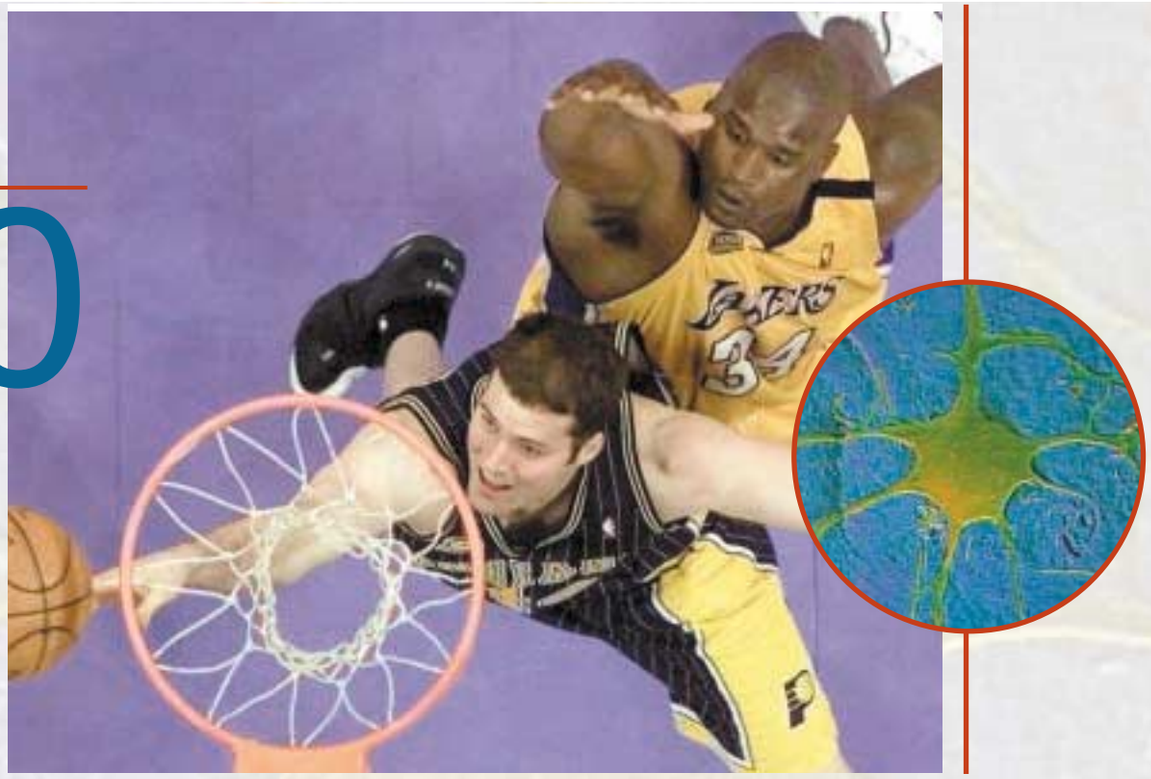
CHAPTER

10



How Does the Brain Produce Movement?

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The Somatosensory Cortex and Complex Movement

Kevork Djansezian/AP Photo
Micrograph: Dr. David Scott/Phototake

Kamala is a female Indian elephant that lives at the zoo in Calgary, Canada. Her trunk, which is really just a greatly extended upper lip and nose, consists of about 2000 fused muscles. A pair of nostrils runs its length and fingerlike projections are located at its tip. The skin of the trunk is soft and supple and is covered sparsely with sensory hairs. Like all elephants, Kamala uses her trunk for many purposes. It can gather food, scratch an ear, rub an itchy eye, or caress a baby. It can also be used to explore. Kamala raises it to sniff the wind, lowers it to examine the ground for scents, and sometimes even pokes it into another elephant's mouth to investigate the food there. She, like other elephants, can inhale as much as 4 liters of water into her trunk, which she can then place in her mouth to drink or squirt over her body to bathe. She can also inhale dust or mud for bathing. Kamala's trunk is a potential weapon, too. She can flick it as a threat, lash out with it in aggression, and throw missiles with it. Her trunk is both immensely strong and very agile. With it, Kamala can lift objects as large as an elephant calf, sometimes uprooting entire trees, yet this same trunk can grasp a single peanut from the palm of a proffered hand.

In one way, however, Kamala uses this versatile trunk very unusually for an elephant (Onodera & Hicks, 1999). She is one of only a few elephants in the world that paints with its trunk (Figure 10-1). Like many artists, she paints when it suits her, but nevertheless she has commemorated many important zoo events, such as the arrival of new species to the zoo. An elephant artist is not as far-fetched as the idea may at first seem. Other elephants, both in the wild and in captivity, pick up small stones and sticks and draw in the dust with them. But Kamala has gone well beyond this simple doodling. When given paints and a brush, she began to produce works of art, many of which have been sold to art collectors. Kamala, in fact, has achieved an international reputation as an artist.

A defining feature of animals is their ability to move. As the example of Kamala illustrates, even very skilled movements are not limited to humans. Although we humans display the most skilled motor control of all animals, members of many species have highly dexterous movements. This chapter explores how the brain produces movement. We begin by considering how the control of movement is organized. Then, we examine the various contributions of the neocortex, the brainstem, and the spinal cord to movement. Of particular interest is how neurons of the motor cortex take part in producing skilled movements. Next, we investigate how the basal ganglia and the cerebellum help to fine-tune our control of movement. Finally, we turn to the role of the somatosensory system. Although other senses, such as vision, play a part in enabling movement, body senses play a special role, as you will soon discover.

The Calgary Zoological Society

The Calgary Zoological Society

Figure 10-1
Kamala (her name means "lotus flower") was born in 1975 in Sri Lanka's Yala National Park and orphaned shortly thereafter. She was adopted by the Calgary, Alberta, Zoological Society. Elephants were first observed to paint with sticks or rocks in the dust, and some have become accomplished artists when given paints and a brush. Kamala began painting as part of an environmental enrichment program and her paintings are widely sold to collectors.

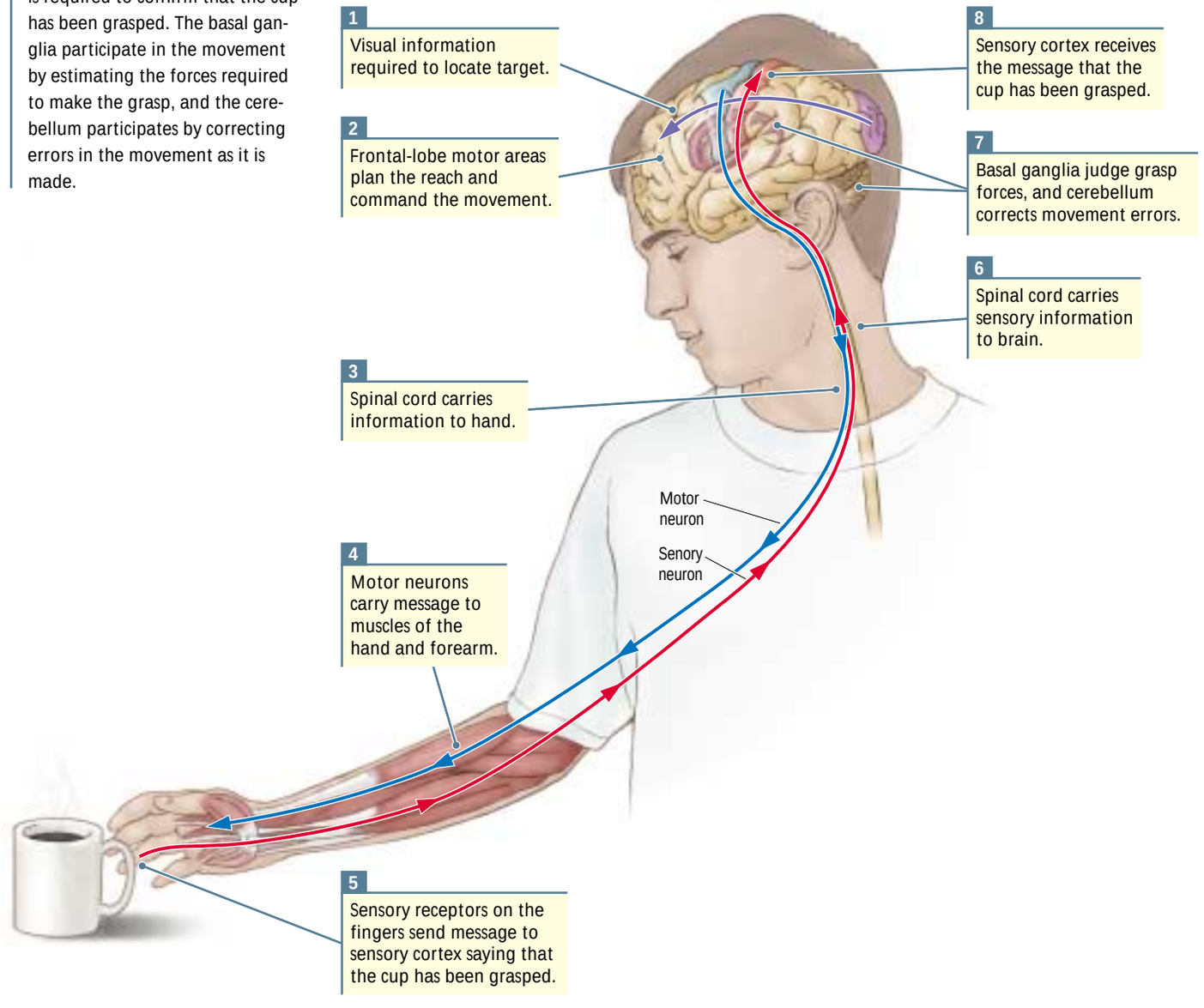
THE HIERARCHICAL CONTROL OF MOVEMENT

When Kamala paints a picture, her behaviors are sequentially organized. First, she looks at her canvas and her selection of paints; then, she considers what she wants to paint; and, finally, she executes her painting. These sequentially organized behaviors are dictated by the hierarchical organization of Kamala's nervous system. The major components of this nervous system hierarchy are the neocortex, the brainstem, and the spinal cord. All contribute to controlling the behaviors required to produce her artwork.

In the same way, your hierarchically organized nervous system controls every movement that you make. Figure 10-2 shows the sequences of steps taken when the human nervous system directs a hand to pick up a coffee mug. The visual system must first inspect the cup to determine what part of it should be grasped. This information is then relayed from the visual cortex to cortical motor regions, which plan and initiate the movement, sending instructions to the part of the spinal cord that controls the muscles of the arm and hand. As the handle of the cup is grasped, information from sensory receptors in the fingers travels to the spinal cord, and from there messages are sent to sensory regions of the cortex that control touch. The sensory cortex, in turn, in-

Figure 10-2

The brain tells the hand to reach, and the hand tells the brain that it has succeeded. Movements such as reaching for a cup require the participation of wide areas of the nervous system. The motor regions of the frontal lobe formulate the plan and command the movements required to reach for the cup. The message to the muscles is carried by pathways from the frontal lobe to the spinal cord. Motor neurons of the spinal cord carry the message to the muscles of the hand and arm. Sensory information from the visual system is required to direct the hand to the cup, and sensory information from sensory receptors in the hand is required to confirm that the cup has been grasped. The basal ganglia participate in the movement by estimating the forces required to make the grasp, and the cerebellum participates by correcting errors in the movement as it is made.



forms the motor cortex that the cup is now being held. Other regions of the brain also participate in controlling the movement, such as the basal ganglia, which help to produce the appropriate amount of force, and the cerebellum, which helps to regulate timing and corrects any errors in movement. Although at this point you probably will not remember all these various steps in controlling a movement, refer to Figure 10-2 when you reach the end of this chapter as a way of reviewing what you have learned. The important concept to remember right now is simply the hierarchical organization of the entire system.

The idea that the nervous system is hierarchically organized originated with the English neurologist John Hughlings-Jackson. He thought of the nervous system as being organized into a number of layers, with successively higher levels controlling more complex aspects of behavior by acting through the lower levels. The three major levels in Hughlings-Jackson's model are the same as those just mentioned for Kamala: the forebrain, the brainstem, and the spinal cord. Hughlings-Jackson also proposed that, within these divisions, further levels of organization could be found.

Hughlings-Jackson adopted the concept of hierarchical organization from evolutionary theory. He knew that the chordate nervous system had evolved in a series of steps: the spinal cord had developed in worms; the brainstem in fish, amphibians, and reptiles; and the forebrain in birds and mammals. Because each level of the nervous system had developed at different times, Hughlings-Jackson assumed that each must have some functional independence. Consequently, if higher levels of the nervous system were damaged, the result would be regression to the simpler behaviors of "lower" animals, a phenomenon that Hughlings-Jackson called dissolution. The brain-damaged person would still possess a repertoire of behaviors, but they would be more typical of animals that had not yet evolved the destroyed brain structure.

A hierarchically organized structure such as the mammalian nervous system, however, does not operate piece by piece. It functions as a whole, with the higher regions working through and influencing the actions of the lower ones. In the control of movement, many parts of the nervous system participate, with some regions engaged in sensory control, others in planning and commanding the movement, and still others in actually carrying the action out. To understand how all these various regions work together to produce even a simple movement, we will consider the major components of the hierarchy one by one, starting at the top with the forebrain.

The Forebrain and Movement Initiation

Complex movements, such as painting a work of art, include many components. For instance, your perceptions of what is appearing on the canvas must be closely coordinated with the brush strokes that your hand makes to achieve the desired effect. The same high degree of control is necessary for many other complex behaviors. Consider playing basketball. At every moment, decisions must be made and actions must be performed. Dribble, pass, and shoot are different categories of movement, and each can be carried out in numerous ways. Good players choose among the categories effortlessly and execute the movements seemingly without thought.

One explanation of how we control movements that was popular in the 1930s centers on the concept of feedback. It holds that, after we perform an action, we wait for feedback about how well that action has succeeded, and then we make the next movement accordingly. But Karl Lashley (1951), in an article titled "The Problem of Serial Order in Behavior," found fault with this explanation. Lashley argued that movements such as those required for playing the piano were performed too quickly to rely on feedback about one movement shaping the next movement. The time required to receive feedback about the first movement, combined with the time needed to develop a

Dissolution. The condition whereby disease or damage in the highest levels of the brain would produce not just loss of function, but a repertory of simpler behaviors as seen in animals that have not evolved that particular brain structure.

Motor sequence. A sequence of movements preprogrammed by the brain and produced as a unit.

plan for the subsequent movement and send a corresponding message to muscles, was simply too long to permit piano playing. Lashley suggested that movements must be performed as motor sequences, with one sequence being held in readiness while an ongoing sequence was being completed. According to this view, all complex behaviors, including playing the piano, painting pictures, and playing basketball, would require the selection and execution of multiple sequences of movements. As one sequence is being executed, the next sequence is being prepared so that the second can follow the first smoothly. Interestingly, Lashley's view seems to be borne out in how we execute speech. When people use complex sequences of words, they are more likely to pause and make "umm" and "ahh" sounds, suggesting that it is taking them more time than usual to organize their word sequences.

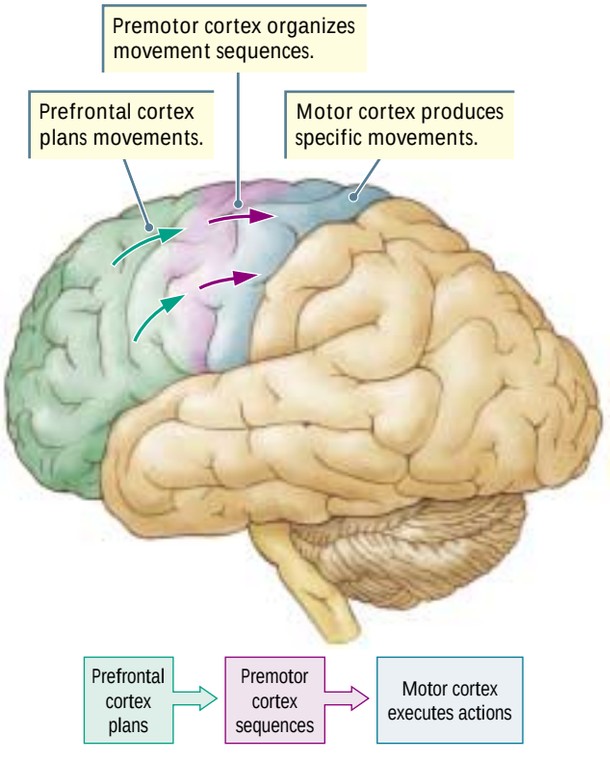


Figure 10-3

The prefrontal cortex of the frontal lobe plans movements. The premotor cortex organizes sequences of movements. The motor cortex executes specific movements. Information flow is from prefrontal to premotor cortex and then to motor cortex.

The frontal lobe of each hemisphere is responsible for planning and initiating sequences of behavior. The frontal lobe is divided into a number of different regions, including the three illustrated in Figure 10-3. From front to back, they are the *prefrontal cortex*, the *premotor cortex*, and the *primary motor cortex*.

A function of the prefrontal cortex is to plan complex behaviors. Such plans might be deciding to get up at a certain hour to arrive at work on time, deciding to stop at the library to return a book that is due, or deciding what kind of picture to paint for an art class. The prefrontal cortex does not specify the precise movements that should be made. It simply specifies the goal toward which movements should be directed.

To bring a plan to completion, the prefrontal cortex sends instructions to the premotor cortex, which produces complex sequences of movement appropriate to the task. If the premotor cortex is damaged, such sequences cannot be coordinated and the goal cannot be accomplished. For example, the monkey in Figure 10-4 has a lesion in the dorsal part of its premotor cortex. It has been given the task of extracting a piece of food wedged in a hole in a table (Brinkman, 1984). If it simply pushes the food with a finger, the food will drop to the floor and be lost. The monkey has to catch the food by holding a palm beneath the hole as the food is being pushed out. This animal is unable to make the two complementary movements together. It can push the food with a finger and extend an open palm, but it cannot coordinate these actions of its two hands.

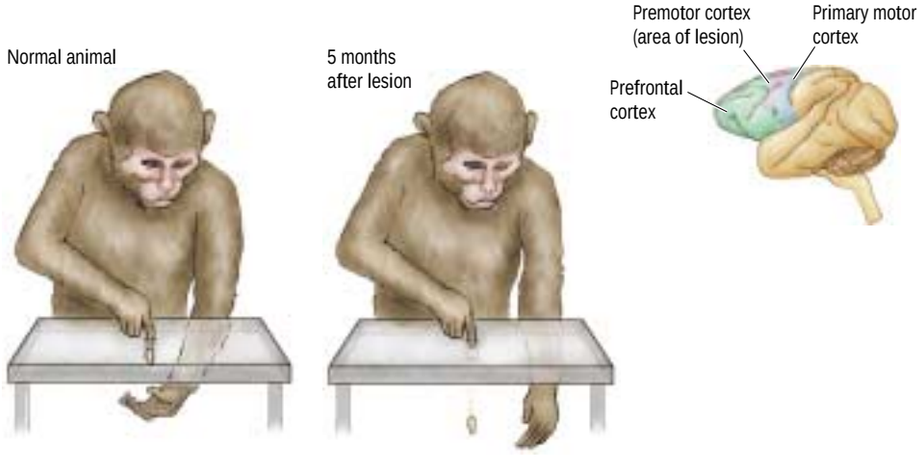


Figure 10-4

A unilateral lesion in the premotor cortex impairs performance by a monkey on a task requiring both hands. The normal monkey can push the peanut out of a hole with one hand and catch it in the other, but the experimental monkey is unable to do so.

Adapted from "Supplementary Motor Area of the Monkey's Cerebral Cortex: Short - and Long-Term Effects After Unilateral Ablation and the Effects of Subsequent Callosal Section," by C. Brinkman, 1984, *Journal of Neuroscience, 4,* p. 925.

Although the premotor cortex organizes movements, it does not specify the details of how each movement is to be carried out. Specifying the details is the responsibility of the primary motor cortex. The primary motor cortex is responsible for executing skilled movements. Its role can be seen by considering some of the movements that we use to pick up objects, illustrated in Figure 10-5. In using the pincer grip, we hold an object between the thumb and index finger. This grip not only allows small objects to be picked up easily, but also allows whatever is held to be used with considerable skill. In contrast, in using the power grasp (Figure 10-5B), we hold an object much less dexterously, by simply closing all of the fingers around it. Clearly, the pincer grip is a more demanding movement because the two fingers must be placed precisely on the object. People with damage to the primary motor cortex have difficulty correctly shaping their fingers to perform the pincer grip and so use the power grasp instead (Jeannerod, 1988).

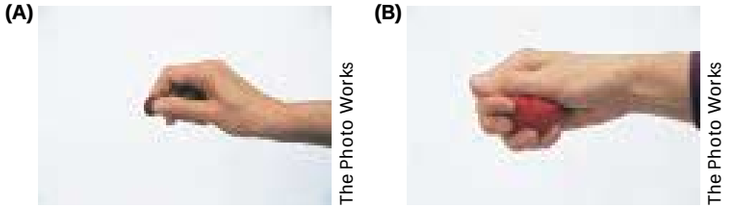


Figure 10-5

(A) In a pincer grip, an object is held between the thumb and index finger. **(B)** In a power grasp, also called a whole-hand grip, an object is held against the palm of the hand with the digits.

In summary, the frontal cortex executes precise movements, as well as planning them and coordinating different body parts to carry them out. The various regions of the frontal cortex that perform these functions are hierarchically related. After the prefrontal cortex has formulated a plan of action, it instructs the premotor cortex to organize the appropriate sequence of behaviors.

The hierarchical organization of frontal-lobe areas in producing movements is supported by studies of cerebral blood flow, which serves as an indicator of neural activity. Figure 10-6 shows the regions of the brain that were active when subjects in one such study were performing different tasks (Roland, 1993). When the subjects were tapping a finger, increases in blood flow were limited to the primary motor cortex. When the subjects were executing a sequence of finger movements, blood flow also increased in the premotor motor cortex. And when the subjects were using a finger to trace their way through a maze, a task that requires coordination of movements in relation to a goal, blood flow increased in the prefrontal cortex, too. Notice that blood flow did not increase throughout the entire frontal lobe as the subjects were performing these tasks. Blood flow increased only in those regions taking part in the required movements.

