throws the ball is also described as ballistic because it cannot be altered once initiated. This type of rapid voluntary movement is not under the same type of sensory feedback control that regulates antigravity postural reflexes (see Chapter 13). The reason is simple: The movement is too fast to be adjusted by sensory feedback. But the movement does not occur in the absence of sensory information. Sensory information *before* the movement was initiated was crucial in order to decide when to initiate the pitch, to determine the starting positions of the limbs and body, and to anticipate any changes in resistance during the throw. And sensory information *during* the movement is also important, not necessarily for the movement at hand, but for improving subsequent similar movements.

The proper functioning of each level of the motor control hierarchy relies so heavily on sensory information that the motor system of the brain might properly be considered a *sensorimotor system*. At the highest level, sensory information generates a mental image of the body and its relationship to the environment. At the middle level, tactical decisions are based on the memory of sensory information from past movements. At the lowest level, sensory feedback is used to maintain posture, muscle length, and tension before and after each voluntary movement.

In this chapter, we investigate this hierarchy of motor control and how each level contributes to the control of the peripheral somatic motor system. We start by exploring the pathways that bring information to the spinal motor neurons. From there we will ascend to the highest levels of the motor hierarchy, and then we'll fill in the pieces of the puzzle that bring the different levels together. Along the way, we'll describe how pathology in specific parts of the motor system leads to particular movement disorders.

DESCENDING SPINAL TRACTS

How does the brain communicate with the motor neurons of the spinal cord? Axons from the brain descend through the spinal cord along two major groups of pathways, shown in Figure 14.2. One is in the lateral column of the spinal cord, and the other is in the ventromedial column. Remember this rule of thumb: The **lateral pathways** are involved in voluntary movement of the distal musculature and are under direct cortical control, and the **ventromedial pathways** are involved in the control of posture and locomotion and are under brain stem control.



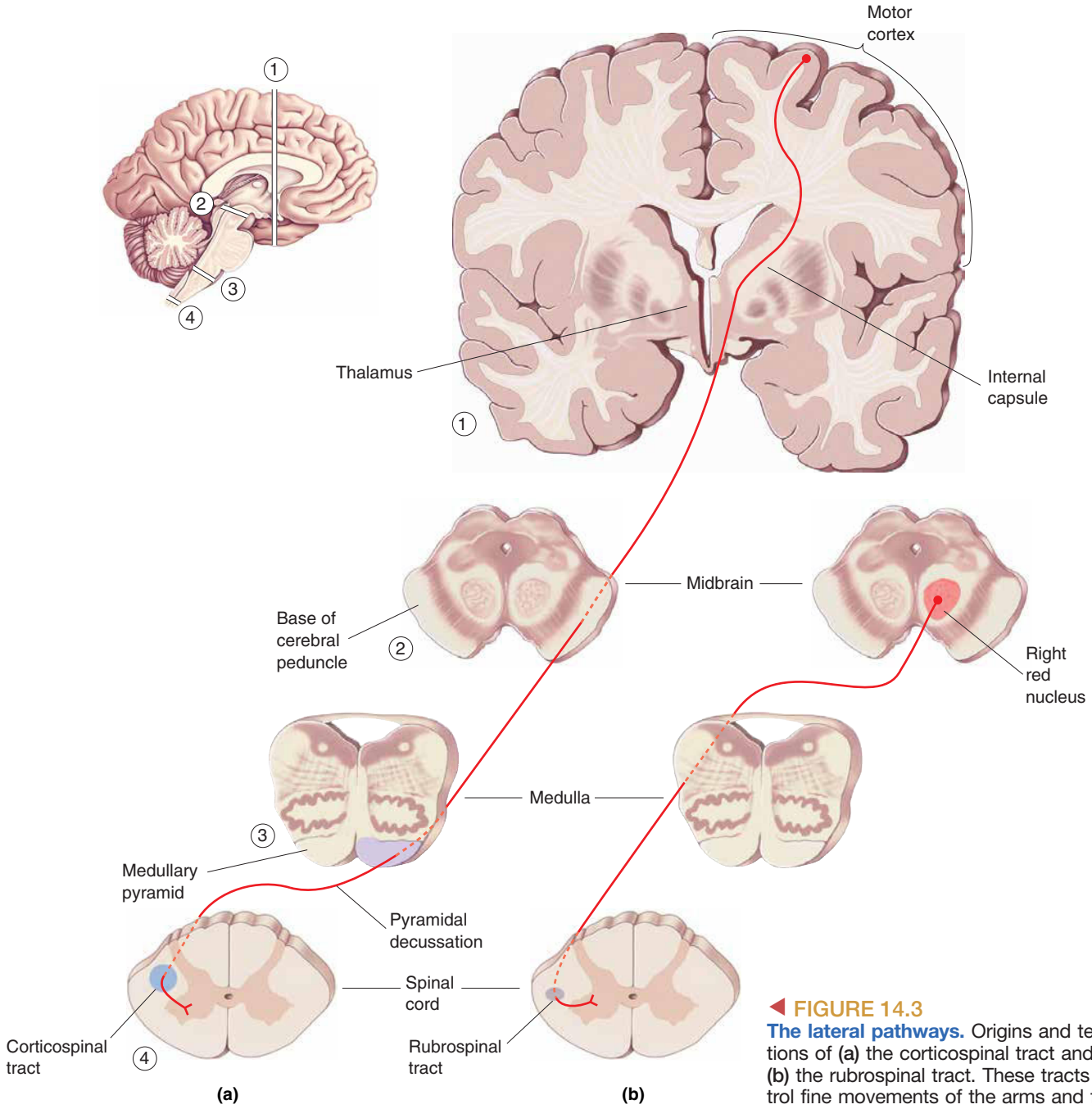
▶ FIGURE 14.2
The descending tracts of the spinal cord. The lateral pathways, consisting of the corticospinal and rubrospinal tracts, control voluntary movements of the distal musculature. The ventromedial pathways, consisting of the medullary reticulospinal, pontine reticulospinal, vestibulospinal, and tectospinal tracts, control postural muscles.