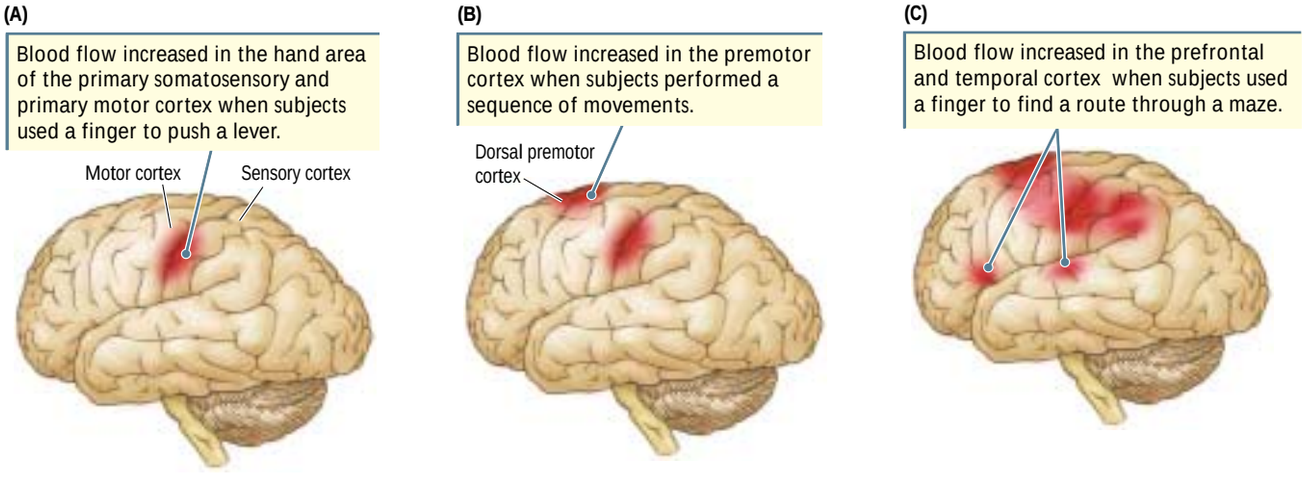


Figure 10-6

Blood flow increases in the cerebral cortex depend on the motor task that the subject performs. The pattern of activation supports the idea that simple motor movements are mainly controlled by the motor cortex, movements requiring sequencing are additionally controlled by the premotor cortex, and movements requiring planning are controlled by other cortical areas, including the prefrontal cortex and regions of the parietal and temporal cortex.

Adapted from *Brain Activation* (p. 63), by P. E. Roland, 1993, New York: Wiley-Liss.

EXPERIMENT

Question: What are the effects of brainstem stimulation under different conditions?

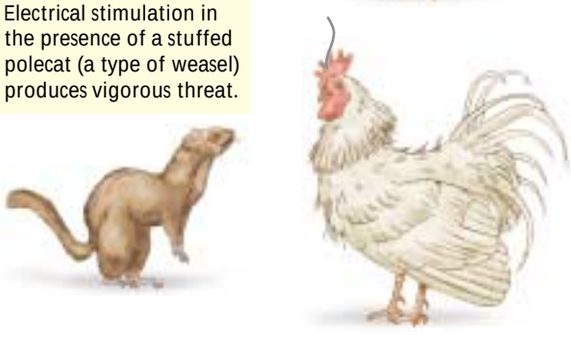
Procedures **Results**

Stimulating electrode in brainstem

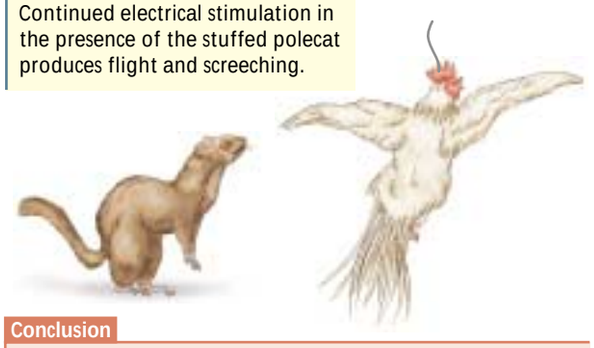
Electrical stimulation alone produces restless behavior.

Electrical stimulation and the presence of a fist produces slight threat.

Electrical stimulation in the presence of a stuffed polecat (a type of weasel) produces vigorous threat.



Continued electrical stimulation in the presence of the stuffed polecat produces flight and screeching.



Conclusion

Stimulation of some brainstem sites produces behavior that depends on context, suggesting that an important function of the brainstem is to produce appropriate species-typical behavior.

Figure 10-7

Electrical brain stimulation of the brainstem of a hen elicits adaptive behavior.

Adapted from *The Collected Papers of Erich von Holst* (p. 121), translated by R. Martin, 1973, Coral Gables, FL: University of Miami Press.

The Brainstem and Species-Typical Movement

Species-typical behaviors are actions displayed by every member of a species, such as the pecking of a robin, the hissing of a cat, or the breaching of a whale. In a series of studies, Swiss neuroscientist Walter Hess (1957) found that the brainstem controls species-typical behaviors. Hess developed the technique of implanting electrodes into the brains of cats and other animals and cementing them in place. These electrodes could then be attached to stimulating leads in the freely moving animal, without causing the animal much discomfort. By stimulating the brainstem, Hess was able to elicit almost every innate movement that the animal might be expected to make. For example, a resting cat could be induced to suddenly leap up with an arched back and erect hair as though frightened by an approaching dog. The movements elicited would begin abruptly when the stimulating current was turned on and end equally abruptly when the stimulating current was turned off. The behaviors were performed in a subdued manner when the stimulating current was low, but they increased in vigor as the stimulating current was turned up. The actions varied, depending on the site that was stimulated. Stimulation of some sites produced turning of the head, others produced walking or running, and still others elicited displays of aggression or fear, and so on. The reaction of the animal toward a particular stimulus could be modified accordingly. For instance, when shown a stuffed toy, a cat would respond to electrical stimulation of some sites by stalking the toy, whereas it would respond to stimulation of other sites with fear and withdrawal behavior.

Hess's experiments have been confirmed and expanded by other researchers using many different kinds of animals. For instance, Figure 10-7 shows the effects of brainstem stimulation on a chicken under various conditions (von Holst, 1973). Notice how the site stimulated interacts with both the presence of an object to react to and the length of stimulation. With stimulation of a certain site alone, the chicken displays only restless behavior. But, when a fist is displayed, the same stimulation elicits slight threatening behavior. When the object displayed is then switched from a fist to a stuffed polecat, the chicken responds with vigorous threats. Finally, with continued stimulation in the presence of the polecat, the chicken flees, screeching. Experiments such as these show that an important function of the brainstem is to produce species-typical behavior. Hess's experiments also gave rise to a sizable science fiction literature in which "mind control" induced by brain stimulation figures centrally in the plot.

Other functions of the brainstem are the control of movements used in eating and drinking and the control of movements used in sexual behavior. Animals can be induced to display these behaviors when certain areas of the brainstem are stimulated. An animal can even be induced to eat nonfood objects, such as chips of wood, if the part of the brainstem that triggers eating is sufficiently stimulated. The brainstem is also important for posture, for the ability to stand upright and to make coordinated movements of the limbs, for swimming and walking, and for movements used in grooming the fur and making nests.

Grooming provides an example of a particularly complex movement pattern that is coordinated mainly by the brainstem (Berridge, 1989). When grooming, a rat sits back on its haunches, licks its paws, wipes its nose with its paws, then wipes its paws across its face, and finally turns to lick the fur on its body. These movements are always performed in the same order. The next time you dry off after a shower or swimming, note the "grooming sequence" that you use. This human grooming sequence is very similar to the one that rats use.

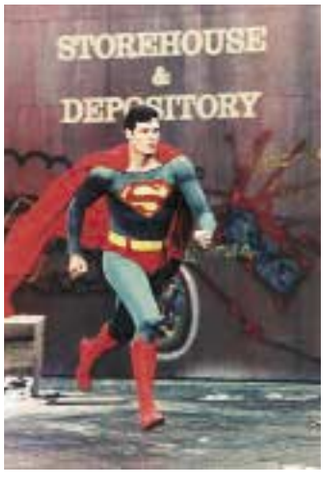
The effects of damage to regions of the brainstem that organize sequences of movement can be seen in a person with cerebral palsy. A disorder primarily of motor function, cerebral palsy is caused by brain trauma. The trauma usually occurs during fetal development or birth, but it can sometimes happen in early infancy, as it did in the case of E. S., whom we examined (see "Cerebral Palsy" on page 248).

> E. S. suffered a cold and infection when he was about 6 months old. Subsequently, he had a great deal of difficulty in making movements. As he grew up, his hands and legs were almost useless, and his speech was extremely difficult to understand. For most of his childhood, he was considered retarded and was sent to a special school. When he was 13 years old, the school bought a computer and one of his teachers attempted to teach him to use it by pushing the keys with a pencil that he held in his mouth. Within a few weeks, the teacher realized that E. S. was extremely intelligent and could communicate and complete school assignments on his computer. He was eventually given a motorized wheelchair that he could control with finger movements of his right hand. With the assistance of his computer and wheelchair, he soon became almost self-sufficient and eventually attended college, where he achieved excellent grades and became a student leader. On graduation with a major in psychology, he became a social worker and worked with children who suffered from cerebral palsy.

Clearly, a brain injury such as that causing cerebral palsy can be extremely damaging to movement, while leaving sensory abilities and cognitive capacities unimpaired. Damage to the brainstem can also cause changes in cognitive function, such as occurs in autism (see "Autism" on pages 362–363).

Cerebral palsy. A group of brain disorders that result from brain damage acquired perinatally.

Paraplegia. Paralysis of the legs due to spinal-cord injury.



Photofest

Ted Thai/*Time* magazine

Figure 10-8

Christopher Reeve portrayed Superman, as illustrated in this 1980 *Superman II* photograph. As a result of a fall from his jumping horse in 1995 in which he suffered damage at the C1–C2 level of the spinal cord, he has little movement below his neck. He is now active in raising money for spinal-cord research, and he still acts in movies.

The Spinal Cord and Movement Execution

On Memorial Day weekend in 1995, Christopher Reeve, a well-known actor who portrayed Superman, was thrown from his horse at the third jump of a riding competition in Culpeper, Virginia. Reeve's spinal cord was severed at the C1–C2 level, near the upper end of the spinal cord. This injury left his brain intact and functioning and the rest of his spinal cord intact and functioning, too, but his brain and spinal cord were no longer connected. As a result, other than movements of his head and slight movement in his shoulders, Reeve's body was completely paralyzed. He was even unable to breathe without assistance. Whereas only a few decades ago such a severe injury would have been fatal, modern and timely medical treatment allowed Reeve to survive. Reeve capitalized on his celebrity status to campaign for disabled people, fighting to prevent lifetime caps on compensation for spinal-cord injuries and raising money for spinal-cord research. As a result of this research, Reeve hopes to someday walk again.

In view of the complex behaviors that the brain produces, the spinal cord is sometimes considered simply a pathway for conveying information between the brain and the rest of the body. It does serve this function. If the spinal cord is severed, a person loses sensation and voluntary movements below the cut. If the cut is relatively low, the paralysis and loss of sensation are in the legs and lower body. This condition, called paraplegia, is described in "Paraplegia" on page 364. If a cut is higher on the spinal

Autism

Focus on Disorders

> Hi! I'm Chris Slater, a 17-year-old guy from Rice Lake, WI. I have High Functioning Autism. My page has been put up so I can teach people about autism, and to help anyone with any question about autism. When I was younger, I had the same characteristics as any other person with autism. Today, I'm very active. I have many friends, some have autism; some don't. I love to play Magic: The Gathering, a collectible card game, and some of my friends without autism play it also. I love to play golf, but I never play alone! What made this possible was that I was "educated" about people that don't have autism. What I mean by that is that I learned how they interact, and how they present themselves. I feel that every person with autism can, in fact, make it in this world—if they can learn how to socialize! (*http://slater.autistics.org/*)

Leo Kanner and Hans Asperger first used the term *autism* in the 1940s to describe children who suffered from a severe set of symptoms, including greatly impaired social interaction, a bizarre and narrow range of interests, and marked abnormalities in language and communication. Although some of these children were classified as mentally retarded, others had their intellectual functioning preserved. Because these children seemed to live in their own self-created worlds, the condition was named autism, from the Greek *autos,* meaning "self."

An estimated 1 of every 500 people has autism. Although it knows no racial, ethnic, or social boundaries, autism is four times as prevalent in boys as in girls. Many autistic children are noticeably different from birth. To avoid physical contact, these babies arch their backs and pull away from their caregivers or they become limp when held. But approximately one-third of autistic children develop normally until somewhere between 1 and 3 years of age. Then the autistic symptoms emerge.

One common characteristic of autism is a failure to interact socially with other people. Some autistic children do not relate to other people on any level. The attachments that they do form are to inanimate objects, not to other human beings. Another common characteristic of autism is an extreme insistence on sameness. Autistic children vehemently resist even small modifications to their surroundings or their daily routines. Objects must always be placed in exactly the same locations, and tasks must always be carried out in precisely the same ways. One possible reason for this insistence on sameness may be an inability to understand and cope with novel situations. Autistic children also have marked impairments in their language development. Many do not speak at all, and others repeat words aimlessly with little attempt to communicate or convey meaning. These children also exhibit what seem like endlessly repetitive body movements, such as rocking, spinning, or flapping the hands. In some cases, they may engage as well in aggressive or self-injurious behavior. The severity of these symptoms varies. Some autistic people are severely impaired, whereas others, like Chris Slater, can learn to function quite well. Still others may have exceptional abilities in certain areas, including music, art, and mathematics.

As might be expected of a disorder with as many symptoms as those of autism, anatomical studies reveal abnormal structures and cells in a number of brain regions, including

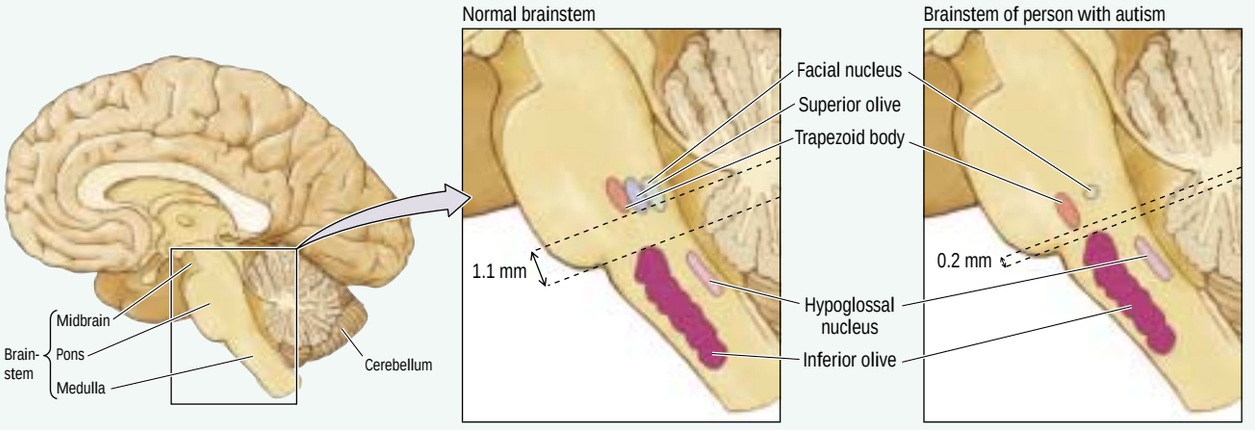
Visit the Web site at **www.worthpublishers.com/kolb/chapter10** for up-to-the-minute links to current research on spinal cord injury.

cord, as in Christopher Reeve's spinal cord, paralysis and loss of sensation can include the arms as well as the legs, a condition called quadraplegia.

In addition to its role in transmitting messages to and from the brain, the spinal cord is capable of producing many movements without any brain involvement. Movements that depend on the spinal cord alone are collectively called *spinal-cord reflexes*. Some of these reflexes entail the movement of limbs. For example, a light touch to the surface of the foot causes the leg to extend reflexively to contact the object that is touching it. This reflex aids the leg in contacting the ground to bear weight in walking. Other limb reflexes consist of withdrawal. For instance, a noxious stimulus applied to a hand causes the whole arm to reflexively pull back, thereby avoiding the injurious object.

Spinal circuits can also produce more complex movements than the simple ones just described. An example is the stepping movement in walking. If body weight is supported while the feet are in contact with a conveyor belt, the legs "walk" reflexively to

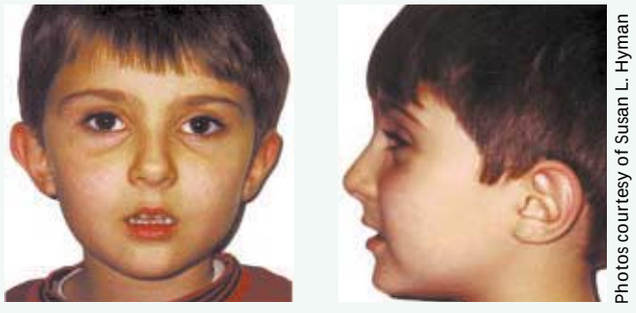
Quadraplegia. Paralysis of the legs and arms due to spinal-cord injury.



the limbic system and the cerebellum. Brain scans have indicated that the cerebellum may be smaller in people with autism than in control subjects.

The cause or causes of the neurological traits that give rise to autism are not yet understood. There is some indication of a genetic influence because both members of identical twins are more likely than those of fraternal twins to develop autism. There is also evidence that a virus can trigger the disorder. For instance, women have an increased risk of giving birth to an autistic child if they are exposed to rubella (German measles) in the first trimester of pregnancy. There is also a suspicion that autism can be caused by industrial toxins, but the evidence for this possibility is uncertain.

Patricia Roder (2000) suggests that one cause of autism may be an abnormality in the HOSA1 gene that plays a central role in the development of the brainstem. She has found that an area of the brainstem in the caudal part of the pons is small in autistic people, as the accompanying drawing shows. Several nuclei in this area, including the nucleus that controls facial muscles, are either small or missing, which may lead to subtle facial abnormalities.



Photos courtesy of Susan L. Hyman

(Upper) Autism's effects include changes to the brainstem in which the posterior part of the pons is reduced in size. Several nuclei in this region, including the facial nucleus, superior olive, and trapezoid body, are smaller than normal. **(Lower)** A child with autism is normal in appearance but may have some physical anomalies characteristic of the disorder. The corners of the mouth may be low compared with the upper lip, the tops of the ears may flop over (*left*), and the ears may be a bit lower than normal and have an almost square shape (*right*).

keep up with the belt. Each leg has its own neural circuit that allows it to step. When the limb is moved backward on the conveyor belt, causing the foot to lose support, the limb reflexively lifts off the belt and swings forward underneath the body. As the foot then touches the surface of the belt again, tactile receptors initiate the reflex that causes the leg to push against the surface and support the body's weight. In this way, several spinal reflexes work together to produce the complex movement of walking. Because this walking is reflexive, even a newborn baby will display it when held in the correct position on a conveyor belt.

One of the more complex reflexes that can be observed in other spinal animals is the scratch reflex. Here an animal reflexively scratches a part of its body in response to a tickle. The complexity of this reflex is revealed in the accuracy of the movement. Without direction from the brain, the tip of a limb, usually a hind limb, can be correctly directed to the part of the body that is irritated.

Scratch reflex

Scratch reflex. A reflex by which the hind limb removes a stimulus from the surface of the body.

Focus on Disorders

Paraplegia

Each year about 11,000 people in the United States and 1000 people in Canada suffer direct damage to the spinal cord. Often in these cases the spinal cord is completely severed, leaving the victim with no sensation or movement from the site of the cut downward. Although 12,000 people annually incurring spinal-cord injury may seem like a large number, it is small relative to the number who suffer other kinds of nervous system damage. Consequently, some of those with spinal-cord injury have become very active in campaigning for public awareness of their condition and for research into possible treatments for it.

Currently, two people who are especially active in this campaign are Christopher Reeve, the actor who once played Superman, and Rick Hansen, a Canadian. Hansen was an athletic teenager when he became a paraplegic as the result of a lower thoracic spinal injury in 1975. Twelve years later, to raise public awareness of the potential of people with disabilities, Rick wheeled himself 40,000 kilometers around the world, generating more than $24 million for the Man in Motion Legacy Trust Fund. To date, this fund has contributed more than $100 million in support of spinal-cord research, rehabilitation, wheelchair sports, and public-awareness programs. Rick Hansen is currently executive director of the Rick Hansen Institute at the University of British Columbia, which provides leadership and support for initiatives in the field of disability, with a special focus on spinal-cord injury.

Research to find treatments for spinal-cord damage is a frustrating field. A severed spinal cord, like a severed electrical cord, entails just a single cut that leaves the machinery on both sides of it intact. If only the cut could somehow be bridged, sensory and motor function might be restored. But the solution is not so easy. Several factors prevent nerve fibers from growing across a cut in a spinal cord. These factors include the formation of scar tissue, the lack of a blood supply, the absence of appropriate growth factors to stimulate growth, and the fact that normal tissue at the edge of the cut actively repels regrowth. Can these obstacles somehow be overcome?

Studies suggest that it may be possible to induce neural fibers to grow across a spinal-cord cut. For instance, if the spinal cord in chicks and other baby animals is cut in the first 2 weeks of life, the spinal cord regrows and apparently normal function returns. Presumably, if the mix of growth factors that enables this spinal-cord regeneration could be identified and applied to the severed spinal cords of adults, the same regrowth could result. Also encouraging is the fact that, when a nerve fiber in the peripheral nervous system is cut, it regrows no matter how old the injured person is. The Schwann cells that form the severed axon's myelin are thought to produce the chemical environment that facilitates this regrowth. This finding has led to experiments in which Schwann cells are implanted into a cut spinal cord. The results have been positive, although no cure has been effected. Other investigators have built little bridges across a severed spinal cord and also have obtained some encouraging evidence of regrowth. Rats that had been unable to move their legs regained postural support and were able to step after receiving this treatment. From a theoretical and experimental perspective, obtaining spinal-cord regeneration and recovery seems an achievable goal.

Courtesy of Nike/Rick Hansen Institute

Rick Hansen on the Man in Motion Tour.

In humans and other animals with a severed spinal cord, spinal reflexes still function, even though the spinal cord is cut off from communication with the brain. As a result, there may be spontaneous movements or spasms in the paralyzed limbs. But the brain can no longer guide the timing of these reflexes. Consequently, reflexes related to bladder and bowel control may need to be artificially stimulated by caregivers.

In Review

The motor system is organized hierarchically. The forebrain, especially the frontal lobe, is responsible for selecting plans of action, coordinating body parts to carry out those plans, and executing precise movements. The brainstem, in contrast, is responsible for species-typical movements, for actions related to survival such as eating, drinking, and sexual behavior, and for posture and walking. Finally, in addition to being a pathway between the brain and the rest of the body, the spinal cord is independently responsible for reflexive movements. Although lower-level functions in this hierarchical system can continue in the absence of higher-level ones, the higher levels are what provide voluntary control over movements. Consequently, when the brain is disconnected from the spinal cord, there is no way to control movement at will.

THE ORGANIZATION OF THE MOTOR SYSTEM

If we compare how Kamala paints a picture with her trunk with how human artists do so with their hands, it may seem remarkable that such different behavioral strategies could be used to achieve the same goal. The use of different body parts for skilled movements is widespread among animals. For instance, dolphins and seals are adept at using their noses to carry and manipulate objects, and many other animals, including domestic dogs, accomplish the same end by using their mouths. Among birds, the beak is often specially designed for getting food, for building nests, and sometimes even for making and using tools. Tails also are useful appendages. Some marsupials and some species of New World primates can pick up and carry objects with them. Among horses, the lips are dexterous enough to manipulate things. Using its lips, a horse can select a single blade of grass of the type that it prefers from a patch of vegetation. Although humans tend to rely primarily on their hands for manipulating objects, they can still learn to handle things with other body parts, such as the mouth or a foot, if they have to. Some people without arms have become extremely proficient at using a foot for writing or for painting, for example.

What are the properties of the motor system that allow such versatility in carrying out skilled movements? In the next section, we will find the answer to this question by examining the organization of the motor cortex and its descending pathways to the brainstem and spinal cord, which in turn connects with the muscles of the body. We will then consider how the electrical activity of neurons executes skilled movements. Finally, we will look at some differences in the motor cortices of different animal species that may be related to their specialized movement abilities.

Link to the area on the organization of the motor system in the module on Control of Movement on your CD for a review of the organization of movement and motor systems.

The Motor Cortex

In 1870, two Prussian physicians, Gustav Fritsch and Eduard Hitzig, electrically stimulated the neocortex of an anesthetized dog and produced movements of the mouth, limbs, and paws on the opposite side of the dog's body. This was the first direct

evidence that the neocortex could control movement. Later researchers confirmed the finding by using a variety of animals as subjects, including primates such as monkeys.

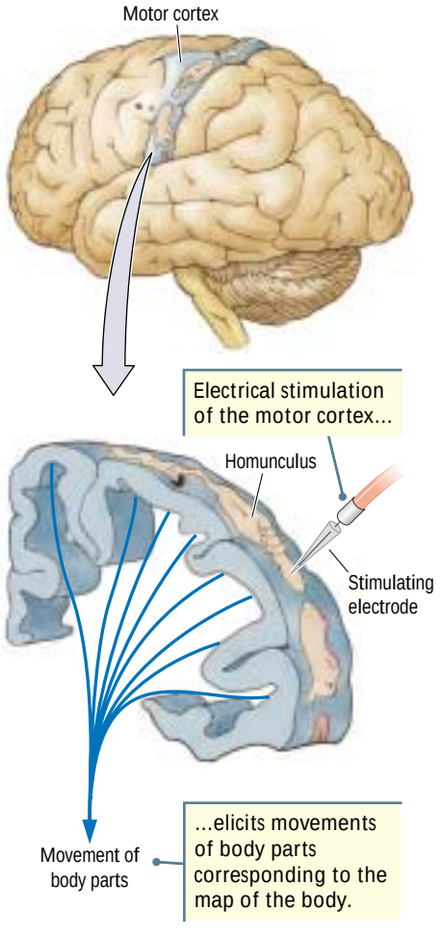


Figure 10-9

Penfield's homunculus. Electrical stimulation, in conscious human patients, of the motor cortex (precentral gyrus, or Brodmann's area 4) elicits movement of the body parts corresponding to the map of the body. Movements are topographically organized so that stimulation of the dorsal medial regions of the cortex produces movements in the lower limbs, and stimulation in ventral regions of the cortex produces movements in the upper body, hands, and face.

Go to the area on the primary motor cortex in the module on the Control of Movement on your CD for a more detailed analysis of the motor homunculus. Notice the exaggerated body parts associated with fine motor control.

Then, in the 1950s, Wilder Penfield used electrical stimulation to map the cortex of conscious human patients who were about to undergo neurosurgery. (See the discussion of Penfield's techniques in Chapter 9.) He and his colleagues found that movements were triggered mainly in response to stimulation of the primary motor cortex (also known as *Brodmann's area 4* or the *precentral gyrus*). Penfield summarized his results by drawing cartoons of body parts to represent the areas of the primary motor cortex that produce movement in those parts. The result was a **homunculus** (little person) that could be spread out across the motor cortex, as illustrated in Figure 10-9. Because the body is symmetrical, an equivalent motor homunculus is represented in the cortex of each hemisphere. Penfield also identified another smaller motor homunculus in the dorsal premotor area of each frontal lobe, a region sometimes referred to as the supplementary motor cortex.

The most striking feature of the motor homunculus is the disproportionate relative sizes of its body parts compared with the relative sizes of actual parts of the body. This distinctive feature is even more clearly illustrated in some of the artistic renditions of the homunculus that others scientists have made, one of which is shown in Figure 10-10. As you can see, the homunculus has very large hands with an especially large thumb. It also has very large lips and a large tongue. In contrast, the trunk, arms, and legs, which constitute most of the area of a real body, are much smaller in relative size. These size distortions illustrate the fact that large parts of the motor cortex regulate the hands, fingers, lips, and tongue, giving us precise motor control over these body parts. Areas of the body over which we have much less motor control have a much smaller representation in the motor cortex.

The British Museum, Natural History

Figure 10-10

An artistic representation of the cortical homunculus illustrates the disproportionate areas of the sensory and motor cortex that control different parts of the body.

Another distinctive feature of the homunculus when it is laid out across the motor cortex is that the body parts are arranged somewhat differently than in an actual body. For instance, the area of the cortex that produces eye movements is located in front of the homunculus's head. The head is oriented with the chin up and the forehead down, with the tongue located below the forehead. But such details aside, the homunculus is still a useful concept for understanding the **topographic organization** (functional layout) of the primary motor cortex. It shows at a glance that relatively larger areas of the brain control the parts of the body that are able to make the most skilled movements.

The discovery of the topographical representation of the motor cortex suggested how movements might be produced. Information from other regions of the neocortex could be sent to the motor homunculus, and neurons in the appropriate part of the homunculus could then execute the movements called for. If finger movements are

needed, for example, messages could be sent to the finger area of the motor cortex, triggering the required activity there. If this model of how the motor system works is correct, damage to any part of the homunculus would result in loss of movements in the corresponding part of the body.

Although the general idea underlying this model is right, more detailed mapping of the motor cortex and more detailed studies of the effects of damage to it indicate that the picture is a bit more complex. When researchers investigated the motor cortex in nonhuman primates, with the use of smaller electrodes than those used by Penfield to examine his patients, they discovered as many as 10 motor homunculi (Galea & Darian-Smith, 1994). As many as 4 representations of the body may exist in the primary motor cortex, and a number of other representations may be found in the premotor cortex. What each of these different homunculi does is still unclear. Perhaps each is responsible for a particular class of movements. Whatever the functions turn out to be, they will have to be determined by future research.

Homunculus. The representation of the human body in the sensory or motor cortex; also any topographical representation of the body by a neural area.

Topographic organization. A neural spatial representation of the body or areas of the sensory world perceived by a sensory organ.

The Corticospinal Tracts

The main pathways from the motor cortex to the brainstem and spinal cord are called the corticospinal tracts. (The term *corticospinal* indicates that these tracts begin in the neocortex and terminate in the spinal cord.) The axons of the corticospinal tracts originate mainly in layer-V pyramidal cells of the motor cortex, although axons also come from the premotor cortex and sensory cortex. The axons from the motor cortex descend into the brainstem, sending collaterals to a few brainstem nuclei and eventually emerging on the brainstem's ventral surface, where they form a large bump on each side of that surface. These bumps, known as pyramids, give the corticospinal tracts their alternate name, the pyramidal tracts. At this point, some of the axons descending from the left hemisphere cross over to the right side of the brainstem, and some of the axons descending from the right hemisphere cross over to the left side of the brainstem. The rest of the axons stay on their original sides. This division produces two corticospinal tracts entering each side of the spinal cord. Figure 10-11 illustrates the division of axons for the tract originating in the left-hemisphere cortex. The dual tracts on each side of the brainstem then descend into the spinal cord.

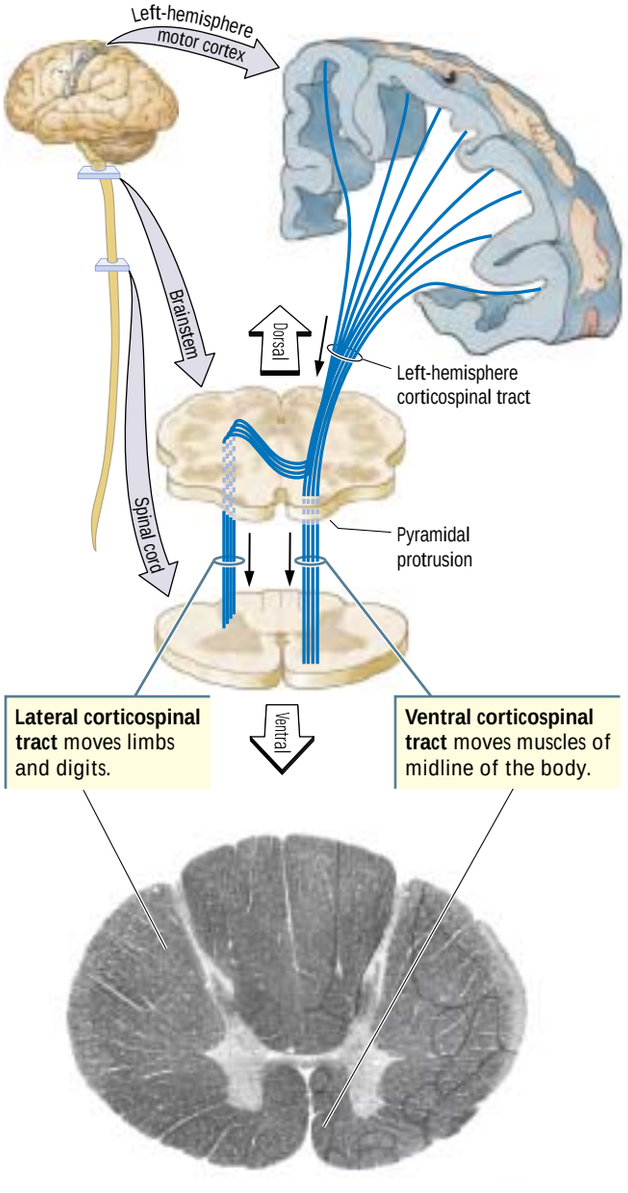


Figure 10-11

The corticospinal (from cortex to spinal cord) tracts descend from the motor cortex to the brainstem. Their location in the lower brainstem produces a protrusion (a pyramid) on the ventral surface of the brain. A tract from each hemisphere (only that from the left hemisphere is shown) divides into a lateral spinothalamic tract, which crosses the midline to the other side of the spinal cord, and a ventral spinothalamic tract, which remains on the same side. Fibers in the lateral spinothalamic tract are represented by the limbs and digits of the cortical homunculus and are destined to move muscles of the limbs and digits. Fibers of the ventral spinothalamic tract are represented by the midline of the homunculus's body and are destined to move muscles of the midline of the body.

Photo of spinal cord reproduced from *The Human Brain: Dissections of the Real Brain* by T. H. Williams, N. Gluhbegovic, and J. Jew, on CD-Rom. Published by Brain University, brain-university.com 2000.

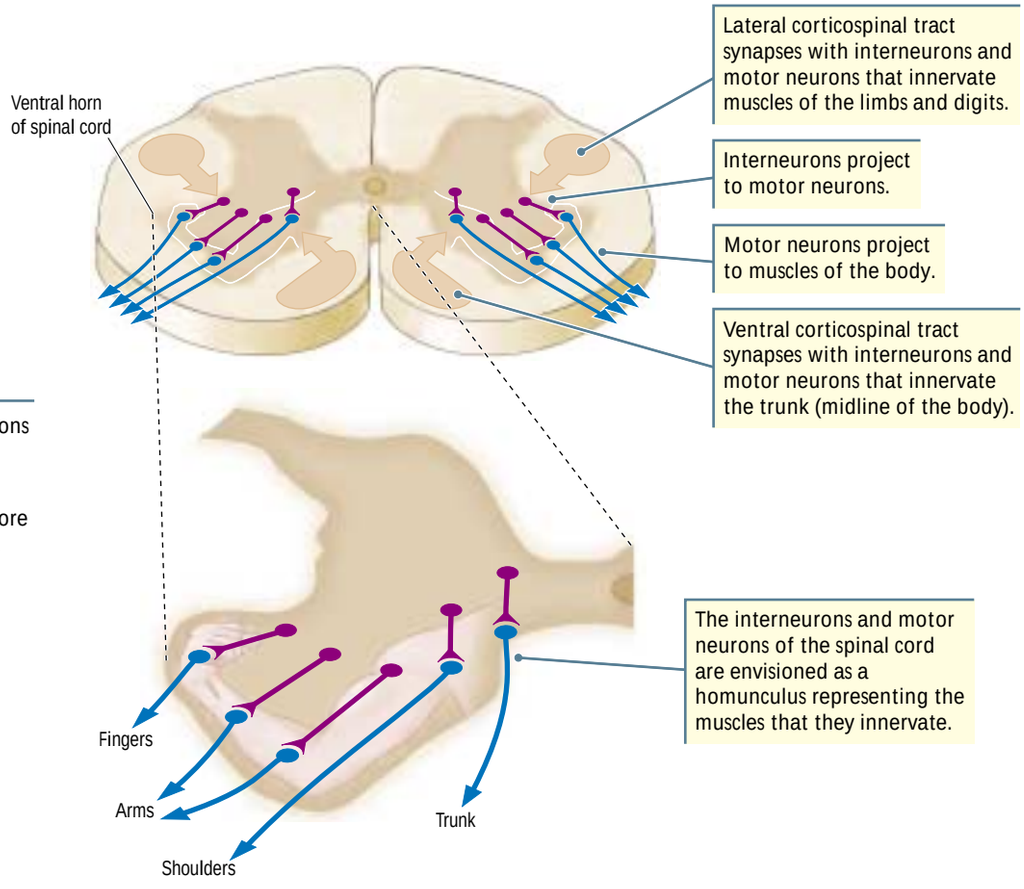


Figure 10-12

The interneurons and the motor neurons of the ventral spinal cord are topographically arranged so that the more lateral neurons innervate the more distal parts of the limbs and the more medial neurons innervate the more proximal muscles of the body.

Click on the area on descending motor tracts in the module on the Control of Movement on your CD for a visual overview of the corticospinal tracts.

In looking at the cross section of the spinal cord in Figure 10-12, you can see the location of the two tracts on each side. Those fibers that cross to the opposite side of the brainstem descend the spinal cord in a lateral position, giving them the name lateral corticospinal tract. Those fibers that remain on their original side of the brainstem continue down the spinal cord in a ventral position, giving them the name ventral corticospinal tract.

The Motor Neurons

The spinal-cord motor neurons that connect to muscles are located in the spinal cord's ventral horns. Interneurons lie just medial to the motor neurons and project onto them. The fibers from the corticospinal tracts make synaptic connections with both the interneurons and the motor neurons, but all nervous system commands to the muscles are carried by the motor neurons. Figure 10-12 shows that the more laterally located motor neurons project to muscles that control the fingers and hands, whereas intermediately located motor neurons project to muscles that control the arms and shoulders. The most medially located motor neurons project to muscles that control the trunk. The lateral corticospinal tract axons connect mainly with the lateral motor neurons, and the ventral corticospinal tract axons connect mainly to the medial motor neurons.

To picture how the motor homunculus in the cortex is related to motor neurons in the spinal cord, imagine placing your right index finger on the index-finger region of the motor homunculus on the left side of the brain and then following the axons of the cortical neurons downward. Your route takes you through the brainstem, across its midline, and down the lateral corticospinal tract, ending on interneurons and motor

neurons in the most lateral region of the spinal cord's right ventral horn—the horn on the opposite side of the nervous system from which you began. If you next follow the axons of these motor neurons, you will find that they synapse with muscles that move the index finger on that same right-hand side of the body. (By the way, the neurons that your brain is using to carry out this task are the same neurons whose pathway you are tracing.) If you repeat the procedure but this time trace the pathway from the trunk of the motor homunculus on the left side of the brain, you will follow the same route through the upper part of the brainstem. However, you will not cross over to the brainstem's opposite side. Instead, you will descend into the spinal cord on the same side of the nervous system as that on which you began (the left side), eventually ending up in the most medially located interneurons and motor neurons of that side's ventral horn. Finally, if you follow the axons of these motor neurons, you will end up at their synapses with muscles that move the trunk on the left side of the body.

This imaginary exercise should help you to remember the routes taken by the axons of the motor system. The limb regions of the motor homunculus contribute most of their fibers to the lateral corticospinal tract. Because these fibers have crossed over to the opposite side of the brainstem, they activate motor neurons that move the arm, hand, leg, and foot on *the opposite side of the body*. In contrast, the trunk regions of the motor homunculus contribute their fibers to the ventral corticospinal tract and, because these fibers do not cross over at the brainstem, they activate motor neurons that move the trunk on *the same side of the body*. In short, the neurons of the motor homunculus in the left-hemisphere cortex control the trunk on the body's left side and the limbs on the body's right side. Similarly, neurons of the motor homunculus in the right-hemisphere cortex control the trunk on the body's right side and the limbs on the body's left side (Kuypers, 1981).

This description of motor-system pathways descending from the brain is a simplified one. There are actually about 26 pathways, including the corticospinal tracts. The other pathways carry instructions from the brainstem, such as information related to posture and balance, and control the autonomic nervous system. For all of these functions, however, the motor neurons are the final common path.

The Control of Muscles

The muscles with which spinal-cord motor neurons synapse control movement of the body. For example, the biceps and triceps of the upper arm control movement of the lower arm. Limb muscles are arranged in pairs, as shown in Figure 10-13. One member of a pair, the extensor, extends the limb away from the trunk. The other member of the pair, the flexor, moves the limb toward the trunk. Connections between the interneurons and motor neurons of the spinal cord ensure that the muscles work together so that, when one muscle contracts, the other relaxes. As you know, the neurotransmitter at the motor neuron– muscle junction is acetylcholine.

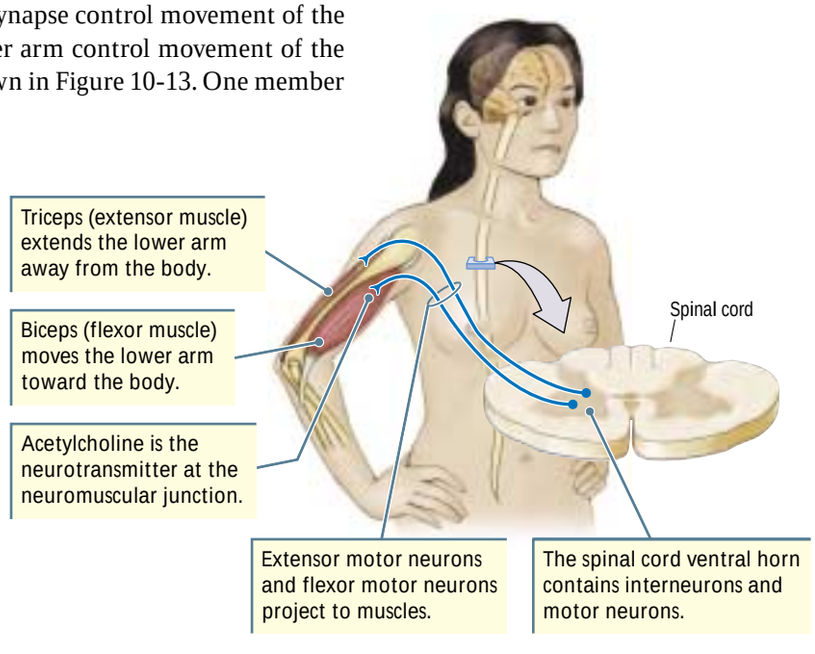


Figure 10-13

Motor neurons of the ventral horn of the spinal cord project to extensor muscles (which move limbs away from the body) and flexor muscles (which move limbs toward the body).

In Review

The motor cortex is topographically organized as a homunculus in which parts of the body that are capable of the most skilled movements (especially the mouth, fingers, and thumb) are regulated by relatively larger cortical regions. Instructions regarding movement travel from the motor cortex through the corticospinal tracts to interneurons and motor neurons in the ventral horn of the spinal cord. The ventral corticospinal tracts carry instructions for trunk movements, whereas the lateral corticospinal tracts carry instructions for arm and finger movements. The axons of motor neurons in the spinal cord then carry instructions to muscles.

THE MOTOR CORTEX AND SKILLED MOVEMENTS

Baseball pitcher winding up

There is remarkable similarity in the way that people perform skilled movements. For instance, most people who reach for a small paperclip on a desk do so with the hand rotated so that the fingers are on the top and the thumb is on the side. They also use the pincer grip to hold the clip—that is, they grasp it between the thumb and index finger. These movements could be learned by watching other people use them, but we have no recollection of having, as children, spent any time observing and mastering such movement patterns. In fact, at about 12 months of age, babies simply spontaneously begin to use the pincer grip to pick up tiny objects such as breadcrumbs. Most other primates use this same grip pattern. All of the evidence therefore suggests that this skilled movement and many others are not learned but, instead, are innate. They are encoded in the neural connections of the motor cortex as basic patterns of movement that are common to the particular species. These patterns are known as synergies. In this section, we will see how neurons produce such synergies. We will also see how the motor cortices of other species produce skilled movements in these species, including the highly dexterous movements of an elephant's trunk.

Investigating Neural Control of Skilled Movements

Visit the area on control of movement in the module on the Control of Movement in your CD for more detail on the role of the central nervous system.

Apostolos Georgopoulos and his coworkers (1999) investigated the neural control of movement by recording from neurons in the motor cortices of monkeys that had been trained to make specific finger movements. They expected that, when a thumb or a certain finger moved, only the area of the motor cortex that represented that particular digit would be active. But this is not what happened. When one finger moved, not only were neurons in that finger's area of the motor cortex active, but so were neurons in the cortical areas of other fingers. Apparently, the entire hand's representation in the motor cortex participates even in simple acts, such as moving one finger. Although at first this finding may seem surprising, it makes intuitive sense. After all, to move one finger, some effort must be exerted to keep the other fingers still. There must be connections between all of the participating neurons to allow them to act in concert. These same connections would be necessary for sequential movements of the fingers, such as those used in playing the piano or painting a work of art.