The Lateral Pathways

The most important component of the lateral pathways is the **corticospinal tract** (Figure 14.3a). Originating in the neocortex, it is the longest and one of the largest central nervous system (CNS) tracts (10^6 axons in humans). Two-thirds of the axons in the tract originate in areas 4 and 6 of the frontal lobe, collectively called **motor cortex**. Most of the remaining axons in the corticospinal tract derive from the somatosensory areas of the parietal lobe and serve to regulate the flow of somatosensory information to the brain (see Chapter 12). Axons from the cortex pass through the internal capsule bridging the telencephalon and thalamus, course through the base of the *cerebral peduncle*, a large collection of axons in the midbrain, then pass through the pons, and collect to form a tract at the base of the medulla. The tract forms a bulge, called the *medullary pyramid*, running down the ventral surface of the medulla. When cut, the tract's cross section is roughly triangular, explaining why it is also called the **pyramidal tract**.

At the junction of the medulla and spinal cord, the pyramidal tract crosses, or decussates, at the pyramidal decussation. This means that the *right* motor cortex directly commands the movement of the *left* side of the body, and the *left* motor cortex controls the muscles on the *right* side. As the axons cross, they collect in the lateral column of the spinal cord and form the lateral corticospinal tract. The corticospinal tract axons terminate in the dorsolateral region of the ventral horns and intermediate gray matter, the location of the motor neurons and interneurons that control the distal muscles, particularly the flexors (see Chapter 13).

A much smaller component of the lateral pathways is the **rubrospinal tract**, which originates in the **red nucleus** of the midbrain, named for its distinctive pinkish hue in a freshly dissected brain (*rubro* is from the Latin for "red"). Axons from the red nucleus decussate in the pons, almost immediately, and parallel those in the corticospinal tract in the lateral column of the spinal cord (Figure 14.3b). A major source of input to the red nucleus is the very region of frontal cortex that also contributes to the corticospinal tract. Indeed, it appears that this indirect corticorubrospinal pathway has largely been replaced by the direct corticospinal path over the course of primate evolution Thus, while the rubrospinal tract contributes importantly to motor control in many mammalian species, in humans it appears to be reduced, most of its functions subsumed by the corticospinal tract.



◀ FIGURE 14.3
The lateral pathways. Origins and terminations of **(a)** the corticospinal tract and **(b)** the rubrospinal tract. These tracts control fine movements of the arms and fingers.

The Effects of Lateral Pathway Lesions. Donald Lawrence and Hans Kuypers laid the foundation for the modern view of the functions of the lateral pathways in the late 1960s. Experimental lesions in both corticospinal and rubrospinal tracts in monkeys rendered them unable to make fractionated movements of the arms and hands; that is, they could not move their shoulders, elbows, wrists, and fingers independently. For example, they could grasp small objects with their hands but only by using all the fingers at once. Voluntary movements were also slower and less accurate. Despite this, the animals could sit upright and stand with normal posture. By analogy, a human with a lateral pathway lesion would be able to stand on the pitcher's mound but would be unable to grip the ball properly and throw it accurately.

BOX 14.1 OF SPECIAL INTEREST

Paresis, Paralysis, Spasticity, and Babinski

The neural components of the motor system extend from the highest reaches of the cerebral cortex to the farthest terminals of the motor axons in muscles. Its sheer size makes the motor system uncommonly vulnerable to disease and trauma. The site of motor system damage has a big effect on the types of deficits patients experience.

Damage to the lower parts of the motor system—alpha motor neurons or their motor axons—leads to easily predicted consequences. Partial damage may cause *paresis* (weakness). Complete severing of a motor nerve leads to *paralysis,* a loss of movement of the affected muscles, and *areflexia*, an absence of their spinal reflexes. The muscles also have no *tone* or resting tension; they are flaccid and soft. Damaged motor neurons can no longer exert their trophic influence on muscle fibers (see Chapter 13). The muscles profoundly *atrophy* (decrease in size) with time, losing up to 70–80% of their mass.