In another study designed to investigate how the motor cortex controls movements, E. V. Evarts (1968) used the simple procedure illustrated in Figure 10-14. He trained a monkey to flex its wrist in order to move a bar to which weights of different heaviness could be attached. An electrode implanted in the wrist region of the motor cortex recorded the activity of neurons there. Evarts discovered that these neurons began to discharge even before the monkey flexed its wrist. Apparently, they took part

Synergy. A pattern of movement that is coded by the motor cortex.

in planning the movement as well as initiating it. The neurons also continued to discharge as the wrist moved, confirming that they played a role in producing the movement. Finally, the neurons discharged at a higher rate when the bar was loaded with a weight. This finding showed that motor-cortex neurons increase the force of a movement by increasing their rate of firing.

Evarts's experiment also revealed that the motor cortex has a role in specifying the direction of a movement. The neurons of the motor-cortex wrist area discharged when the monkey flexed its wrist inward but not when the wrist was extended back to its starting position. These on–off responses of the neurons, depending on whether the

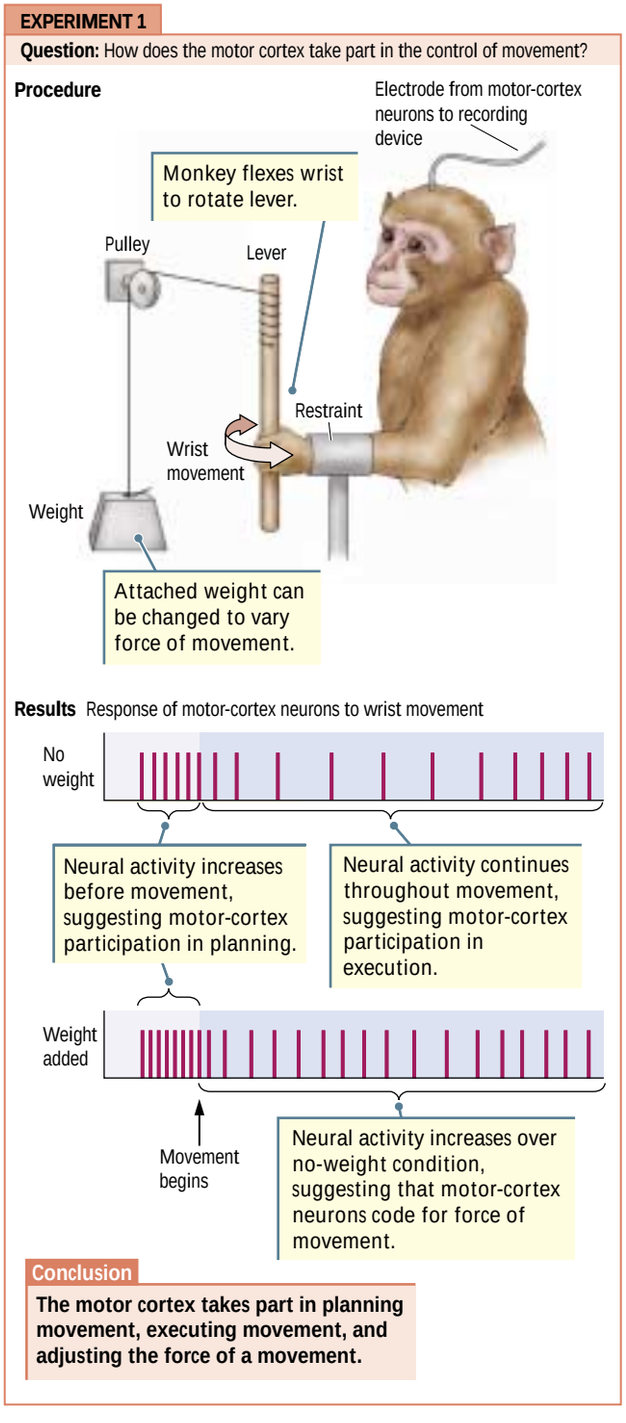


Figure 10-14

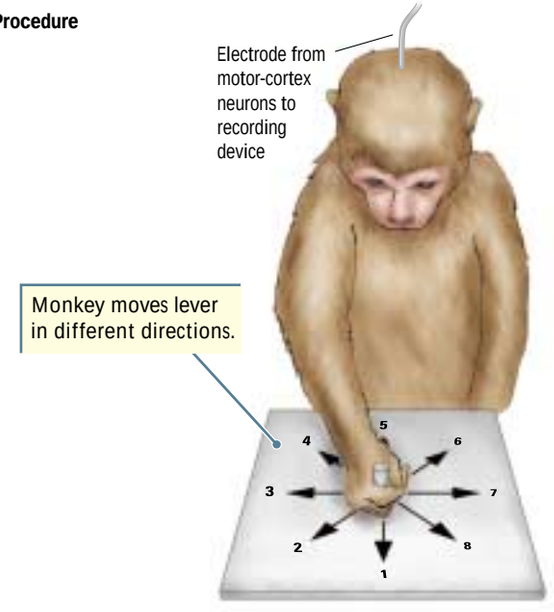
This experiment demonstrates that motor-cortex neurons take part in planning movements, executing movements, and regulating movement force and duration.

Adapted from "Relation of Pyramidal Tract Activity to Force Exerted During Voluntary Movement," by E. V. Evarts, 1968, *Journal of Neurophysiology, 31,* p. 15.

EXPERIMENT

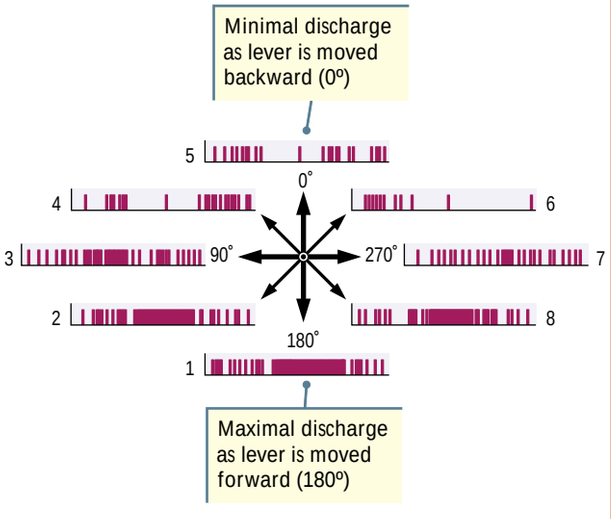
Question: What is the activity of a motor-cortex neuron during changes in the direction of movement?

Procedure



Results

Activity of a single motor-cortex neuron



Conclusion

The firing of individual motor-cortex neurons is tuned to the direction of a movement.

Figure 10-15

Individual motor-cortex neurons are maximally responsive to movements in a particular direction.

Adapted from "On the Relations Between the Direction of Two-Dimensional Arm Movements and Cell Discharge in Primate Motor Cortex," by A. P. Georgopoulos, J. F. Kalaska, R. Caminiti, and J. T. Massey, 1982, *Journal of Neuroscience, 2,* p. 1530.

flexor or extensor muscle is being used, are a simple way of coding the direction in which the wrist is moving.

Georgopoulos (1993) and his coworkers used a method similar to that of Evarts to further examine the coding of movement direction. They trained monkeys to move a lever in different directions across the surface of a table. Recording from single cells in the arm region of the motor cortex, they found that each cell was maximally active when the monkey moved its arm in a particular direction. Figure 10-15 summarizes the results. As the monkey's arm moved in directions other than the one to which a particular cell maximally responded, the cell would decrease its activity in proportion to the displacement from the "preferred" direction. For example, if a neuron discharged maximally as the arm moved directly forward, its discharge would be halved if the arm moved to one side, and discharge would cease altogether if the arm moved backward. According to Georgopoulos and his coworkers, the motor cortex seems to calculate both the direction and the distance of movements. Each neuron in a large population of motor-cortex neurons could participate in producing a particular movement, just as other studies have suggested. But the discharge rate of a particular neuron would depend on that movement's direction.

Georgopoulos proposed a different hypothesis from that of Evarts about how the motor cortex exerts control over movement. Both researchers believe that motor-cortex neurons plan and execute movements, but they disagree about what those plans and execution strategies entail. To better understand the difference between their hypotheses, imagine that you are preparing to throw a ball to a catcher. Does your throw require calculating which muscles to use and how much force to apply to each one? This is Evarts's position. It is based on his findings about how neurons of the motor cortex change their rates of discharge in response not only to which muscle is needed (flexor or extensor, for instance), but also to how much force is required to make a particular movement. Alternatively, perhaps your throw to the catcher simply requires determining the location at which you want the ball to arrive. This is Georgopoulos's position. He maintains that the cortex needs to specify only the spatial target of a movement—that is, its basic direction. Other brain structures, such as the brainstem and spinal cord, will look after the details of the throw.

Georgopoulos's hypothesis is very appealing in its simplicity. But is it sufficient to explain how the motor cortex controls a skilled movement? When you move an arm in a particular direction, many arm muscles are very active, whereas others are less active. When you then alter the direction of the movement, most of the same arm muscles remain active, but the force produced by each muscle changes, with some becoming less active and others more active. Recording from a single neuron associated with a single muscle might give the impression that the neuron is coding the movement's directional target, but the neuron might also be coding the force associated with the muscle's particular contribution to that movement. Exactly what the code entails is still not understood. The directional hypothesis and the force hypothesis are both topics of current debate in the study of how the motor cortex controls movement (Fetz, 1992).

The Control of Skilled Movements in Other Species

Humans are far from the only species making skilled movements. Kamala, the elephant, as you know, paints works of art with her trunk, and primates other than humans are very skillful with their hands, as we are. What is it about the motor cortex in other species that enables these skilled movement patterns?

Studies of a wide range of animals show that the motor cortex is organized to correspond to the skilled movements of a species. Just as in humans, larger parts of the motor cortex regulate body parts that carry out these movements. Figure 10-16 shows the human homunculus and comparable cartoon figures for four other animals—the rabbit, cat, monkey, and elephant. As you can see, rabbits have a large motor-cortex representation for the head and mouth, cats for the mouth and front claws, and monkeys for the hands, feet, and digits. Although no one has mapped the motor cortex of an elephant, it is likely that elephants have a disproportionately large area of motor cortex regulating the trunk.

How did these specialized representations of the motor cortex evolve? One possibility is that they were constructed from the outside inward (Woolsey & Wann, 1976). Chance mutations caused an increase in the number of muscles in a particular part of the body, which led to more motor neurons in the spinal cord. This increase in motor neurons, in turn, led to an increase in the area of the motor cortex controlling those spinal-cord motor neurons. Finally, the larger motor-cortex representation, along with an increased possibility of making connections between these cortical neurons, led to a capacity for making new and more complex movements.

Let us apply this scenario to the development of the elephant's trunk. First, chance mutations led to the expansion of muscles in the elephant's lip and nose and the spinal-cord motor neurons needed to move them. These developments were retained because they were useful for feeding. The area of the motor cortex then expanded to represent the new muscles of the trunk.

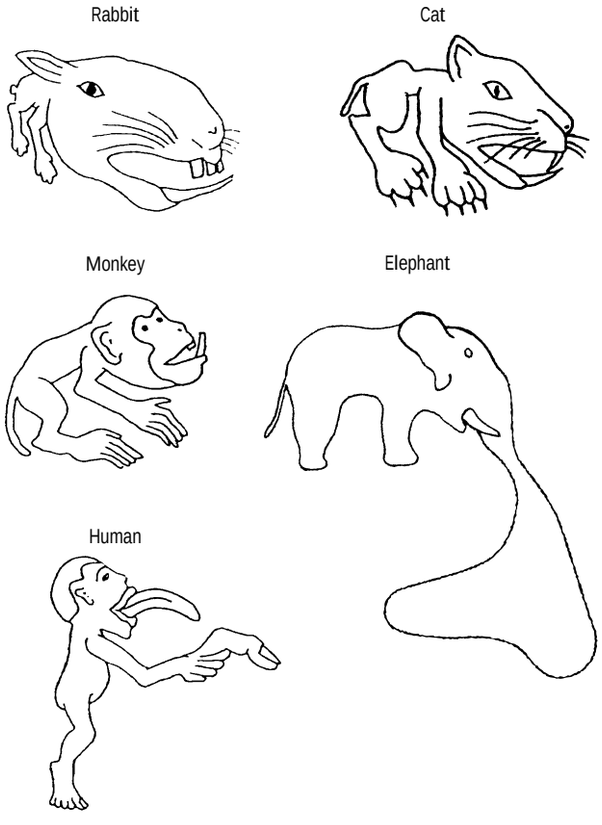


Figure 10-16

The difference in the size of the motor-cortex representation of different body parts in several species of animals suggests that the size of the cortical area regulating a body part corresponds to the skill required to move that body part. The representation for the elephant is only surmised.

Adapted from *Principles of Neural Science* (3rd ed., p. 373), by E. R. Kandel, J. H. Schwartz, and T. M. Jessel, 1991, New York: Elsevier.

How Motor Cortex Damage Affects Skilled Movements

In the 1940s, when scientists were first producing maps of the motor cortex, a number of researchers got slightly different results when they repeated the mapping procedures on the same subjects. These findings led to a debate. Some scientists held that the map of the motor cortex was capable of changing—that areas controlling particular body parts might not always stay in exactly the same place and retain exactly the same dimensions. But other researchers felt that this view was unlikely. They argued that, given the enormous specificity of topographic maps of the motor cortex, these maps must surely be quite stable. If they appeared to change, it must be because the relatively large electrodes used for stimulating and recording from cortical neurons must be producing inexact results. As the mapping procedures improved, however, and as smaller and smaller electrodes were used, it became clear that these maps can indeed change. They can change as a result of sensory or motor learning (a topic to be explored in Chapter 13), and they can change when part of the motor cortex is damaged, as the following example shows.

A study by Randy Nudo and his coworkers (1996), summarized in Figure 10-17, illustrates change in a map of the motor cortex that is due to cortical damage. These

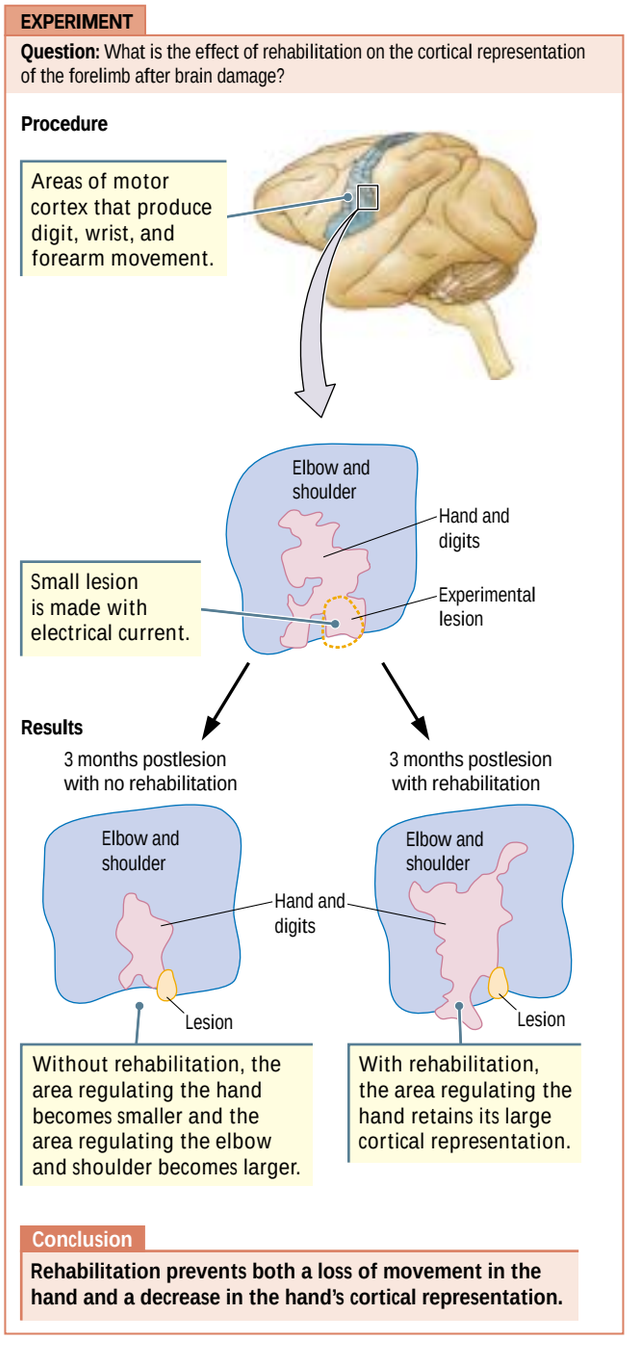


Figure 10-17

Effect of experience on the cortical representation of the forelimb in the motor cortex of a monkey after brain damage. Weak electrical stimulation shows the areas of the cortex that produce digit, wrist, and forearm movements in a monkey before a small lesion is made with a larger electrical current through the electrode. The area that receives the lesion is indicated by the dashed lines. In the monkey that was not forced to use its affected limb, the area of cortex representing the hand has become smaller and the area of motor cortex representing the elbow and shoulder has become larger. In a monkey that was forced to use the affected limb because the good limb was bound, the hand area retains its large cortical representation.

Adapted from "Neural Substrates for the Effects of Rehabilitative Training on Motor Recovery After Ischemic Infarct," by R. J. Nudo, B. M. Wise, F. SiFuentes, and G. W. Milliken, 1996, *Science, 272,* p. 1793.

researchers mapped the motor cortices of monkeys to identify the hand and digit areas. They then surgically removed a small part of the digit area. After the surgery, the monkeys used the affected hand much less, relying mainly on the good hand. Three months later, the researchers examined the monkeys. They found that the animals were unable to produce many movements of the lower arm, including the wrist, the hand, and the digits surrounding the area with the lesion. They also discovered that much of the area representing the hand and lower arm was gone from the cortical map. The shoulder, upper arm, and elbow areas had spread to take up what had formerly been space representing the hand and digits. Figure 10-17 (Results, left) shows this topographic change.

The experimenters wondered whether the change could have been prevented had they forced the monkeys to use the affected arm. To find out, they used the same procedure on other monkeys, except that, during the postsurgery period, they made the animals rely on the bad arm by binding the good arm in a sling. Three months later, when the experimenters reexamined the motor maps of these monkeys, they found that the hand and digit area retained its large size, even though there was no neural activity in the spot with the lesion. Nevertheless, the monkeys had gained some function in the digits that had formerly been connected to the damaged spot. Apparently, the remaining digit area of the cortex was now controlling the movement of these fingers.

The motor-cortex reorganization that Nudo and his colleagues observed probably explains some kinds of recovery from brain damage observed in humans. For instance, Paul Bucy and his coworkers (1964) studied a man who had had the corticospinal tract cut on one side of his nervous system to stop involuntary movement of his muscles. During the first 24 hours after the surgery, the side of his body contralateral to the cut was completely flaccid, and he was unable to make any movements on that side. (The impairment was on the side of the body opposite that of the cut because the corticospinal tract crossed to the other side just below the location of the cut.) Then gradually there was some recovery of function. By the 10th day after the surgery, the patient could stand alone and walk with assistance. By the 24th day, he could walk unaided. Within 7 months, he could move his feet, hands, fingers, and toes with only slight impairment.

The explanation of this man's remarkable recovery is twofold. First, when the man died about 2½ years later, an autopsy revealed that approximately 17 percent of the corticospinal fibers were intact in the tract that had been cut. Apparently, the remaining corticospinal fibers were able to take over much of the function formerly served by the entire pathway. Second, extensive reorganization likely took place in the map of the man's motor cortex, so many cortical regions could use the fibers that had remained intact to send messages to motor neurons in the spinal cord.

In Review

Basic patterns of movement that are common to a particular species are organized in the motor cortex as synergies. The discharge patterns of motor-cortex neurons suggest that these neurons take part in planning and initiating movements, as well as in carrying movements out. The discharge rate of these neurons is related both to the force of muscle contraction and to the direction of a movement. The topographic map of the motor cortex in a particular species is related to the species' body parts that are capable of making the most skillful movements. The relation between neurons in the motor cortex and the movement of specific muscles is not fixed. Considerable change can take place in the cortical motor map after injury to the motor cortex.

Investigate the area on control of movement in the module on Control of Movement in the CD. Look for details on what happens when there is damage to these regions.

THE BASAL GANGLIA AND THE CEREBELLUM

The main evidence that the basal ganglia and the cerebellum have motor functions is that damage to either structure impairs movement. Both structures also have extensive connections with the motor cortex, further suggesting their participation in movement. After an overview of the anatomy of the basal ganglia and cerebellum, we will look at some of the symptoms that arise after they are damaged. Then we will consider some experiments that illustrate the roles that they might play in controlling movement.

The Basal Ganglia and Movement Force

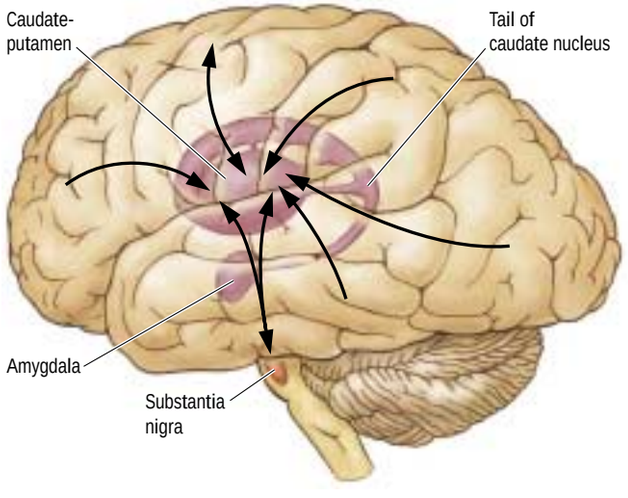
The basal ganglia are a collection of nuclei in the forebrain that make connections with the motor cortex and with the midbrain. As shown in Figure 10-18, a prominent structure in the basal ganglia is the *caudate putamen*, a large cluster of nuclei located beneath the frontal cortex. Part of the caudate extends as a "tail" into the temporal lobe, ending in the amygdala.

The basal ganglia receive inputs from two main sources. First, all areas of the neocortex and limbic cortex, including the motor cortex, project to the basal ganglia. Second, there is a dopaminergic projection to the basal ganglia from the substantia nigra, a cluster of darkly pigmented cells of the midbrain. The basal ganglia project back to both the motor cortex and the substantia nigra.

Two different, and in many ways opposite, kinds of movement disorders result from basal ganglia damage. If cells of the caudate putamen are damaged, unwanted choreiform (writhing and twitching) movements occur. For example, Huntington's chorea, in which caudate putamen cells are destroyed, is characterized by involuntary and exaggerated movements. Other examples of involuntary movements related to caudate putamen damage are the unwanted tics

Figure 10-18

The basal ganglia consist of the caudate putamen, the tail of the caudate nucleus, and the amygdala. The caudate-putamen makes reciprocal connections with the substantia nigra. It also receives input from most regions of the cortex and sends input into the frontal lobes via the thalamus.



Focus on Disorders

Tourette's Syndrome

The neurological disorder known as Tourette's syndrome (TS) was first described in 1885 by Georges Gilles de la Tourette, a young French neurologist and friend of Sigmund Freud. Here is how Tourette described the symptoms as they appeared in Madame de D., one of his own patients:

> Madame de D., presently age 26, at the age of 7 was afflicted by convulsive movements of the hands and arms. These abnormal movements occurred above all when the child tried to write, causing her to crudely reproduce the letters she was trying to trace. After each spasm, the movements of the hand became more regular and better controlled until another convulsive movement would again interrupt her work. She was felt to be suffering from over-excitement and mischief, and because the movements became more and more frequent, she was subject to reprimand and punishment. Soon it became clear that these movements were indeed involuntary and convulsive in nature. The movements involved the shoulders, the neck, and the face, and resulted in contortions and extraordinary grimaces. As the disease progressed, and the spasms spread to involve her voice and speech, the young lady made strange screams and said words that made no sense. (Friedhoff & Chase, 1982)

Tourette's syndrome has an incidence of less than 1 per 1000 people. It is found in all racial groups and seems to be hereditary. The average age of onset is between 2 and 25 years. The most frequent symptoms are involuntary tics and involuntary complex movements, such as hitting, lunging, or jumping. People with the syndrome may also suddenly emit cries and other vocalizations or inexplicably utter words that do not make sense in the context, including swear words. TS is not associated with any other disorders, although much milder cases of tics may be related to it.

TS is thought to be due to an abnormality of the basal ganglia, especially the right basal ganglia. It is an example of one of the hyperkinetic disorders that can result from basal ganglia dysfunction. The symptoms of TS can be controlled with haloperidol, which blocks dopamine synapses in the basal ganglia.

Many people with TS function quite well, coping successfully with their symptoms. There are people with Tourette's syndrome in all walks of life, even surgeons who must perform delicate operations. With the existence of the Tourette's Society in the past 20 years, public awareness of the disorder has increased. Children with TS are now less likely to be diagnosed as having a psychiatric condition, being hyperactive, or being troublemakers.

and vocalizations peculiar to Tourette's syndrome, which is discussed in "Tourette's Syndrome" above. In addition to causing involuntary movements, called **hyperkinetic symptoms**, damage to the basal ganglia can result in a loss of motor ability, called **hypokinetic symptoms**. One such hypokinetic disorder, Parkinson's disease, was discussed in preceding chapters. It is caused by the loss of dopamine cells in the substantia nigra and is characterized by an inability to produce normal movements. The two different kinds of symptoms that arise subsequent to basal ganglia damage—hyperkinetic and hypokinetic symptoms—suggest that a major function of these nuclei is to modulate movement.

Steven Keele and Richard Ivry (1991) tried to relate the two different kinds of basal ganglia symptoms by suggesting that the underlying function of the basal ganglia is to generate the force required for each particular movement. According to this idea, some types of basal ganglia damage cause errors of too much force and so result in excessive movement, whereas other types of damage cause errors of too little force and so result in insufficient movement. Keele and Ivry tested their hypothesis by giving

Hyperkinetic symptom. A symptom of brain damage that involves involuntary excessive movements.

Hypokinetic symptom. A symptom of brain damage that involves a paucity of movement.

healthy subjects as well as patients with various kinds of basal ganglia disorders a task that tested their ability to exert appropriate amounts of force. The subjects viewed a line on a television screen; by pushing a button with varying amounts of force, they could produce a second line to match the length of the first. After a number of practice trials, the subjects were then asked to press the button with the appropriate amount of force even when the first line was no longer visible as a guide. In contrast to control subjects, patients with basal ganglia disorders were unable to reliably do so. The force that they exerted was usually too little or too much, resulting in a line too short or too long.

What neural pathways enable the basal ganglia to modulate the force of movements? Basal ganglia circuits are quite complex, but one theory holds that there are two pathways through which the activity of the motor cortex is affected: an inhibitory pathway and an excitatory pathway (Alexender & Crutcher, 1990). Both these pathways converge on an area of the basal ganglia called the internal part of the globus pallidus (GP_i), as shown in Figure 10-19. The GP_i in turn projects to the thalamus (more specifically, to the ventral thalamic nucleus), and the thalamus projects to the motor cortex. The thalamic projection modulates the size or force of a movement that the cortex produces, but the thalamic projection is influenced by the GP_i. The GP_i is thought of as acting like the volume dial on a radio because its output determines whether a movement will be weak or strong.

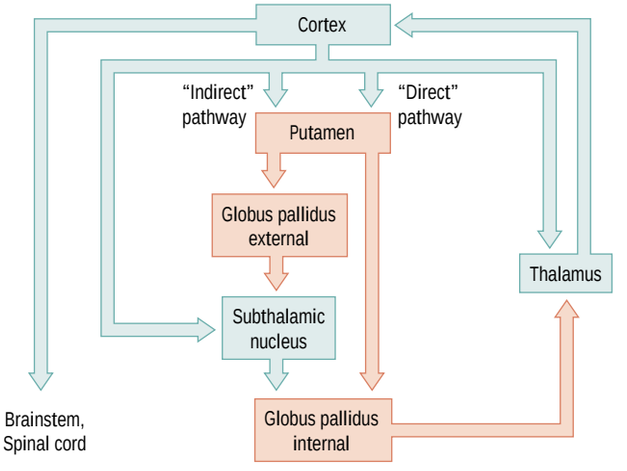


Figure 10-19

Two pathways in the basal ganglia modulate cortically produced movements. Green indicates the parts of the pathways that are excitatory, and red indicates the parts that are inhibitory. The indirect pathway has an excitatory effect on the internal part of the globus pallidus (GP_i), whereas the direct pathway has an inhibitory effect on the GP_i. If inhibition dominates, the thalamus is shut down and the cortex is unable to produce movement. If excitation predominates, the thalamus can become overactive, thus amplifying movement. GP_e refers to the external part of the globus pallidus; STN represents the subthalamic nucleus.

Adapted from "Functional Architecture of Basal Ganglia Circuits: Neural Substrates of Parallel Processing," by R. E. Alexander and M. D. Crutcher, 1990, *Trends in Neuroscience, 13,* p. 269.

The inputs to the GP_i are shown in red and green in Figure 10-19 to illustrate how they affect movement. If activity in the inhibitory pathway (red) is high relative to that in the excitatory pathway (green), inhibition of the GP_i will predominate and the thalamus will be free to excite the cortex, thus amplifying movement. If, on the other hand, activity in the excitatory pathway is high relative to that in the inhibitory pathway, excitation of the GP_i will predominate and the thalamus will be inhibited, thus reducing input to the cortex and decreasing the force of movements.

The idea that the GP_i acts like a volume control over movement is currently receiving a great deal of attention. If the GP_i is surgically destroyed in Parkinson patients, muscular rigidity is reduced and the ability to make normal movements is improved. Also consistent with this "volume hypothesis," recordings made from cells of the globus pallidus show that they are excessively active in people with Parkinson's disease.

The Cerebellum and Movement Skill

In referring to the amount of practice required to play a musical instrument, musicians have a saying: "Miss a day of practice and you're OK; miss two days and you notice; miss three days and the world notices." Apparently, some change must take place in the brain when practice of a motor skill is neglected. The cerebellum may be the part of the motor system that is affected. Whether the skill is playing a musical instrument, pitching a baseball, or typing on a computer keyboard, the cerebellum is critical for acquiring and maintaining motor skills.

The cerebellum, a large and conspicuous part of the motor system, sits on top of the brainstem and is clearly visible just behind the cerebral cortex. The cerebellum is divided into two hemispheres, as is the cerebral cortex. A small lobe called the flocculus projects from its ventral surface. Despite the cerebellum's relatively small size, it contains about one-half of all the neurons of the nervous system.

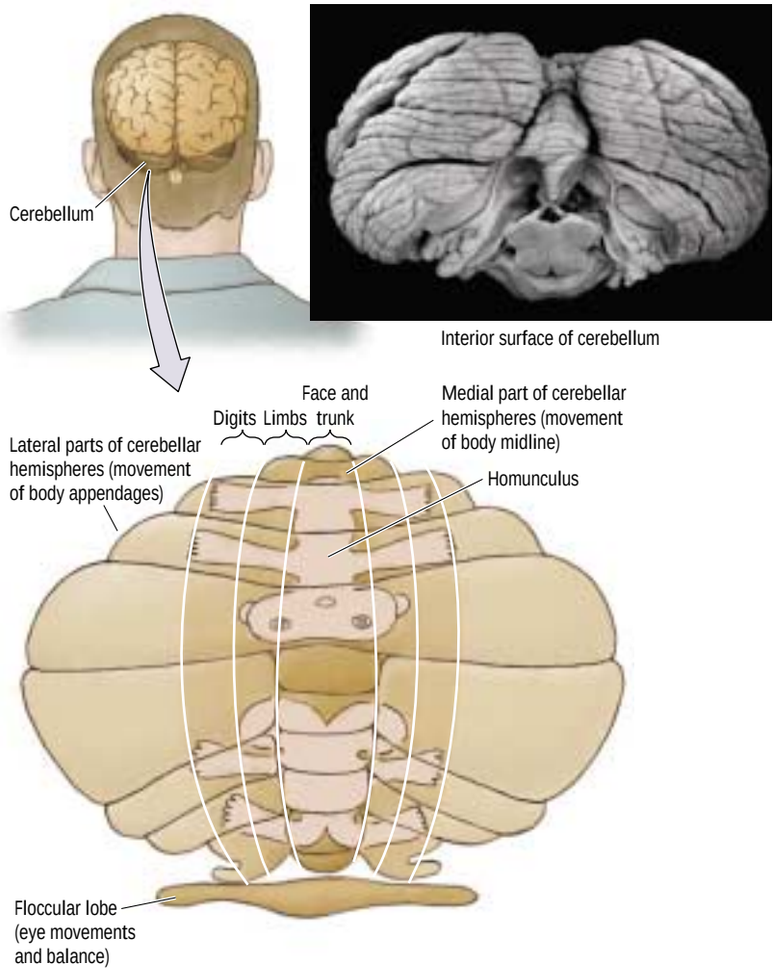


Figure 10-20

The cerebellum consists of the cerebellar hemispheres and the flocculus. The hemispheres control body movements, and the flocculus controls balance. The cerebellum is topographically organized, with its more medial parts representing the midline of the body and its more lateral parts representing the limbs and digits.

Photo of cerebellum reproduced from *The Human Brain: Dissections of the Real Brain* by T. H. Williams, N. Gluhbegovic, and J. Jew, on CD-Rom. Published by Brain University, brain-university.com 2000.

As Figure 10-20 shows, the cerebellum can be divided into several regions, each of which specializes in a different aspect of motor control. The flocculus receives projections from the vestibular system, which will be described shortly, and takes part in the control of balance and eye movements. Many of its projections go to the spinal cord and to the motor nuclei that control eye movements. The hemispheres of the cerebellum can be subdivided as shown by the white lines in the drawing. The most medial part controls the face and the midline of the body. The more lateral parts are connected to areas of the motor cortex and are associated with movements of the limbs, hands, feet, and digits. The pathways from the hemispheres project to nuclei of the cerebellum, which in turn project to other brain regions, including the motor cortex.

To summarize the cerebellum's topographic organization, the midline of the homunculus is represented in the central part of the cerebellum, whereas the limbs and digits are represented in the cerebellum's lateral parts. Tumors or damage to midline areas of the cerebellum disrupt balance, eye movements, upright posture, and walking but do not substantially disrupt other movements such as reaching, grasping, and using the fingers. For example, a person with medial damage to the cerebellum may, when lying down, show few symptoms. Damage to lateral parts of the cerebellum disrupts arm, hand, and finger movements much more than movements of the body's trunk.

Attempts to understand how the cerebellum controls movements have centered on two major ideas: (1) that the cerebellum plays a role in the timing of movements and (2) that the cerebellum maintains movement accuracy. Keele and Ivry support the first of these two ideas. They suggest that the underlying impairment in disorders of the cerebellum is a loss of timing. According to them, the cerebellum acts like a clock

or pacemaker to ensure that both movements and perceptions are appropriately timed. In a motor test of timing, subjects were asked to tap a finger to keep time with a metronome. After a number of taps, the metronome was turned off and the subjects were to continue to tap with the same beat. Those with damage to the cerebellum, especially to the lateral cerebellum, were impaired on the task. In a perceptual test of timing, subjects were presented with two pairs of tones. The silent period between the first two tones was always the same length, whereas the silent period between the second two tones changed from trial to trial. The subjects had to tell whether the second silent period was longer or shorter than the first. Those with damage to the cerebellum were also impaired on this task. Apparently, the cerebellum can act like a clock to time perceptions as well as movements.

Not all researchers believe that the cerebellum's major contribution to controlling movements is one of timing, however. Tom Thach and his coworkers (1992) argue that the primary role of the cerebellum is to help make the adjustments needed to keep movements accurate. They gathered evidence in support of this view by having subjects throw darts at a target, as shown in Figure 10-21. After a number of throws, the subjects put on glasses containing wedge-shaped prisms that displaced the apparent location of the target to the left. Then when the subjects threw a dart, it landed to the left of the intended target. All subjects showed this initial distortion in aim. But then came an important difference. When normal subjects saw the dart miss the mark, they adjusted each successive throw until reasonable accuracy was restored. In contrast, subjects with damage to the cerebellum could not correct for this error. They kept missing the target far to the left time after time. Next the subjects removed the prism glasses and threw a few more darts. Again, another significant difference emerged. The first dart thrown by each normal subject was much too far to the right (owing to the previous adjustment that the subject had learned to make), but soon each adjusted once again until his or her former accuracy was regained. In contrast, subjects with damage to the cerebellum showed no aftereffects from having worn the prisms, as if they had never compensated for the glasses to begin with. This experiment suggests that many movements that we make—whether throwing a dart, hitting a ball with a bat, writing neatly, or painting a work of art—depend on moment-to-moment learning and adjustments that are made by the cerebellum.

To better understand how the cerebellum improves motor skills by making required adjustments to movements, imagine throwing a dart yourself. Suppose you aim at the bull's eye, throw the dart, and find that it misses the board completely. You then aim again, this time adjusting your throw to correct for the original error. Notice that there are actually two versions of your action: (1) the movement that you intended to make and (2) the actual movement as recorded by sensory receptors in your arm and shoulder. If the intended movement is successfully carried out, you need make no correction on your next try. But, if you miss, an adjustment is called for. One way in which the adjustment might be made is through the

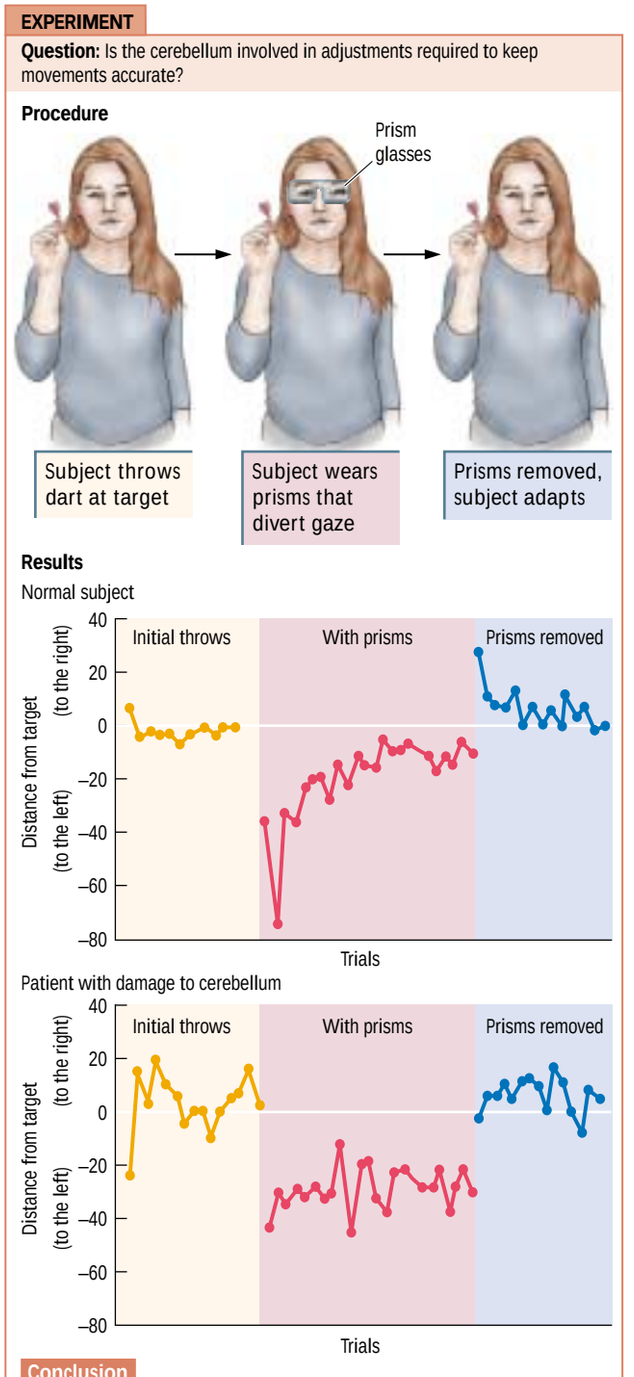


Figure 10-21

This experiment demonstrates that the cerebellum is required to adapt movements to compensate for visual displacement produced by prisms that divert gaze.

Adapted from "The Cerebellum and the Adaptive Coordination of Movement," by W. T. Thach, H. P. Goodkin, and J. G. Keating, 1992, *Annual Review of Neuroscience, 15,* p. 429.

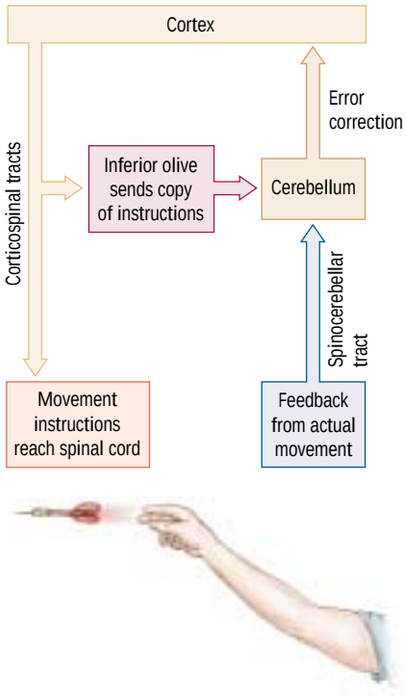


Figure 10-22

A feedback circuit allows the cerebellum to correct movements. The cerebellum receives information about the instructions sent to motor neurons through the inferior olive. It receives information about the actual movement through the spinocerebellar tract (a sensory pathway carrying information from the spinal cord to the cerebellum that provides information about movements that have been made). By comparing the message for the intended movement with the movement that was actually performed, the cerebellum can send an error message to the cortex to improve the accuracy of a subsequent movement.

circuit shown in Figure 10-22. The cortex sends instructions to the spinal cord to throw a dart at the target. A copy of the same instructions is sent to the cerebellum through the inferior olive. When you then throw the dart, the sensory receptors in your arm and shoulder code the actual movement that you make and send a message about it to the cerebellum. The cerebellum now has information about both versions of the movement: what you intended to do and what you actually did. The cerebellum can now calculate the error and tell the cortex how it should correct the movement. When you next throw a dart, you incorporate that correction into your throw.

In Review

The basal ganglia contribute to motor control by adjusting the force associated with each movement. Consequently, damage to the basal ganglia results either in unwanted involuntary movements (too much force being exerted) or in such rigidity that movements are difficult to perform (too little force being exerted). The cerebellum contributes to the control of movement by improving movement skill. One way in which it may do so is by keeping track of the timing of movements. Another way is by making adjustments in movements to maintain their accuracy. In the latter case, the cerebellum compares an intended movement with an actual movement and calculates any necessary corrections.

THE ORGANIZATION OF THE SOMATOSENSORY SYSTEM

The somatosensory system tells us what the body is up to by providing information about bodily sensations, such as touch, temperature, pain, position in space, movement of the joints, and so forth. In addition to helping us learn about the world, the somatosensory system allows us to distinguish what the world does *to us* from what we do *to it*. For example, when someone pushes you sideways, your somatosensory system tells you that you have been pushed. Similarly, if you lunge to the side yourself, your somatosensory system tells you that you did the moving.

Although, in this book, the visual system and the auditory system are treated in separate chapters, here we explore the somatosensory system and the motor system in a single chapter. The reason is that somatosensation has a closer relation to movement than the other senses do. If we lose sight or hearing or even both, we are still able to move around, and the same is true of other animals. For instance, fish that inhabit deep, dark caves cannot see at all, yet they are able to move about normally. And animals, such as the butterfly, that cannot hear can still move very well. If an animal were to lose its body senses, however, its movements would quickly become so impaired that it would not be able to survive. Some aspects of somatosensation are absolutely essential to movement, so the two topics are covered together.

In considering the motor system, we started at the cortex and followed the motor pathways to the spinal cord. This route makes sense because it follows the direction in which instructions regarding movements flow. As we explore the somatosensory system, we will proceed in the opposite direction, because it is the direction in which sensory information flows. We will start at sensory receptors in various parts of the body and follow sensory pathways to the cortex.

The somatosensory system is unique among sensory systems because it is distributed throughout the entire body; it is not just localized in the head as are vision, hearing, taste, and smell. Somatosensory receptors are found in all parts of the body, and neurons from these receptors carry information to the spinal cord. Within the spinal cord, two somatosensory pathways project to the brain and, eventually, to the so-

Review the somatosensory system in the brain overview section in the Central Nervous System module on your CD.

matosensory cortex. One part of the somatosensory system, the vestibular system, is confined to a single organ, however. The vestibular system, located in the middle ear, contributes to our sense of movement and balance. In the following sections, we will look at the anatomy of the different parts of the somatosensory system and at examples of how each contributes to movement.