Damage to the upper parts of the motor system—the motor cortex or the various motor tracts that descend into the spinal cord—can cause a distinctly different set of motor problems. These are common after a stroke, which damages regions of the cortex or brain stem by depriving them of their blood supply, or traumatic injury, such as a knife or gunshot wound, or even a demyelinating disease that damages axons (see Box 4.5).

Immediately following severe upper motor system damage, there is a period of *spinal shock*: reduced muscle tone (*hypotonia*), areflexia, and paralysis. Paralysis is known as *hemiplegia* if it occurs on one side of the body, *paraplegia* if it involves only the legs, and *quadriplegia* if it involves all four limbs. With the loss of descending brain influences, the functions of the spinal cord appear to shut down. Over the next several days, some of its reflexive functions mysteriously reappear. This is not necessarily a good thing. A condition called *spasticity* sets in, often permanently. Spasticity is characterized by a dramatic and sometimes painful increase of muscle tone (*hypertonia*) and spinal reflexes (*hyperreflexia*), compared to normal. Overactive stretch reflexes often cause *clonus*, rhythmic cycles of contraction and relaxation when limb muscles are stretched.

Another indication of motor tract damage is the *Babinski sign,* described by the French neurologist Joseph Babinski in 1896. Sharply scratching the sole of the foot from the heel toward the toes causes reflexive upward flexion of the big toe and an outward fanning of the other toes. The normal response to this stimulus, for anyone older than about 2 years, is to curl the toes downward. Normal infants also exhibit the Babinski sign, presumably because their descending motor tracts have not yet matured.

By systematically testing a patient's reflexes, muscle tone, and motor ability across his body, a skilled neurologist can often deduce the site and severity of motor system damage with impressive precision.

Lesions in the monkeys' corticospinal tracts alone caused a movement deficit as severe as that observed after lesions in the lateral columns. Interestingly, however, many functions gradually reappeared over the months following surgery. In fact, the only permanent deficit was some weakness of the distal flexors and an inability to move the fingers independently. A subsequent lesion in the rubrospinal tract completely reversed this recovery, however. These results suggest that the corticorubrospinal pathway was able, over time, to partially compensate for the loss of the corticospinal tract input.

Strokes that damage the motor cortex or the corticospinal tract are common in humans. Their immediate consequence can be paralysis on the contralateral side, but considerable recovery of voluntary movements may occur over time (Box 14.1). As in Lawrence and Kuypers' lesioned monkeys, it is the fine, fractionated movements of the fingers that are least likely to recover.

The Ventromedial Pathways

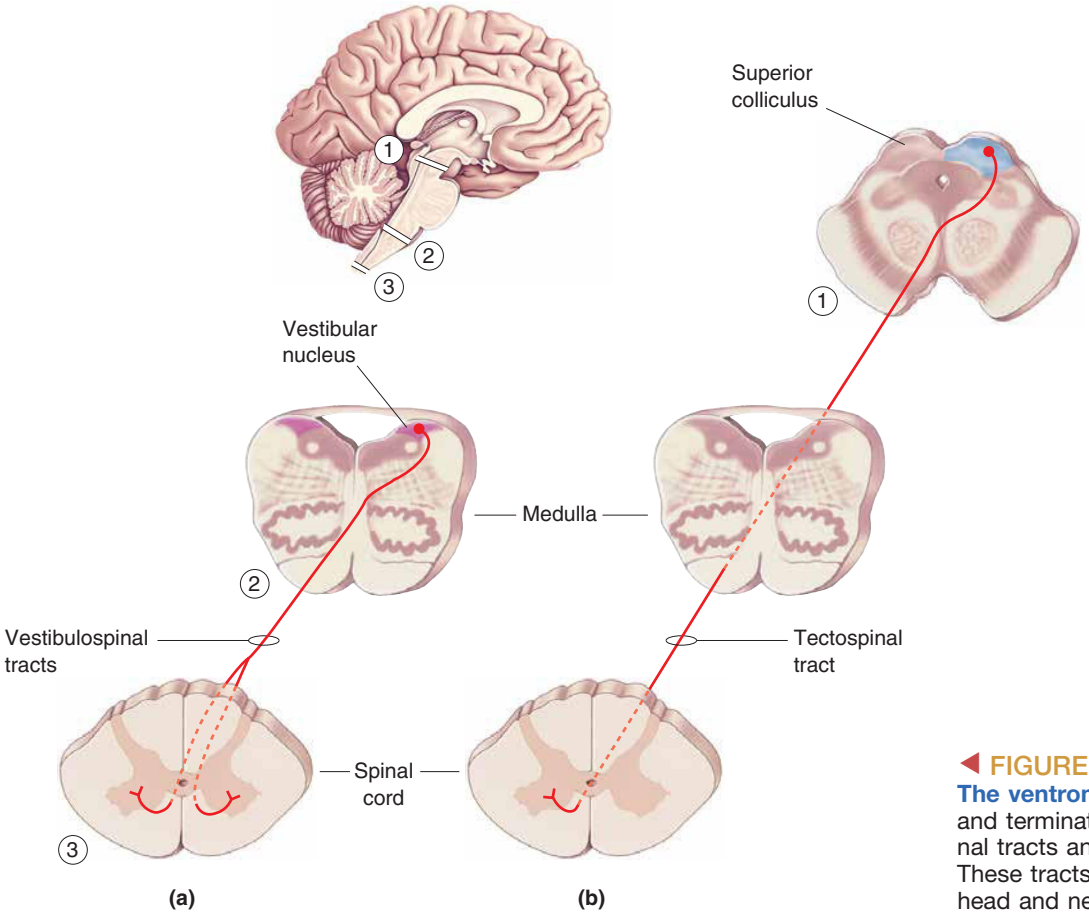
The ventromedial pathways contain four descending tracts that originate in the brain stem and terminate among the spinal interneurons controlling proximal and axial muscles. These tracts are the vestibulospinal tract, the tectospinal tract, the pontine reticulospinal tract, and the medullary reticulospinal tract. The ventromedial pathways use sensory

information about balance, body position, and the visual environment to reflexively maintain balance and body posture.

The Vestibulospinal Tracts. The vestibulospinal and tectospinal tracts function to keep the head balanced on the shoulders as the body moves through space and to turn the head in response to new sensory stimuli. The **vestibulospinal tracts** originate in the *vestibular nuclei* of the medulla, which relay sensory information from the vestibular labyrinth in the inner ear (Figure 14.4a). The *vestibular labyrinth* consists of fluid-filled canals and cavities in the temporal bone that are closely associated with the cochlea (see Chapter 11). The motion of the fluid in this labyrinth, which accompanies movement of the head, activates hair cells that signal the vestibular nuclei via cranial nerve VIII.

One component of the vestibulospinal tracts projects bilaterally down the spinal cord and activates the cervical spinal circuits that control neck and back muscles and thus guide head movement. Stability of the head is important because the head contains our eyes, and keeping the eyes stable, even as our body moves, ensures that our image of the world remains stable. Another component of the vestibulospinal tracts projects ipsilaterally as far down as the lumbar spinal cord. It helps us maintain an upright and balanced posture by facilitating extensor motor neurons of the legs.

The Tectospinal Tract. The **tectospinal tract** originates in the superior colliculus of the midbrain, which receives direct input from the retina (Figure 14.4b). (Recall from Chapter 10 that optic tectum is another name



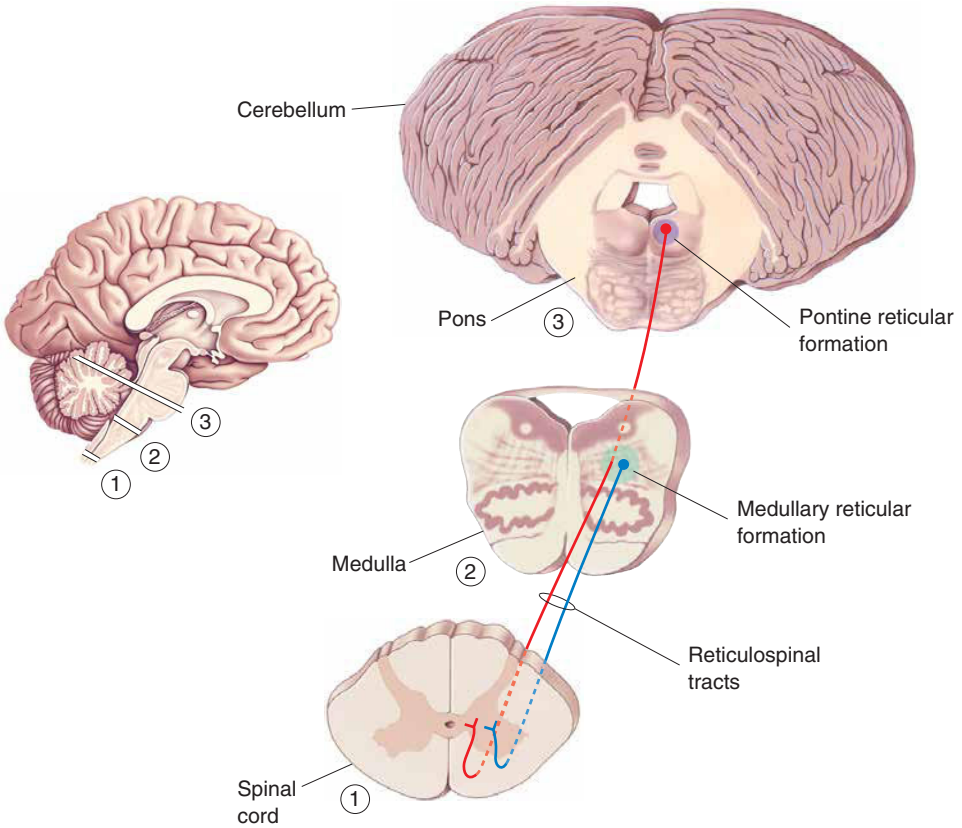
◀ FIGURE 14.4
The ventromedial pathways. Origins and terminations of **(a)** the vestibulospinal tracts and **(b)** the tectospinal tract. These tracts control the posture of the head and neck.

for the superior colliculus.) Besides its retinal input, the superior colliculus receives projections from visual cortex, as well as afferent axons carrying somatosensory and auditory information. From this input, the superior colliculus constructs a map of the world around us; stimulation at one site in this map leads to an orienting response that directs the head and eyes to move so that the appropriate point of space is imaged on the fovea. Activation of the colliculus by an image of a runner sprinting toward second base, for example, would cause the pitcher to orient his head and eyes toward this important new stimulus.