Glabrous skin. Skin that does not have hair follicles but contains larger numbers of sensory receptors than do other skin areas.

Somatosensory Receptors and Sensory Perception

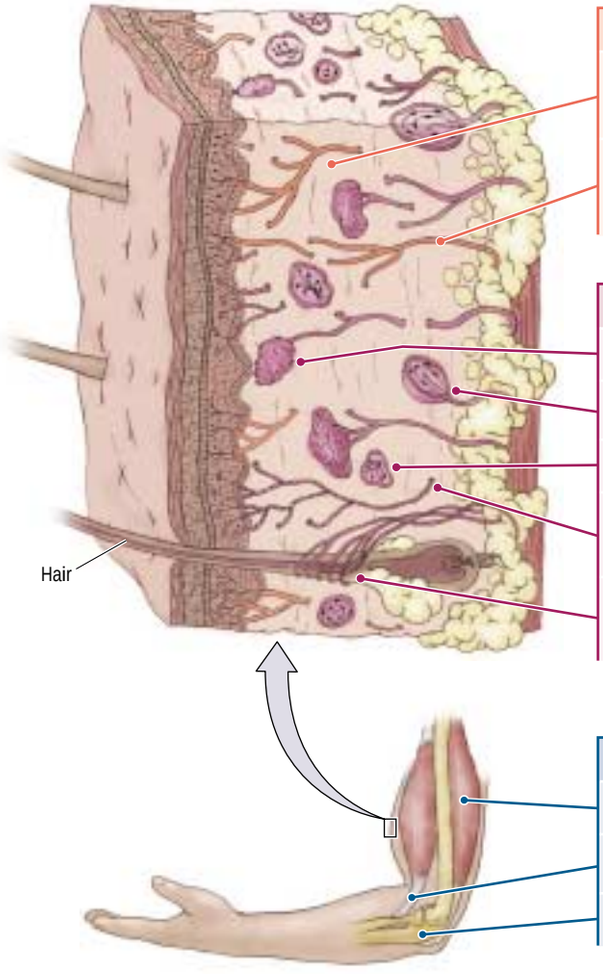
Our bodies are covered with sensory receptors. They are attached to body hairs. They are located in both surface layers and deeper layers of the skin. They are embedded in muscles, tendons, and joints. Some consist simply of the ending of a sensory neuron dendrite. On others, the dendrite is covered by a special capsule or it is attached by a sheath of connective tissue to adjacent tissue.

The density of sensory receptors varies greatly in different parts of the body, not only in the skin, but in the muscles, tendons, and joints as well. The variation in density is one reason why different parts of the body are more or less sensitive to somatosensory stimulation. Parts of the body that are very sensitive to touch or capable of fine movements—including the hands, feet, lips, and eyes—have many more somatosensory receptors than other body parts do. Sensitivity to different somatosensory stimuli is also a function of the kinds of receptors that are found in a particular region.

Figure 10-23 includes examples of somatosensory receptors located in the skin. Humans have two kinds of skin, hairy skin (shown in Figure 10-23) and glabrous skin

Figure 10-23

The perceptions derived from the body senses depend on different receptors located in different parts of the skin, muscles, joints, and tendons.



Nocioception (pain and temperature)	Adaptation
Free nerve endings for pain (sharp pain and dull pain)	Slow
Free nerve endings for temperature (heat or cold)	Slow

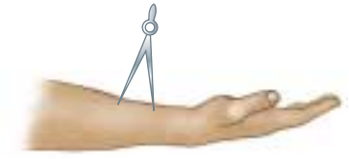
Damage to the dendrite or to surrounding cells releases chemicals that stimulate the dendrite to produce action potentials.

Hapsis (fine touch and pressure)	Adaptation
Meissner's corpuscle (touch)	Rapid
Pacinian corpuscle (flutter)	Rapid
Ruffini corpuscle (vibration)	Rapid
Merkel's receptor (steady skin indentation)	Slow
Hair receptors (flutter or steady skin indentation)	Slow

Pressure on the various types of tissue capsules mechanically stimulates the dendrites within them to produce action potentials.

Proprioception (body awareness)	Adaptation
Muscle spindles (muscle stretch)	Rapid
Golgi tendon organs (tendon stretch)	Rapid
Joint receptors (joint movement)	Rapid

Movements stretch the receptors to mechanically stimulate the dendrites within them to produce action potentials.



Two-point sensitivity

(which is hairless). Glabrous skin, which includes the skin on the hands, lips, and tongue, is much more richly endowed with receptors than hairy skin is, which makes it exquisitely sensitive to a wide range of stimuli. The need for sensitivity in glabrous skin is due to the fact that it covers the parts of the body with which we explore objects.

The touch sensitivity of skin is often measured with a two-point sensitivity test. This test consists of touching the skin with two sharp points simultaneously and observing how close together they can be placed while still being detected as two points rather than one. On glabrous skin, we can detect the two points when they are as close as 3 millimeters apart. On hairy skin, in contrast, two-point sensitivity is much less. The two points seem to merge into one below a separation distance ranging from 2 to 5 centimeters, depending on exactly which part of the body is tested. You can confirm these differences in sensitivity on your own body by touching two sharp pencil points to a palm and to a forearm, varying the distances that you hold the points apart. Be sure not to look as you touch each surface.

Although there may be as many as 20 or more kinds of somatosensory receptors in the human body, they can be classified into three groups, depending on the type of perception that they enable. These three types of perception, as listed in Table 10-1, are *nocioception*, *hapsis*, and *proprioception*. Nocioception is the perception of pain and temperature. Nociceptors consist of free nerve endings. When these endings are damaged or irritated, they secrete chemicals, usually peptides, that stimulate the nerve to produce an action potential. The action potential then conveys a message about pain or temperature to the central nervous system. Hapsis is the perception of objects that we grasp and manipulate or that contact the body. It is also called the perception of fine touch and pressure. Haptic receptors are found in both superficial layers and deep layers of the skin and are attached to body hairs as well. A haptic receptor consists of a dendrite encased in a capsule of tissue. Mechanical stimulation of the capsule activates special channels on the dendrite, which in turn initiate an action potential. Differences in the tissue forming the capsule determine the kinds of mechanical energy conducted through it to the nerve. For example, pressure that squeezes the capsule of a Pacinian corpuscle is the necessary stimulus for initiating an action potential. Proprioception is the perception of the location and movement of the body. Proprioceptors, which also are encapsulated nerve endings, are sensitive to the stretch of muscles and tendons and the movement of joints. In the Golgi tendon organ, for instance, an action potential is triggered when the tendon moves, stretching the receptor attached to it.

Table 10-1 Somatosensory Receptors

Nocioception (pain and temperature)
Free nerve endings for pain (sharp pain and dull pain)
Free nerve endings for temperature (heat or cold)
Hapsis (fine touch and pressure)
Meissner's corpuscle (touch)
Pacinian corpuscle (flutter)
Ruffini corpuscle (vibration)
Merkel's receptor (steady skin indentation)
Hair receptors (flutter or steady skin indentation)
Proprioception (body awareness)
Muscle spindles (muscle stretch)
Golgi tendon organs (tendon stretch)
Joint receptors (joint movement)

Somatosensory receptors are specialized to tell two things about a sensory event: when it occurs and whether it is still occurring. Information about when a stimulus occurs is handled by rapidly adapting receptors. These receptors respond to the beginning and the end of a stimulus and produce only brief bursts of action potentials. Meissner's corpuscles (which respond to touch), Pacinian corpuscles (which respond to fluttering sensations), and Ruffini corpuscles (which respond to vibration) are all rapidly adapting receptors. In contrast, slowly adapting receptors detect whether a stimulus is still occurring. These receptors continue to respond as long as a sensory event is present. For instance, after you have put on an article of clothing and become accustomed to the feel of it, only slowly adapting receptors (such as Merkel's receptors and hair receptors) remain active. The difference between a rapidly adapting and a slowly adapting receptor is due in part to the way in which each is stimulated and in part to the way in which ion channels in the membrane of the dendrite respond to mechanical stimulation.

Nocioception. The perception of pain and temperature.

Hapsis. The perceptual ability to discriminate objects on the basis of touch.

Proprioception. Perception of the position and movement of the body, limbs, and head.

Rapidly adapting receptor. A body sensory receptor that responds briefly to the onset of a stimulus on the body.

Slowly adapting receptor. A body sensory receptor that responds as long as a sensory stimulus is on the body.

Dorsal-Root Ganglion Neurons

The dendrites that form somatosensory receptors belong to *dorsal-root ganglion neurons*. A dorsal-root ganglion neuron contains a single long dendrite, only the tip of which is responsive to sensory stimulation. This dendrite is continuous with the neuron's axon because the cell body is off to one side. The cell body, as the name of this neuron implies, is located in one of the dorsal-root ganglions that lie just beside the spinal cord. Each segment of the spinal cord has one dorsal-root ganglion on each of its sides. The axons of the dorsal-root ganglion neurons enter the spinal cord, forming the spinal cord's dorsal roots. In the spinal cord, these axons may synapse with other neurons or continue to the brain, as shown in Figure 10-24.

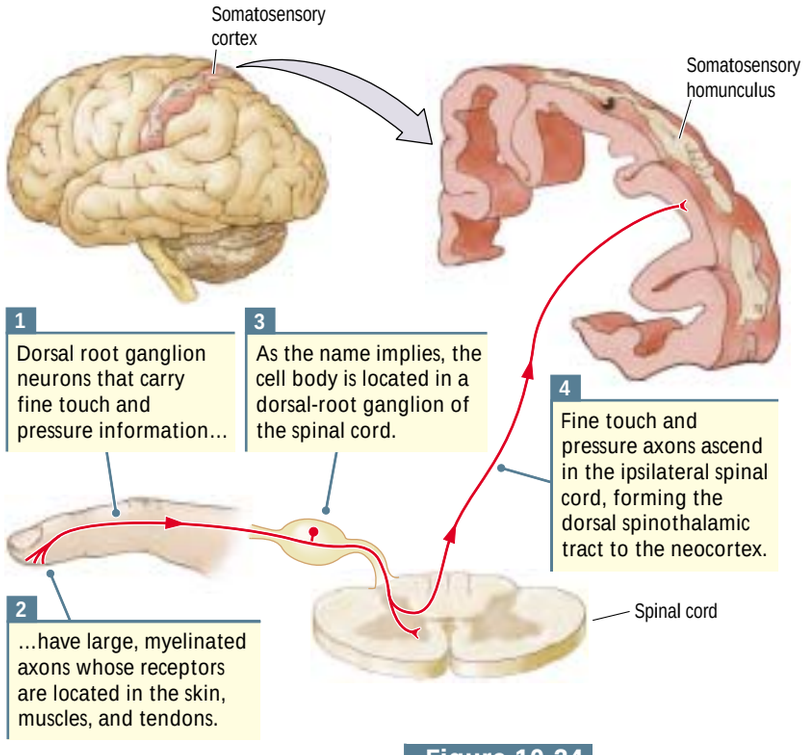


Figure 10-24

Somatosensory information is carried from the body to the central nervous system by dorsal-root ganglion neurons. The dendrite and axon of the ganglion neuron are contiguous and carry sensory information from the skin, muscles, and tendons. Fine touch and pressure information is carried by large myelinated axons, and pain and temperature information is carried by smaller unmyelinated axons. The large axons travel up the spinal cord to the brain in the dorsal columns, whereas the small axons synapse with neurons whose axons cross the spinal cord and ascend on the other side.

The axons of dorsal-root ganglion neurons vary in diameter and myelination. These structural features are related to the kind of information that the neurons carry. Proprioceptive (location and movement) information and haptic (touch and pressure) information are carried by dorsal-root ganglion neurons that have large, well-myelinated axons. Nociceptive (pain and temperature) information is carried by dorsal-root ganglion neurons that have smaller axons with little myelination. Because of their size and myelination, the larger neurons carry information much more quickly than the smaller neurons do. One explanation of why proprioceptive and haptic neurons are designed to carry messages quickly is that their information requires rapid responses. For instance, the nervous system must react to moment-to-moment changes in posture and to the equally rapid sensory changes that take place as we explore an object with our hands. In contrast, when the body is injured or cold, such rapid responding is not as essential, because these forms of stimulation usually continue for quite some time.

What happens when dorsal-root ganglion neurons are damaged? A clue comes from a visit to the dentist. If you have ever had a tooth "frozen" for dental work, you have experienced the very strange effect of losing sensation on one side of your face. Not only do you lose pain perception, you also lose the ability to move your facial muscles properly, making it difficult to talk, eat, and smile. So, even though the anesthetic is blocking only sensory nerves, your movement ability is affected, too.

In much the same way, damage to sensory nerves affects both sensory perceptions and motor abilities. For instance, John Rothwell and his coworkers (1982) described a patient, G. O., who was deafferented (lost sensory fibers) by a disease that destroyed sensory neurons. G. O. had no somatosensory input from his hands. He could not, for example, feel when his hand was holding something. However, G. O. could still accurately produce a range of finger movements, and he could outline figures in the air even with his eyes closed. He could also move his thumb accurately through different distances and at different speeds, judge weights, and match forces by using his thumb. Nevertheless, his hands were relatively useless to him in daily life. Although he was able to drive his old car, he was unable to learn to drive a new one. He was also unable to write, to fasten shirt buttons, or to hold a cup. He could begin movements quite normally, but, as he proceeded, the movement patterns gradually fell apart, ending in

Deafferented. Refers to loss of incoming sensory input usually due to damage to sensory fibers; also refers to loss of any afferent input to a structure.

failure. Part of his difficulties lay in maintaining muscle force for any length of time. When he tried to carry a suitcase, he would quickly drop it unless he continually looked down to confirm that it was there. Clearly, although G. O. had damage only to his sensory neurons, he suffered severe motor disability as well, including the inability to learn new motor skills.

Abnormalities in movement also result from more selective damage to sensory neurons, such as damage to neurons that carry proprioceptive information about body location and movement. Neurologist Oliver Sacks (1998) gives a dramatic example in his description of a patient, named Christina, who suffered damage to proprioceptive sensory fibers throughout her body after taking megadoses of vitamin B6. Christina was left with very little ability to control her movements and spent most of each day lying prone. Here is how she describes what a loss of proprioception means:

> "What I must do then," she said slowly, "is use vision, use my eyes, in every situation where I used—what do you call it?—proprioception before. I've already noticed," she added, musingly, "that I may lose my arms. I think they are in one place, and I find they're in another. This proprioception is like the eyes of the body, the way the body sees itself. And if it goes, as it's gone with me, *it's like the body's blind*. My body can't see itself if it's lost its eyes, right? So I have to watch it—be its eyes." (Sacks, 1998, p. 46)

Clearly, although Christina's motor system is intact, she is almost completely immobilized without a sense of where her body is in space and what her body is doing. She tries to use her eyes to compensate for loss of proprioception, but visual monitoring is less than satisfactory. Just imagine what it would be like to have to look at each of your limbs in order to move them to appropriate locations, which is why proprioception is so essential for movement (Cole, 1995).

The Somatosensory Pathways to the Brain

As the axons of somatosensory neurons enter the spinal cord, they divide, forming two pathways to the brain. The haptic-proprioceptive axons ascend the spinal cord on the same side of the body from which they have entered, whereas nocioceptive fibers synapse with neurons whose axons cross to the other side of the spinal cord before ascending to the brain. Figure 10-24 shows the first of these two routes through the spinal cord, whereas Figure 10-25 shows the second.

The haptic-proprioceptive axons form the dorsal spinothalamic tract. These axons synapse in the dorsal-column nuclei located at the base of the brain. Axons of neurons in the dorsal-column nuclei then cross over to the other side of the brainstem and ascend through the brainstem as part of a pathway called the medial lemniscus. These axons synapse in the ventrolateral thalamus. The neurons of the ventrolateral thalamus send most of their axons to the somatosensory cortex, but some axons go to the motor cortex.

The nocioceptive axons, as already stated, take a different route to the brain. They synapse with neurons in the dorsal part of the spinal cord's gray matter. These neurons, in turn, send their axons to the other side of the spinal cord, where they form the ventral spinothalamic tract. This tract joins the medial lemniscus in the brainstem to continue on to the ventrolateral thalamus. Some of the thalamic neurons receiving input from ventral spinothalamic tract axons also send their axons to the somatosensory cortex.

Because somatosensory information is conveyed by two separate pathways in the spinal cord, unilateral damage in the spinal cord results in distinctive sensory losses to both sides of the body below the site of injury. As is illustrated in Figure 10-26, there is loss of hapsis and proprioception on the side of the body on which the damage oc-

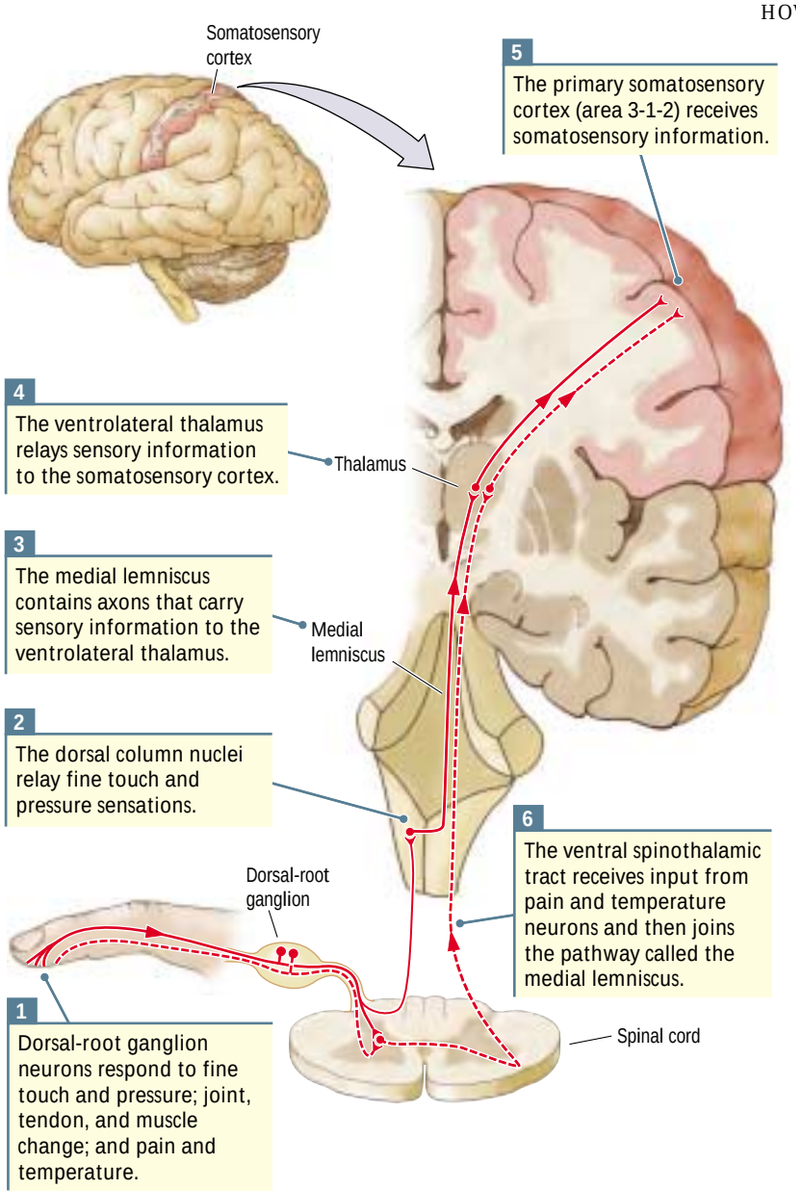


Figure 10-25

Somatosensory pathways to the brain diverge as dorsal-column neurons enter the spinal cord. Fine touch and pressure axons ascend in the dorsal column of the spinal cord, forming the dorsal spinothalamic tract. The axons synapse in the dorsal-column nuclei of the brainstem. Dorsal-column nucleus neurons cross the brainstem and ascend to synapse with ventrolateral thalamic neurons. The thalamic neurons project to the primary somatosensory cortex (area 3-1-2). Pain and temperature axons synapse with neurons as they enter the spinal cord. The pain and temperature axons cross the midline and ascend to the ventrolateral thalamus as the ventral spinothalamic tract. Both pathways form the medial lemniscus as they ascend through the brainstem.

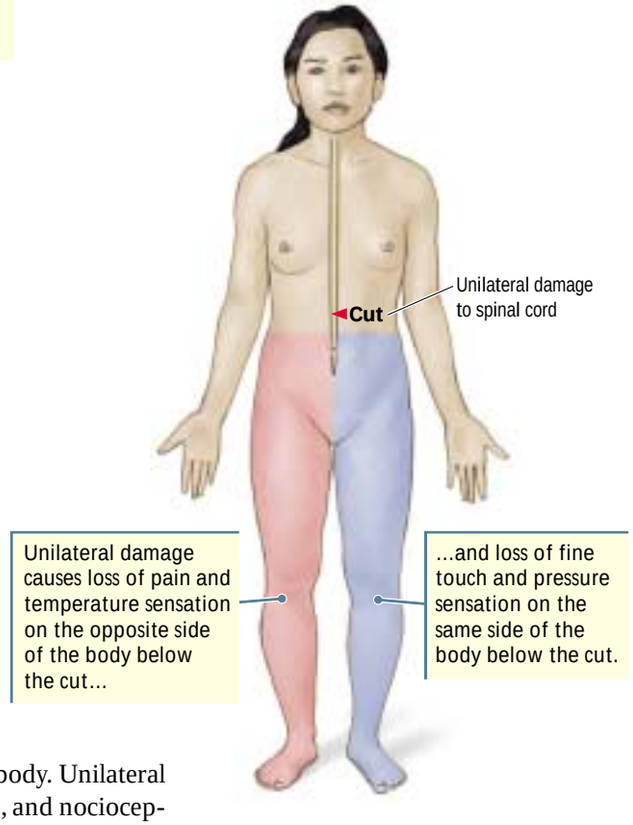


Figure 10-26

Unilateral damage to the spinal cord has different effects on fine touch and pressure versus pain and temperature sensations. Because fine touch and pressure information is conducted to the brain ipsilaterally through the dorsal spinothalamic tract, fine touch and pressure sensations are lost below the level of damage ipsilateral to the damage. Because pain and temperature information is conducted to the thalamus contralaterally through the ventral spinothalamic tract, pain and temperature sensations are lost below the level of damage contralateral to the damage.

curred, and there is a loss of nocioception on the opposite side of the body. Unilateral damage in the brainstem or the thalamus affects hapsis, proprioception, and nocioception equally, because the pathway for hapsis and proprioception and that for nocioception lie in close proximity.