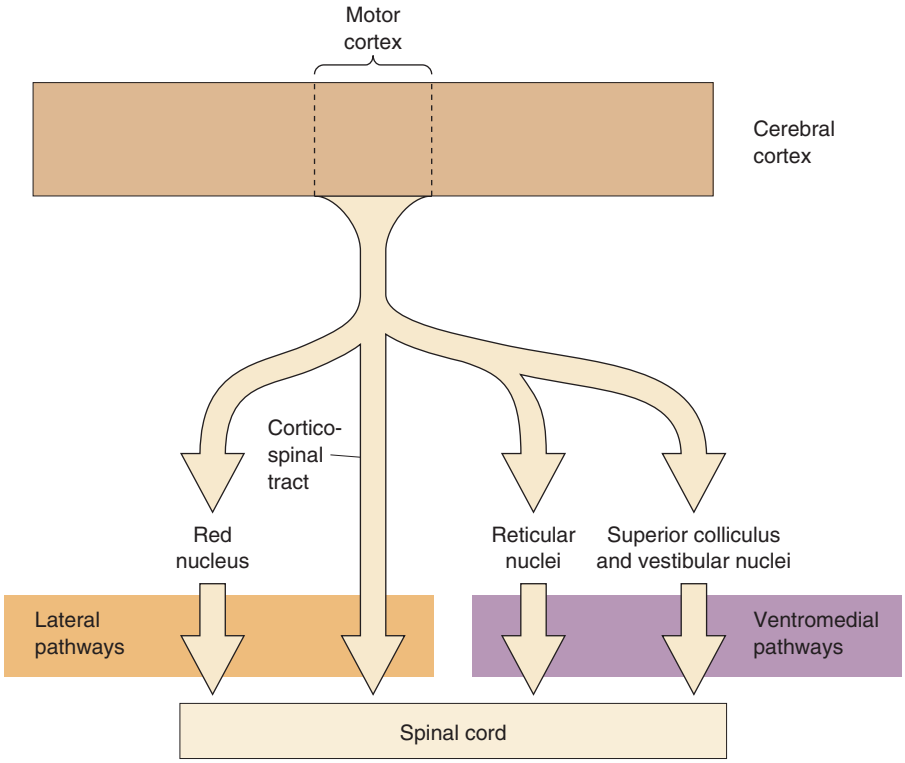
After leaving the colliculus, axons of the tectospinal tract quickly decussate and project close to the midline into cervical regions of the spinal cord, where they help to control muscles of the neck, upper trunk, and shoulders.

The Pontine and Medullary Reticulospinal Tracts. The reticulospinal tracts arise mainly from the **reticular formation** of the brain stem, which runs the length of the brain stem at its core, just under the cerebral aqueduct and fourth ventricle. A complex meshwork of neurons and fibers, the reticular formation receives input from many sources and participates in many different functions. For the purposes of our discussion of motor control, the reticular formation may be divided into two parts that give rise to two different descending tracts: the pontine (medial) reticulospinal tract and the medullary (lateral) reticulospinal tract (Figure 14.5).

The **pontine reticulospinal tract** enhances the antigravity reflexes of the spinal cord. Activity in this pathway, by facilitating the extensors of the lower limbs, helps maintain a standing posture by resisting the effects of gravity. This type of regulation is an important component of motor control: Keep in mind that most of the time, the activity of ventral horn neurons maintains, rather than changes, muscle length and tension.



▶ FIGURE 14.5
The pontine (medial) and medullary (lateral) reticulospinal tracts. These components of the ventromedial pathway control posture of the trunk and the antigravity muscles of the limbs.



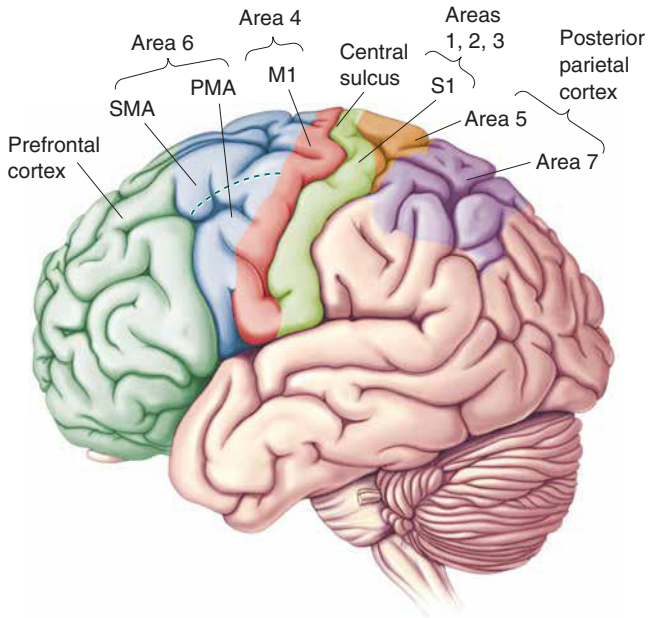
◀ FIGURE 14.6
A summary of the major descending spinal tracts and their origins.

The **medullary reticulospinal tract**, however, has the opposite effect; it liberates the antigravity muscles from reflex control. Activity in both reticulospinal tracts is controlled by descending signals from the cortex. A fine balance between them is required as the pitcher goes from standing on the mound to winding up and throwing the ball.

Figure 14.6 provides a simple summary of the major descending spinal tracts. The ventromedial pathways originate from several regions of the brain stem and participate mainly in the maintenance of posture and certain reflex movements. Initiation of a voluntary, ballistic movement, such as throwing a baseball, requires instructions that descend from the motor cortex along the lateral pathways. The motor cortex directly activates spinal motor neurons and also liberates them from reflex control by communicating with the nuclei of the ventromedial pathways. It is clear that the cortex is critical for voluntary movement and behavior, so we will now focus our attention there.

THE PLANNING OF MOVEMENT BY THE CEREBRAL CORTEX

Although cortical areas 4 and 6 are called *motor cortex*, it is important to recognize that the control of voluntary movement engages almost all of the neocortex. Goal-directed movement depends on knowledge of where the body is in space, where it intends to go, and on the selection of a plan to get it there. Once a plan has been selected, it must be held in memory until the appropriate time. Finally, instructions must be issued to implement the plan. To some extent, these different aspects of motor control are localized to different regions of the cerebral cortex. In this section, we explore some of the cortical areas implicated in motor planning. Later we'll look at how a plan is converted into action.



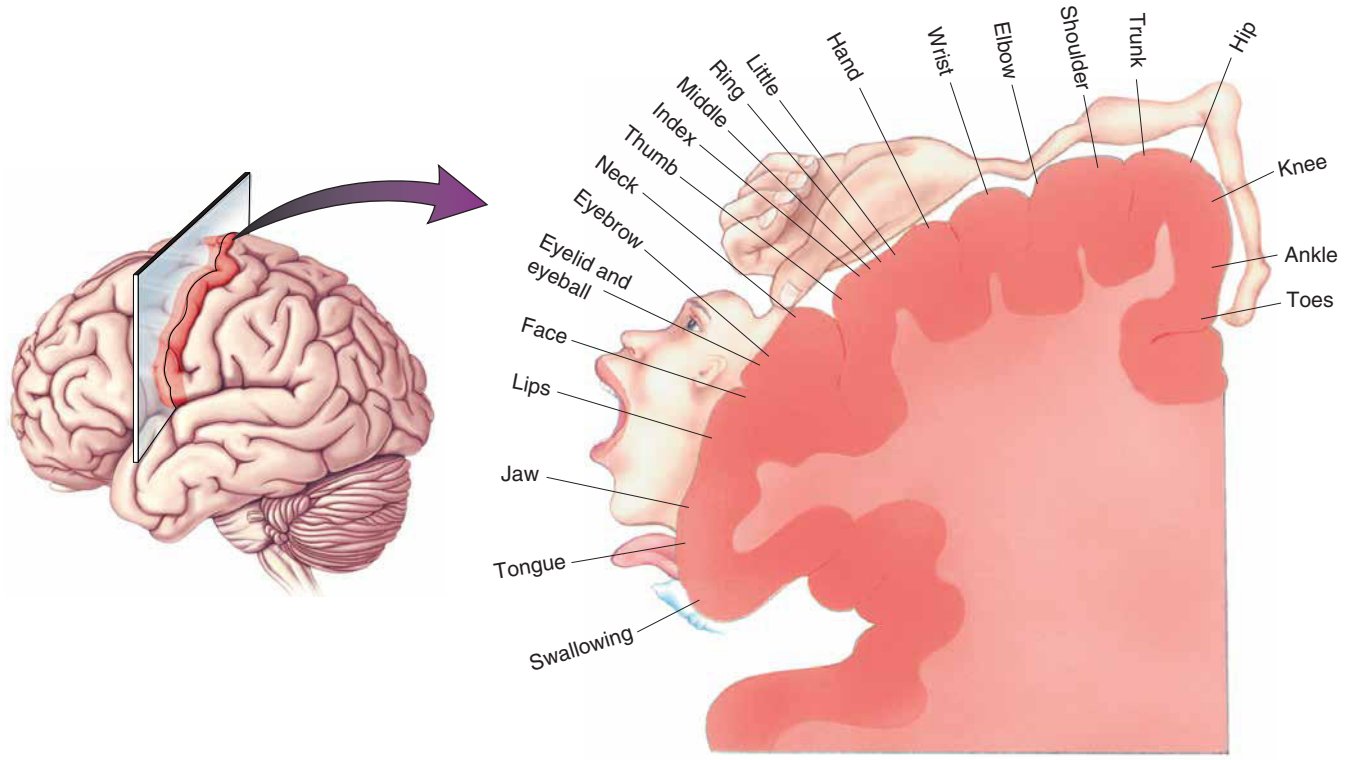
▲ FIGURE 14.7
Planning and directing voluntary movements. These areas of the neocortex are involved in the control of voluntary movement. Areas 4 and 6 constitute the motor cortex.