Go to the area on the CD on the spinal reflexes in the Control of Movement module for more illustrations of the spinal cord and the spinal reflexes.

Spinal-Cord Responses to Somatosensory Input

Spinal-cord somatosensory axons, even those ascending in the dorsal columns, give off collaterals that synapse with interneurons and motor neurons on both sides of the spinal cord. The circuits made between sensory receptors and muscles through these connections mediate spinal reflexes. The simplest of these reflexes consists of a single synapse between a sensory neuron and a motor neuron. Figure 10-27 illustrates such a monosynaptic reflex. It concerns the quadriceps muscle of the thigh, which is anchored to the leg bone by the patellar tendon. When this tendon is tapped with a small hammer, the quadriceps muscle is stretched, activating the stretch-sensitive sensory receptors embedded in it. The sensory receptors then send a signal to the spinal cord through sensory neurons that synapse with motor neurons projecting back to the same thigh muscle. The discharge from the motor neurons stimulates the muscle, causing it to contract to resist the stretch. Because the tap is brief, the stimulation is over before the motor message arrives, so the muscle contracts even though it is no longer stretched. This contraction pulls the leg up, thereby producing the reflexive knee jerk.

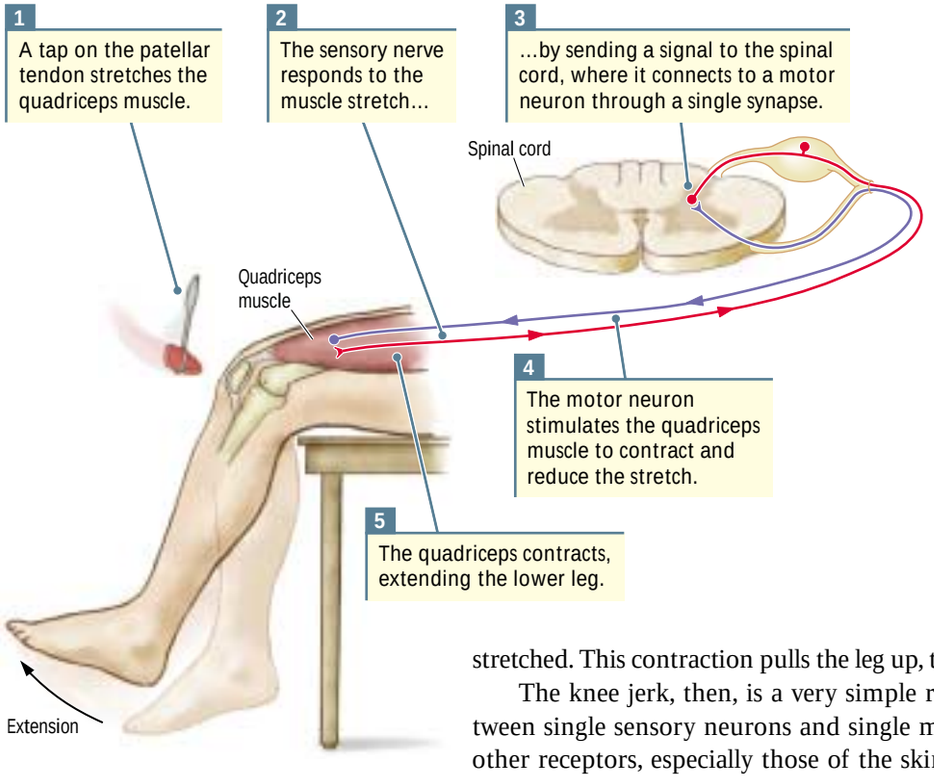


Figure 10-27

The "knee-jerk," or stretch, reflex produced by a light tap on the patellar tendon. The subject is seated on a table so that the lower leg hangs free. The tap on the patellar stretches the quadriceps muscle to which it is attached. Stretch receptors in the muscle send a brief burst of action potentials to the spinal cord to activate the motor neuron to the quadriceps by a single synapse. The contraction of the quadriceps causes the lower leg to extend.

The knee jerk, then, is a very simple reflex, with monosynaptic connections between single sensory neurons and single motor neurons. Somatosensory axons from other receptors, especially those of the skin, make much more complex connections with both interneurons and motor neurons. These multisynaptic connections are responsible for more complex reflexes that include many muscles on both sides of the body.

Circuits in the spinal cord also allow haptic-proprioceptive and nocioceptive pathways to interact. Such interactions may be responsible for our very puzzling and variable responses to pain. For example, people who are engaged in combat or intense athletic competition may receive a serious injury to the body but start to feel the pain only much later. For example, a friend of the authors, F. V., who was attacked by a grizzly bear while hiking, received 200 stitches to repair the bites that he received. When friends asked if it hurt to be bitten by a grizzly bear, he surprisingly answered no. As he explained it:

> I had read the week before about someone who was killed and eaten by a grizzly bear. So I was thinking that this bear was going to eat me unless I got away. I did not have time for pain. I was fighting for my life. It was not until the next day that I started feeling pain and fear.

Pain gate. A hypothetical neural circuit in which activity in fine touch and pressure pathways diminishes the activity in pain and temperature pathways.

Referred pain. Pain felt on the surface of the body that is actually due to pain in one of the internal organs of the body.

Pain is also puzzling in the variety of ways in which it can be lessened. Treatments for pain include opioid drugs (such as morphine), acupuncture (which entails the rapid vibration of needles embedded in the skin), and even simply rubbing the area surrounding the injury. To explain how pain can be suppressed in so many different ways, Ronald Melzack and Patrick Wall (1965) proposed a gate theory of pain. They argued that activity in the haptic-proprioceptive pathway can inhibit the pain pathway in the spinal cord through collaterals to spinal-cord interneurons. This pain gate, as it is

called, is illustrated in Figure 10-28. Notice that both the haptic-proprioceptive fibers and the nocioceptive fibers synapse with the interneuron. Collaterals from the haptic-proprioceptive pathway excite the interneuron, whereas collaterals from the nocioceptive pathway inhibit the interneuron. The interneuron, in turn, inhibits the neuron that relays pain information to the brain. Consequently, when the haptic-proprioceptive pathway is active, the pain gate partly closes, reducing the sensation of pain.

The gate theory can help explain how different treatments for pain work. For instance, when you stub your toe, you feel pain because the pain pathway to the brain is open. If you then rub the toe, activating the haptic-proprioceptive pathway, the flow of information in the pain pathway is reduced because the pain gate partly closes, which relieves the pain sensation. Similarly, acupuncture may have its pain-relieving effects because the vibrating needles used in this treatment selectively activate haptic and proprioceptive fibers, closing the pain gate. Interestingly, the interneurons in the pain gate may use opioid peptides as a neurotransmitter. If so, the gate theory can also explain how natural and endogenous opiods reduce pain.

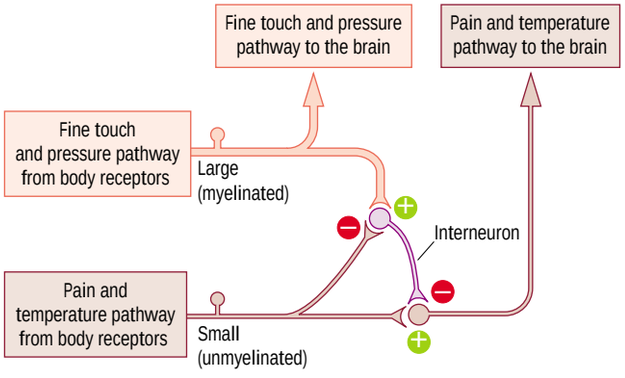


Figure 10-28

A pain gate. An interneuron in the spinal cord receives excitatory input from the fine touch and pressure pathway and inhibitor input from the pain and temperature pathway. The relative activity of the interneuron then determines whether pain and temperature information is sent to the brain. For example, if the fine touch and pressure pathway is active, it will excite the interneuron, which will in turn inhibit the second-order neurons in the pain and temperature pathway.

Adapted from *The Puzzle of Pain* (p. 154), by R. Melzack, 1973, New York: Basic Books.)

The gate theory even suggests an explanation for the painlike sensation of "pins and needles" that we feel after sitting too long in one position. Loss of oxygen from reduced blood flow may first deactivate the large myelinated axons that carry touch and pressure information, leaving the small unmyelinated fibers that carry pain and temperature messages unaffected. As a result, "ungated" sensory information flows in the pain and temperature pathway, leading to the curious pins and needles sensation.

Melzack and Wall propose that pain gates may be located in the brainstem and cortex in addition to the spinal cord. These additional gates could help explain how other approaches to pain relief work. For example, researchers have found that feelings of severe pain can be lessened when people have a chance to shift their attention from the pain to other stimuli. Dentists have long used this pain-reducing technique by giving their patients something soothing to listen to while undergoing painful work on their teeth. This influence of attention on pain sensations may work through a cortical pain gate. Electrical stimulation in a number of sites in the brainstem also can reduce pain, perhaps by closing brainstem pain gates. Another way in which pain perceptions might be lessened is through descending pathways from the forebrain and the brainstem to the spinal-cord pain gate.

Many internal organs of the body, including the heart, the kidneys, and the blood vessels, have pain receptors, but the ganglion neurons carrying information from these receptors do not have their own pathway to the brain. Instead, these ganglion neurons synapse with spinal-cord neurons that receive nocioceptive information from the body's surface. Consequently, the neurons in the spinal cord that relay pain and temperature messages to the brain receive two sets of signals: one from the body's surface and one from internal organs. These neurons cannot distinguish between these two sets of signals; nor can we. As a result, pain in body organs is often felt as pain from the surface of the body. Such pain is called referred pain. For example, the pain in the heart associated with a heart attack is felt as pain in the shoulder and upper arm. Similarly, pain in the stomach is felt as pain in the midline of the trunk, whereas pain in the kidneys is felt as pain in the lower back, and pain in blood vessels in the head is felt as diffuse pain that we call a headache. Figure 10-29 illustrates the referred pain felt in a heart attack.

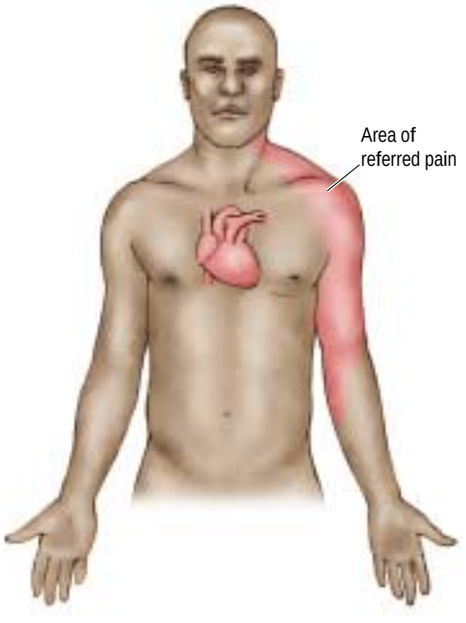


Figure 10-29

In a heart attack, pain can be felt in the shoulder and upper arm.

The Vestibular System and Balance

Vestibular system. A set of receptors in the middle ear that indicate position and movement of the head.

The vestibular system, also a part of the somatosensory system, consists of two organs, one located in each middle ear. As Figure 10-30 shows, each vestibular organ is made up of two groups of receptors: the semicircular canals, of which there are three, and the otolith organs, which consist of the utricle and the saccule. These vestibular receptors do two jobs. First, they tell us the position of the body in relation to gravity. Second, they signal changes in the direction and the speed of movements.

You can see in Figure 10-30 that the semicircular canals are oriented in three different planes: two vertical planes that are perpendicular to each other and one horizontal plane. Each canal furnishes information about movement in its particular plane. The semicircular canals are filled with a fluid called *endolymph.* Immersed in the endolymph is a set of hair cells very much like the hair cells on the arm. When the head moves, the endolymph also moves, splashing against the hair cells and bending the hairs. The force of the hairs bending is converted into action potentials that are sent over ganglion cells to the brain. The axons from these hair cells are normally quite active, but bending the hairs in one direction increases their activity, whereas bending the hairs in the other direction decreases it. Typically, when the head turns in one direction, the message on the side of the body to which the turn is made is an increase in neural firing, whereas the message on the body's opposite side is a decrease in firing.

The utricle and saccule are located just beneath the semicircular canals. They also contain hair cells, but their hair cells are embedded in a gelatin-like substance that contains small crystals of calcium carbonate know as otoconia. When the head is tilted, the gelatin and otoconia press against the hair cells, bending the hairs. When the hairs bend, the mechanical action produces action potentials in their neurons. In this way, the neurons' axons convey messages about the position of the head in space.

The receptors in the vestibular system tell us about our location relative to gravity, about acceleration and deceleration of our movements, and about changes in movement direction. They also allow us to ignore the otherwise very destabilizing influence that our movements might have on us. For example, when

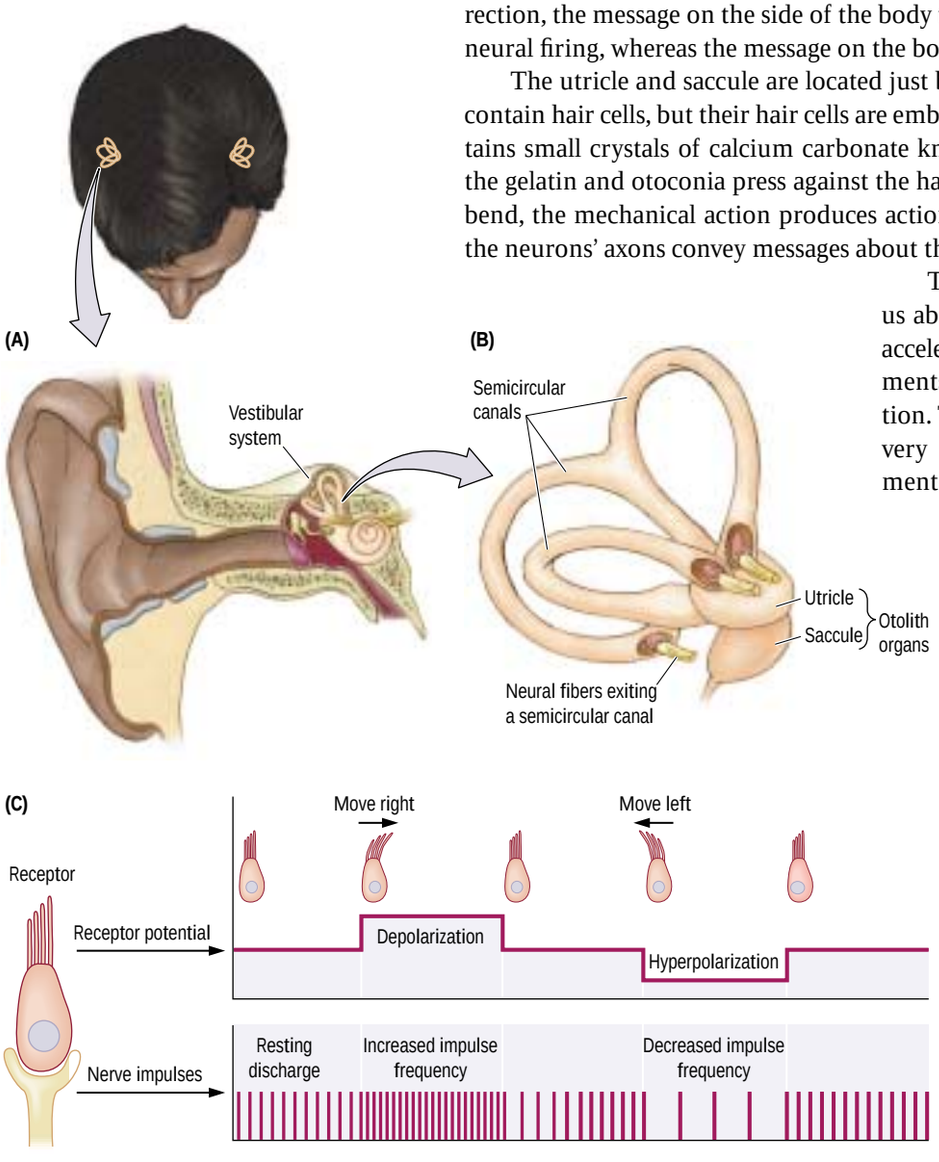


Figure 10-30

(A) The vestibular system. **(B)** The vestibular system consists of the three semicircular canals and the utricle and saccule. The receptors in the vestibular system are sensitive to movement of the head and to gravity. **(C)** Hair-cell receptors are located in the semicircular canals and in the utricle and the saccule. A vestibular neuron is normally active, and its activity increases if its hair-cell receptors are bent in one direction, but it decreases if the hair-cell receptors are bent in the opposite direction.

you are standing on a bus, even slight movements of the vehicle could potentially throw you off balance, but they do not. Similarly, when you make movements yourself, you easily avoid tipping over, despite the constant shifting of your body weight. Your vestibular system enables you to keep from tipping over.

Here is an experiment that you can perform to illustrate the role of vestibular receptors in helping you to compensate for your own movements. If you hold your hand in front of you and shake it, your hand appears blurry. But, if you shake your head instead of your hand, the hand remains in focus. Compensatory signals from your vestibular system allow you to see the hand as stable even though you are moving around.

In Review

Body senses contribute to the perception of hapsis (touch and pressure), proprioception (location and movement), and nocioception (temperature and pain). Haptic-proprioceptive information is carried by the dorsal spinothalamic tract, whereas nocioceptive information is carried by the ventral spinothalamic tract. The two systems interact in the spinal cord to regulate the perception of pain by a pain gate. Another part of the somatosensory system, the vestibular system, signals information about head position and movement.

EXPLORING THE SOMATOSENSORY SYSTEM

We have yet to explore a major part of the somatosensory system—the somatosensory cortex. As illustrated in Figure 10-31, there are two main somatosensory cortex areas. The primary somatosensory cortex is the area that receives projections from the thalamus. It consists of Brodmann's areas 3-1-2 (all shaded red, below). This area begins the process of constructing perceptions from somatosensory information. It mainly consists of the postcentral gyrus just behind the central fissure, which means that the primary somatosensory cortex is adjacent to the primary motor cortex. The secondary somatosensory cortex (Brodmann's areas 5 and 7, shaded orange and yellow, below) is located in the parietal lobe just behind the primary somatosensory cortex.

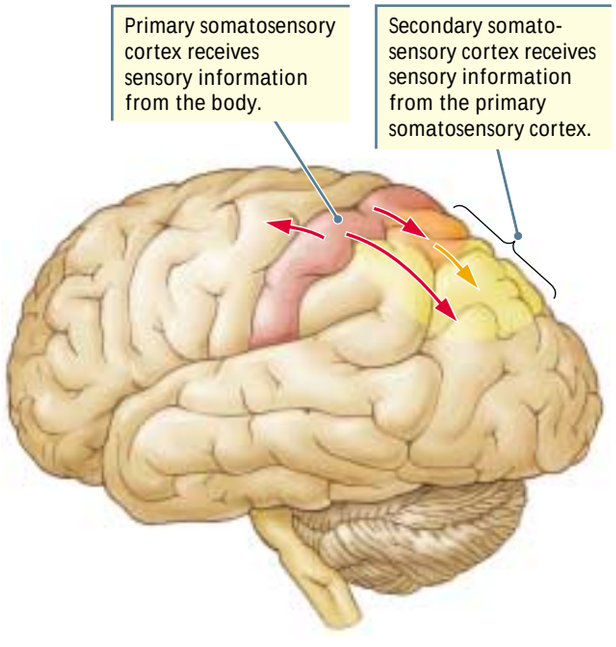


Figure 10-31

Locations of the primary and the secondary somatosensory cortex. Information in the primary somatosensory cortex is sent to the secondary somatosensory cortex. Stimulation of the primary somatosensory cortex produces sensations that are referred to appropriate body parts. As is the motor cortex, the primary somatosensory cortex is organized as a homunculus, with a large area representing body parts that are highly sensitive.

The Somatosensory Homunculus

In his studies of human patients undergoing brain surgery, Wilder Penfield electrically stimulated the somatosensory cortex and recorded the patients' responses. Stimulation at some sites elicited sensations in the foot, whereas stimulation of other sites produced sensations in a hand, the body, or the face. By mapping these responses, Penfield was able to construct a somatosensory homunculus in the cortex. This homunculus looks very similar to the motor homunculus in that the areas of the body that are most sensitive to sensory stimulation are accorded a relatively larger area of somatosensory cortex.

Using slightly different recording techniques in monkeys, John Kaas (1987) stimulated sensory receptors on the body and recorded the activity of cells in the sensory cortex. He found that the somatosensory cortex is actually composed of four representations of the body, each associated with a certain class of sensory receptors. In a progression across the cortex from front to back, as shown in Figure 10-32, area 3a cells are responsive to muscle receptors, area 3b cells are responsive to skin receptors, area 1 cells are responsive to rapidly adapting skin receptors, and area 2 cells are responsive to deep tissue pressure and joint receptors. In other studies, Hiroshi Asanuma (1989) and his coworkers found still another sensory representation in the motor cortex (area 4) in which cells responded to muscle and joint receptors.

Research by Vernon Mountcastle (1978) showed that cells in the somatosensory cortex are arranged in functional columns running from layer I to layer VI, similar to columns found in the visual cortex. Every cell in a column responds to a single class of receptors. Some columns of cells are activated by rapidly adapting skin receptors, others by slowly adapting skin receptors, still others by pressure receptors, and so forth. All neurons in a column receive information from the same local area of skin. In this way, neurons lying within a column seem to provide an elementary functional module of the somatosensory cortex.

Single-cell recordings in the sensory cortex suggest a hierarchical organization, with basic sensations being combined to form more complex perceptions. This combining of information occurs as areas 3a and 3b project onto area 1, which in turn projects onto area 2. With each successive relay of information, both the size of the pertinent receptive fields and the synthesis of somatosensory modalities increase. For example, whereas a cell in area 3a or 3b may respond to activity in only a certain area on a certain finger, cells in area 1 may respond to similar information from a number of different fingers. At the next level of synthesis, cells in area 2 may respond to stimulation in a number of different locations on a number of different fingers, as well as to stimulation from different kinds of receptors. Thus, area 2 contains neurons that are responsive to movement, orientation, and direction of movement, all of which are properties that we perceive when we hold an object in our hands and manipulate it.