Motor Cortex

The motor cortex is a circumscribed region of the frontal lobe. Area 4 lies just anterior to the central sulcus on the precentral gyrus, and area 6 lies just anterior to area 4 (Figure 14.7). The definitive demonstration that these areas constitute motor cortex in humans came from the work of neurosurgeon Wilder Penfield. Recall from Chapter 12 that Penfield electrically stimulated the cortex in patients who were undergoing surgery to remove bits of brain thought to be inducing epileptic seizures. The stimulation was used in an attempt to identify which regions of cortex were so critical that they should be spared from the knife. In the course of these operations, Penfield discovered that weak electrical stimulation of area 4 in the precentral gyrus would elicit a twitch of the muscles in a particular region of the body on the contralateral side. Systematic probing of this region established that there is a somatotopic organization in the human precentral gyrus much like that seen in the somatosensory areas of the postcentral gyrus (Figure 14.8). Area 4 is now often referred to as **primary motor cortex** or **M1**.

The foundation for Penfield's discovery had been laid nearly a century before by Gustav Fritsch and Eduard Hitzig, who in 1870 had shown that stimulation of the frontal cortex of anesthetized dogs would elicit movement of the contralateral side of the body (see Chapter 1). Then, around the turn of the century, David Ferrier and Charles Sherrington discovered that the motor area in primates was located in the precentral gyrus. By comparing the histology of this region in Sherrington's apes with that of the human brain, Australian neuroanatomist Alfred Walter Campbell concluded that cortical area 4 is motor cortex.

Campbell speculated that cortical area 6, just rostral to area 4, might be an area specialized for skilled voluntary movement. Penfield's studies 50 years later supported the conjecture that this was a "higher" motor area in humans by showing that electrical stimulation of area 6 could



▲ FIGURE 14.8
A somatotopic motor map of the human precentral gyrus. Area 4 of the precentral gyrus is also known as *primary motor cortex* (M1).

evoke complex movements of either side of the body. Penfield found two somatotopically organized motor maps in area 6: one in a lateral region he called the **premotor area (PMA)** and one in a medial region called the **supplementary motor area (SMA)** (see Figure 14.7). These two areas appear to perform similar functions but on different groups of muscles. While the SMA sends axons that innervate distal motor units directly, the PMA connects primarily with reticulospinal neurons that innervate proximal motor units.

The Contributions of Posterior Parietal and Prefrontal Cortex

Recall the baseball player standing on the mound, preparing to pitch. It should be apparent that before the detailed sequence of muscle contractions for the desired pitch can be calculated, the pitcher must have information about the current position of his body in space and how it relates to the positions of the batter and the catcher. This mental body image seems to be generated by somatosensory, proprioceptive, and visual inputs to the posterior parietal cortex.

Two areas are of particular interest in the posterior parietal cortex: area 5, which is a target of inputs from the primary somatosensory cortical areas 3, 1, and 2 (see Chapter 12); and area 7, which is a target of higher order visual cortical areas such as MT (see Chapter 10). Recall that human patients with lesions in these areas of the parietal lobes, as can occur after a stroke, show bizarre abnormalities of body image and the perception of spatial relations. In its most extreme manifestation, the patient will simply neglect the side of the body, and even the rest of the world, opposite the parietal lesion.

The parietal lobes are extensively interconnected with regions in the anterior frontal lobes that in humans are thought to be important for abstract thought, decision making, and anticipating the consequences of action. These "prefrontal" areas, along with the posterior parietal cortex, represent the highest levels of the motor control hierarchy, where decisions are made about what actions to take and their likely outcome (a curve ball followed by a strike). The prefrontal cortex and parietal cortex both send axons that converge on cortical area 6. Recall that areas 6 and 4 together contribute most of the axons to the descending corticospinal tract. Thus, area 6 lies at the junction where signals encoding *what* actions are converted into signals that specify *how* the actions will be carried out.

This general view of higher order motor planning received dramatic support in a series of studies on humans carried out by Danish neurologist Per Roland and his colleagues. They used positron emission tomography (PET) to monitor changes in the patterns of cortical activation that accompany voluntary movements (see Box 7.3). When the subjects were asked to perform a series of finger movements from memory, the following regions of cortex showed increased blood flow: the somatosensory and posterior parietal areas, parts of the prefrontal cortex (area 8), area 6, and area 4. These are the very regions of the cerebral cortex that, as discussed earlier, are thought to play a role in generating the intention to move and converting that intention into a plan of action. Interestingly, when the subjects were asked only to mentally rehearse the movement without actually moving the finger, area 6 remained active but area 4 did not.

Neuronal Correlates of Motor Planning

Experimental work on monkeys further supports the idea that area 6 (SMA and PMA) plays an important role in the planning of movement, particularly complex movement sequences of the distal musculature. Using a method developed in the late 1960s by Edward Evarts at the National Institutes of Health, researchers have recorded the activity of neurons in the motor areas of awake, behaving animals (Box 14.2). Cells in the SMA typically increase their discharge rates about a second before the execution of a hand or wrist movement, consistent with their proposed role in planning movement (recall Roland's findings in humans). An important feature of this activity is that it occurs in advance of the movements of *either* hand, suggesting that the supplementary areas of the two hemispheres are closely linked via the corpus callosum. Indeed, movement deficits observed following an SMA lesion on one side, in both monkeys and humans, are particularly pronounced for tasks requiring the coordinated actions of the two hands, such as buttoning a shirt. In humans, a selective inability to perform complex (but not simple) motor acts is called *apraxia*.

You've heard the expression "ready, set, go." The preceding discussion suggests that readiness ("ready") depends on activity in the parietal and frontal lobes, along with important contributions from the brain centers that control levels of attention and alertness. "Set" may reside in the supplementary and premotor areas, where movement strategies are devised and held until they are executed. A good example is shown in Figure 14.9, based on the work of Michael Weinrich and Steven Wise at the National Institutes of Health. They monitored the discharge of a neuron in the PMA as a monkey performed a task requiring a specific arm movement to a target. The monkey was first given an *instruction stimulus* informing him what the target would be ("Get set, monkey!"), followed after a variable delay by a *trigger stimulus* informing the monkey that it was OK to move ("Go, monkey!"). Successful performance of the task (i.e., waiting for the "go" signal and

BOX 14.2 OF SPECIAL INTEREST

Behavioral Neurophysiology

Showing that a brain lesion impairs movement and that brain stimulation elicits movement does not tell us how the brain *controls* movement. To address this problem, we need to know how the activity of neurons relates to different types of voluntary movement in the intact organism. PET scans and fMRI are extremely valuable for plotting out the distribution of activity in the brain as behaviors are performed, but they lack the resolution to track the millisecond-by-millisecond changes in the activity of individual neurons. The best method for this purpose is extracellular recording with metal microelectrodes (see Box 4.1). But how is this done in awake, behaving animals?

This problem was solved by Edward Evarts and his colleagues at the National Institutes of Health. Monkeys were trained to perform simple tasks; when the tasks were performed successfully, the monkeys were rewarded with a sip of fruit juice. For example, to study the brain's guidance of hand and arm movements, the monkey might be trained to move its hand toward the brightest of several spots on a computer screen. Pointing to the correct spot earned it a juice reward. After training, the animals were anesthetized. In a simple surgical procedure, each monkey was fitted with a small headpiece so that a microelectrode could be introduced into the brain through a small opening in the skull. When the animals recovered from surgery, they showed no signs of discomfort from either the headpiece or the insertion of a microelectrode into the brain (recall from Chapter 12 that there are no nociceptors in the brain). Evarts and his colleagues then recorded the discharges of individual cells in the motor cortex as the animals made voluntary movements. In the example above, one could then see how the neuron's response changes when the animal points to different spots on the screen.

This is an example of what is now called *behavioral neurophysiology*, the recording of cellular activity in the brain of awake, behaving animals. By altering the task that the animal performs, the same method can be applied to the investigation of a wide range of neuroscientific topics, including attention, perception, learning, and movement. Some types of human neurosurgery are also done with the patient awake, at least during part of the procedure. By applying the techniques of behavioral neurophysiology to informed, consenting adults, we have also learned some fascinating information about uniquely human skills.

In recent years, technical developments have made it possible to insert large numbers of microelectrodes into the same or different parts of an animal's brain and to record from dozens or even hundreds of neurons simultaneously. This approach yields a massive amount of information about brain activity and its relationship to behavior. Understanding this relationship is one of the greatest challenges in neuroscience.

then making the movement to the appropriate target) was rewarded with a sip of juice. The neuron in the PMA began firing if the instruction was to move the arm to the left, and it continued to discharge until the trigger stimulus came on and the movement was initiated. If the instruction was to move to the right, this neuron did not fire (presumably another population of PMA cells became active under this condition). Thus, the activity of this PMA neuron reported the direction of the upcoming movement and continued to do so until the movement was made. Although we do not yet understand the details of the coding taking place in the SMA and PMA, the fact that neurons in these areas are selectively active well before movements are initiated is consistent with a role in planning the movement.

Mirror Neurons

We mentioned previously that some neurons in cortical area 6 respond not only when movements are executed but also when the same movement is only imagined—mentally rehearsed. Remarkably, some neurons in motor areas of cortex fire not only when a monkey makes a specific movement himself but also when the monkey simply observes another monkey, or even a human, making the same type of movement (Figure 14.10). These cells were called **mirror neurons** by Giacomo Rizzolatti and his colleagues when they discovered them in the PMA of monkeys at the University of Parma in the early 1990s. Mirror neurons seem to represent