That the different kinds of somatosensory information are both separated and combined in the cortex raises the question of why both segregation and synthesis are needed. One reason why sensory information remains segregated at the level of the cortex could be that we often need to distinguish between different kinds of sensory stimuli coming from different kinds of sources. For example, we need to be able to tell the difference between tactile stimulation on the surface of the skin, which is usually produced by some external agent, and stimulation coming from muscles, tendons, and joints, which is likely produced by our own movements. Yet, at the same time, we also often need to know about the combined sensory properties of a stimulus. For instance, when we manipulate an object, it is useful to "know" the object both in regard to its sensory properties, such as temperature and texture, and in regard to the movements that we make as we handle it. For this reason, the cortex provides for somatosensory synthesis, too.

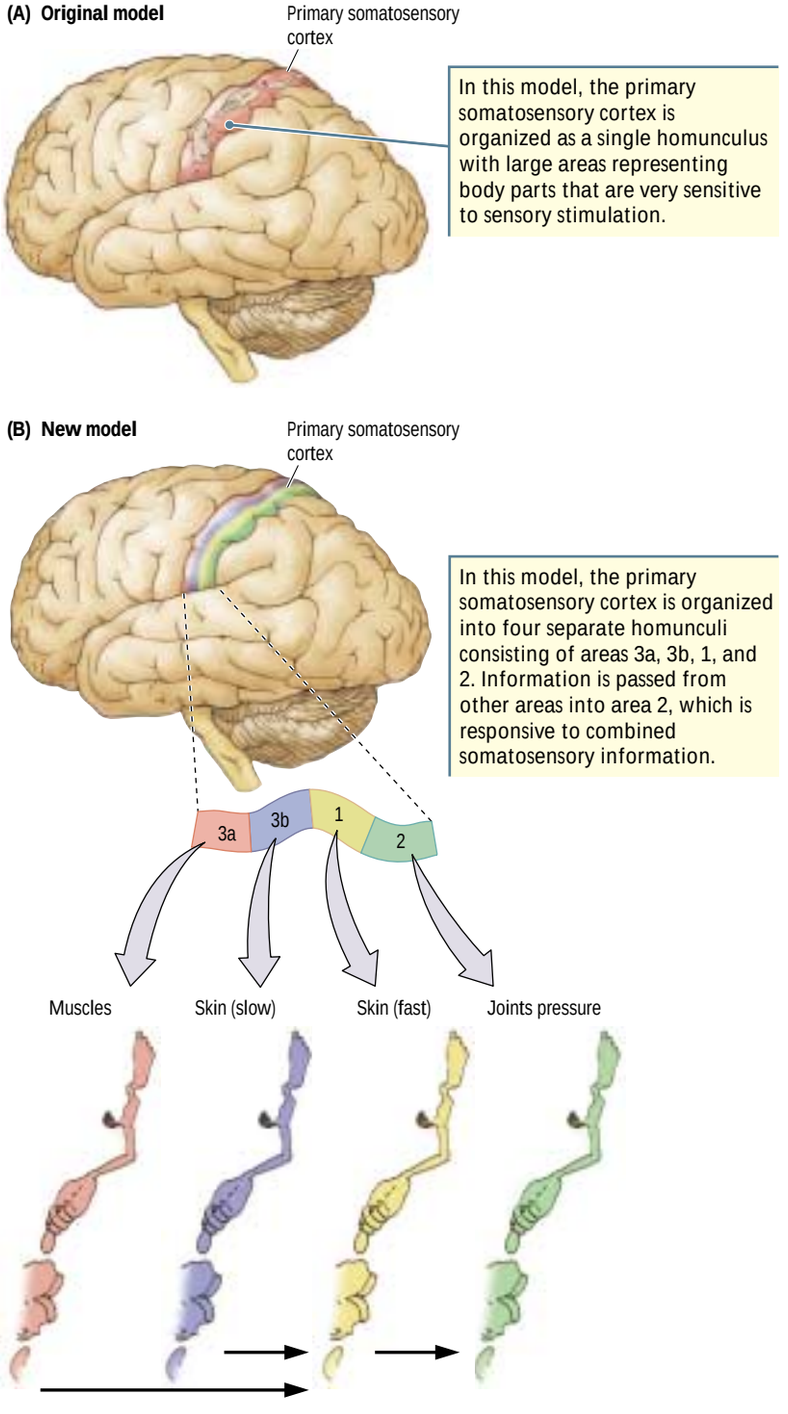


Figure 10-32

A comparison of two models of the somatosensory cortex organization: **(A)** a single-homunculus model and **(B)** a four-homunculi model, which is based on the stimulation of sensory receptors on the body surface and recording from the somatosensory cortex. In the model in (B), four separate homunculi are obtained in the primary somatosensory cortex. Neurons in area 3a are responsive to muscle length, neurons in area 3b are responsive to slowly adapting receptors in the skin, neurons in area 1 are responsive to rapidly adapting skin receptors, and neurons in area 2 are responsive to joint and pressure sensations. Information is passed from the other areas into area 2, in which neurons that are responsive to a number of kinds of somatosensory information (multimodal neurons) are found.

The Effects of Damage to the Somatosensory Cortex

Damage to the primary somatosensory cortex impairs the ability to make even simple sensory discriminations and movements, as was clearly demonstrated in a study by Suzanne Corkin and her coworkers (1970), who examined patients with cortical lesions that included most of area 3-1-2 in one hemisphere. The researchers mapped the

sensory cortices of these patients before their undergoing elective surgery for removal of a carefully defined piece of that cortex, including the hand area. The patients' sensory and motor skills in both hands were tested on three different occasions: before the surgery, shortly after the surgery, and almost a year afterward. The tests included pressure sensitivity, two-point touch discrimination, position sense (reporting the direction in which a finger was being moved), and haptic sense (using touch to identify objects, such as a pencil, a penny, eyeglasses, and so forth). For all the sensory abilities tested, the surgical lesions produced a severe and seemingly permanent deficit in the contralateral hand. Sensory thresholds, proprioception, and hapsis were all greatly impaired. The results of other studies in both humans and animals have shown that damage to the somatosensory cortex also impairs simple movements. For example, limb use in reaching for an object is impaired, as is the ability to shape the hand to hold an object (Leonard et al., 1991).

Interestingly, the somatosensory cortex can dramatically reorganize itself after the cutting of sensory fibers (deafferentation). In 1991, Tim Pons and his coworkers reported a dramatic change in the somatosensory maps of monkeys that had had the ganglion cells for one arm deafferented a number of years earlier. The researchers had wanted to develop an animal model of damage to sensory nerves that could be a source of insight into human injuries, but they were interrupted by a legal dispute with an animal advocacy group. Years later, as the health of the animals declined, a court injunction allowed the mapping experiment to be conducted. Pons and his coworkers discovered that the area of the somatosensory cortex that had previously represented the arm no longer did so. Light touches on the lower face of a monkey now activated cells in what had previously been the cortical arm region. As illustrated in Figure 10-33, the face area had expanded by as much as 10 to 14 millimeters, virtually doubling its original size by entering the arm area. This massive change was completely unexpected. The stimuli–response patterns associated with the new expanded facial area of the cortex appeared indistinguishable from those associated with the original facial area. Furthermore, the trunk area, which bounded the other side of the cortical arm area, did not expand into the vacated arm area.

What could account for this expansion of the face area into the arm area? One possibility is that axons grew across the cortex from the face area into the arm area, but no evidence supports this possibility. It is also possible that the thalamic neurons representing the face area projected axon collaterals to the cortical neurons representing the arm area. These collaterals might be preexisting or they might be new growths subsequent to deafferentation. There is evidence for preexisting collaterals that are not normally active, but these collaterals would probably not be able to extend far enough to account for all of the cortical reorganization. A third possibility is that, within the dorsal columns, face-area neurons projected collaterals to arm-area neurons. These neurons are close together, so the collaterals need travel only a millimeter or so. Whatever the mechanism, the very dramatic cortical reorganization observed in this study eventually had far-reaching consequences for understanding other remarkable phenomena. We will return to this story in Chapter 13, which looks at how the brain changes in response to experience.

The Somatosensory Cortex and Complex Movement

This chapter began with a description of the remarkable painting skills of Kamala. To accomplish this task, Kamala first needs to have some idea of what she wants to paint. She must then execute the movements required to apply paint to her canvas, and she must also use somatosensory information to confirm that she has produced the move-

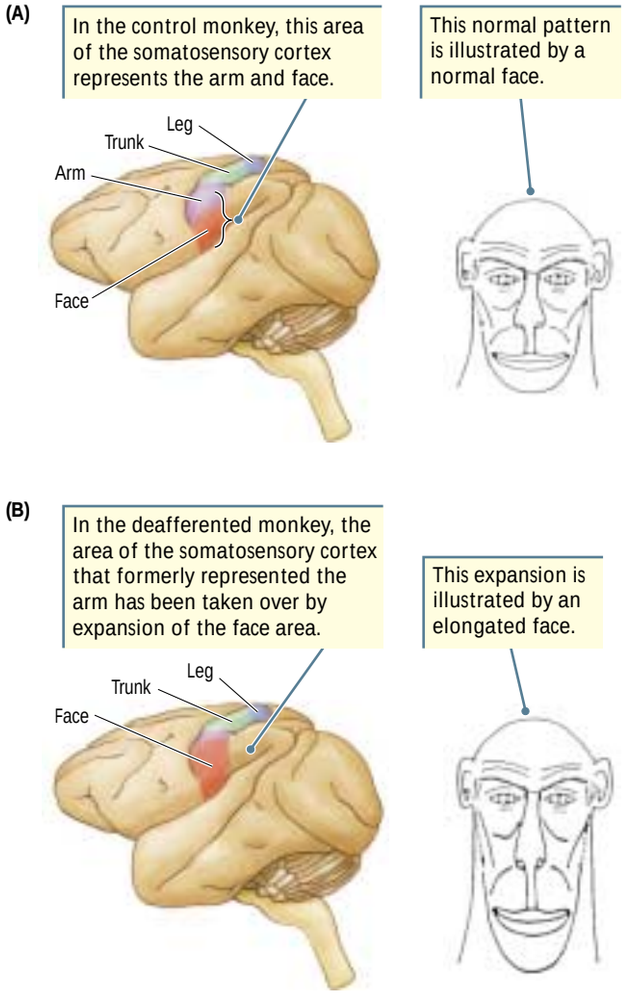


Figure 10-33

Somatosensory cortex of a control monkey **(A)** and a monkey that had received arm deafferentation **(B)**. In the deafferented monkey, the area of sensory cortex that had formerly represented the hand and arm area of the body now represents the face. Thus, the area that represents the face is greatly expanded, as illustrated by the elongated face illustrated to the right. The face representations are shown right side up for simplicity. Electrical recordings show that only the lower face area has expanded into the arm and hand region.

Adapted from "Massive Cortical Reorganization After Sensory Deafferentation in Adult Macaques," by T. P. Pons, P. E. Garraghty, A. K. Ommaya, J. H. Kaas, and M. Miskin, 1991, *Science, 252,* p. 1858.

ments that she intended. So to paint, or to perform virtually any other complex movement, the motor system and the somatosensory system must closely interact. In this final section of the chapter, we explore that interaction.

The secondary somatosensory cortex plays an important role in confirming which movements have already taken place and in deciding which movements should follow. Damage to the secondary somatosensory cortex does not disrupt the plans for making movements, but it does disrupt how the movements are performed, leaving them fragmented and confused. The inability to complete a plan of action accurately is called apraxia (from the Greek words for "not" and "action"). The following example highlights the symptoms of apraxia:

> A woman with a biparietal lesion (that is, a lesion in both sides of the secondary somatosensory cortex) had worked for years as a fish-filleter. With the development of her symptoms, she began to experience difficulty in carrying on with her job. She did not seem to know what to do with her knife. She would stick the point in the head of a fish, start the first stroke, and then come to a stop. In her own mind she knew how to fillet fish, but yet she could not execute the maneuver. The foreman accused her of being drunk and sent her home for mutilating fish.
>
> The same patient also showed another unusual phenomenon that might possibly be apraxic in nature. She could never finish an undertaking. She

Apraxia. An inability to make voluntary movements in the absence of paralysis or other motor or sensory impairment, especially an inability to make proper use of an object.

would begin a job, drop it, start another, abandon that one, and within a short while would have four or five uncompleted tasks on her hands. This would cause her to do such inappropriate actions as putting the sugar bowl in the refrigerator, and the coffeepot inside the oven. (Critchley, 1953, pp. 158–159)

How does an intact secondary somatosensory cortex contribute to the organization of movement? Recall from Chapter 8 that visual information influences movement through the dorsal and ventral streams. The dorsal stream, working without conscious awareness, provides vision for action, as when we use the visual form of a cup to shape a hand so as to grasp that cup. The ventral stream, in contrast, works with conscious awareness and provides the vision needed to identify objects. As Figure 10-34 illustrates, the secondary somatosensory cortex participates in both these visual streams. The dorsal visual stream projects to the secondary somatosensory cortex and then to the prefrontal cortex. In this way, visual information is integrated with somatosensory information to produce movements that are appropriately shaped and directed for their targets. Much less is known about how the secondary somatosensory cortex contributes to the ventral stream, but it is likely that somatosensory information about the identity of objects and completed movements is relayed by the ventral stream to the prefrontal cortex. The prefrontal cortex can then select the actions that should follow those that are already completed.

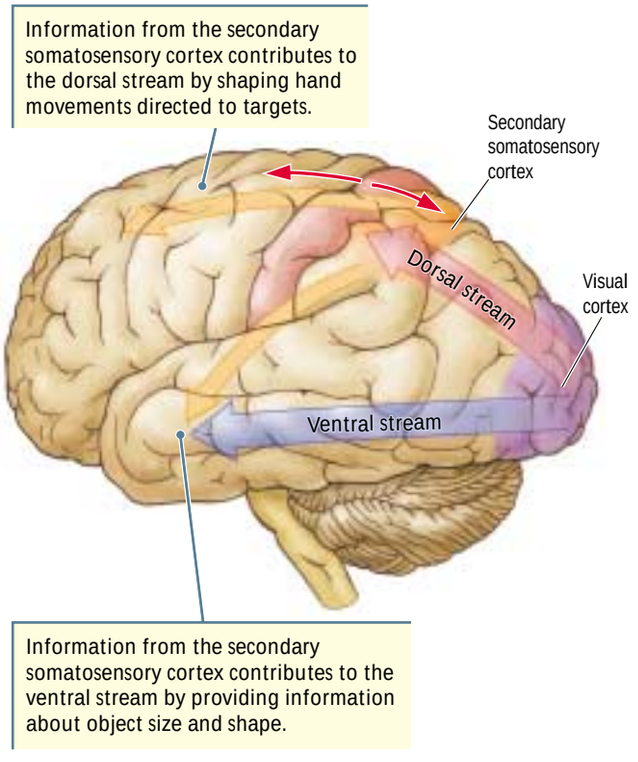


Figure 10-34

The somatosensory cortex contributes to information flow in dorsal and ventral streams.

A close interrelation between the somatosensory system and the motor system exists at all levels of the nervous system. It can be seen in the spinal cord, where sensory information contributes to spinal reflexes. It can also be seen in the brainstem, where various species-specific behaviors, such as attack, withdrawal, and grooming, require both appropriate patterns of movement and appropriate sensory information. The close interrelation is found as well at the level of the neocortex, where skilled movements elicited by the motor regions of the frontal lobes require information about actions that have just taken place and about objects that have been or could be manipulated. In short, an interaction between the motor cortex, which decides what should be done, and the sensory cortex, which knows what has been done, is central to how the brain produces movement.

In Review

The primary somatosensory cortex is arranged as a series of homunculi, each of which represents a different body sense. This area of the cortex provides information to the secondary somatosensory cortex, which in turn contributes to the dorsal and ventral streams. Damage to the secondary somatosensory cortex produces apraxia, an inability to complete a series of movements. A person with this condition has trouble knowing both what action has just been completed and what action should follow in a movement sequence.

SUMMARY

1. *How is the motor system organized?* The organization of movement is hierarchical, with almost all of the brain contributing to it in some way. The forebrain plans, organizes, and initiates movements, whereas the brainstem coordinates regulatory functions, such as eating and drinking, and controls neural mechanisms that maintain posture and produce locomotion. Many reflexes are organized at the level of the spinal cord and occur without any involvement of the brain.
2. *How is the motor cortex organized?* Maps produced by stimulating the motor cortex show that it is organized topographically as a homunculus, with parts of the body capable of fine movements associated with larger regions of motor cortex. There are two pathways from the motor cortex to the spinal cord, the lateral corticospinal tract and the ventral corticospinal tract. The lateral corticospinal tract consists of axons from the digit, hand, and arm regions of the motor cortex. The tract synapses with spinal interneurons and motor neurons located laterally in the spinal cord, on the side of the cord opposite the side of the brain on which the corticospinal tract started. The ventral corticospinal tract consists of axons from the trunk region of the motor cortex. This tract synapses with interneurons and motor neurons located medially in the spinal cord, on the same side of the cord as the side of the brain on which the corticospinal tract started. Interneurons and motor neurons of the spinal cord also are topographically organized, with more laterally located motor neurons projecting to digit, hand, and arm muscles and more medially located motor neurons projecting to trunk muscles.
3. *How do motor-cortex neurons produce movement?* Movements are organized as synergies, or movement patterns. Motor neurons initiate movement, produce movement, control the force of movement, and indicate movement direction. Different species of animals have topographic maps in which areas of the body capable of the most-skilled movements have the largest motor-cortex representation. Disuse of a limb, such as that which might follow motor-cortex injury, results in shrinkage of that limb's representation in the motor cortex. This shrinkage of motor-cortex representation can be prevented, however, if the limb can be somehow forced into use.
4. *How do the basal ganglia and the cerebellum contribute to controlling movement?* Damage to the basal ganglia or to the cerebellum results in abnormalities of movement. This result tells us that both these brain structures somehow participate in movement control. The results of experimental studies suggest that the basal ganglia regulate the force of movements whereas the cerebellum plays a role in movement timing and in maintaining the accuracy of movements.
5. *How is the somatosensory system organized?* The somatosensory system is distributed throughout the entire body and consists of more than 20 types of specialized receptors, each of which is sensitive to a particular form of mechanical energy. Each somatosensory receptor projecting from skin, muscles, tendons, or joints is associated with a dorsal-root ganglion neuron that carries the sensory information into the brain. Fibers carrying proprioceptive (location and movement) information and haptic (touch and pressure) information ascend the spinal cord as the dorsal spinothalamic tract. These fibers synapse in the dorsal-column nuclei at the base of the brain, at which point axons cross over to the other side of the brainstem to form the medial lemniscus, which ascends to the ventrolateral thalamus. Most of the ventrolateral thalamus cells project to the somatosensory cortex. Nocioceptive (pain and temperature) dorsal-root ganglion neurons synapse on entering the spinal cord. Their relay neurons cross the spinal cord to ascend to the thalamus as the ventral spinothalamic tract. Because there are two somatosensory

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- **www.circleoffriends.org**

Go to this site to learn more about current research on spinal cord injury.

- **tsa.mgh.harvard.edu**

Learn more about this misunderstood disorder at the home page of the Tourette's Syndrome Association.

On your CD-ROM you'll be able to quiz yourself on your comprehension of the chapter. The module in your CD on Control of Movement offers interactive illustrations to reinforce your understanding of key concepts.

pathways that take somewhat different routes, unilateral spinal-cord damage impairs proprioception and hapsis ipsilaterally below the site of injury and nociception contralaterally below the site.

6. *How is somatosensory information represented in the neocortex?* The somatosensory system is represented topographically as a homunculus in the primary somatosensory region of the parietal cortex (area 3-1-2) such that the most sensitive parts of the body are accorded the largest regions of neocortex. A number of homunculi represent different sensory modalities, and these regions are hierarchically organized. If sensory input from a part of the body is cut off from the cortex by damage to sensory fibers, adjacent functional regions of the sensory cortex can expand into the now-unoccupied region.
7. *How are the somatosensory system and the motor system interrelated?* The somatosensory system and the motor system are interrelated at all levels of the nervous system. At the level of the spinal cord, sensory information contributes to motor reflexes; in the brainstem, sensory information contributes to complex regulatory movements. At the level of the neocortex, sensory information is used to record just-completed movements, as well as to represent the sizes and shapes of objects. The somatosensory cortex contributes to the dorsal visual stream to direct hand movements to targets. The somatosensory cortex also contributes to the ventral visual stream to create representations of external objects.

KEY TERMS

apraxia, p. 393
cerebral palsy, p. 361
deafferented, p. 383
dissolution, p. 357
glabrous skin, p. 381
hapsis, p. 382
homunculus, p. 366
hyperkinetic symptom, p. 376
hypokinetic symptom, p. 376
motor sequence, p. 358
nocioception, p. 382
pain gate, p. 386
paraplegia, p. 361
proprioception, p. 382
quadraplegia, p. 362
rapidly adapting receptor, p. 382
referred pain, p. 387
scratch reflex, p. 363
slowly adapting receptor, p. 382
synergy, p. 370
topographic organization, p. 366
vestibular system, p. 388

REVIEW QUESTIONS

1. How are the somatosensory system and the motor system related?
2. Describe the pathways that convey somatosensory information to the brain.
3. Describe the pathways that convey motor instructions to the spinal cord.
4. What are the contributions of the cortex, the basal ganglia, and the cerebellum to movement?
5. Describe two theories of how our motor cortex moves a hand to a target to grasp it.
6. Describe the changes that the somatosensory cortex and the motor cortex might undergo in response to injury to the cortex or to a limb.

FOR FURTHER THOUGHT

1. Why is the somatosensory system so much more intimately linked to movement than the other sensory systems are?
2. Why might the dorsal and ventral streams be separate systems for controlling hand movements?

RECOMMENDED READING

Asanuma, H. (1989). *The motor cortex*. New York: Raven Press. An excellent summary of the motor system by a scientist who made important advances in studying the role of the motor cortex in behavior.

Cole, J. (1995). *Pride and a daily marathon*. London: MIT Press. At the age of 19 Ian Waterman was struck down by a rare neurological condition that deprived him of joint position and proprioception. This is the story of how he gradually adapted to his strange condition by using vision and elaborate tricks to monitor his every movement and regain his life.

Melzack, R. (1973). *The puzzle of pain*. New York: Basic Books. For ages, physicians and scientists have attempted with little success to understand and control pain. Here one of the world's leading researchers in the field of pain theory and treatment presents a totally readable book to unravel the mystery of pain.

Sacks, O. (1974). *Awakenings*. New York: Vintage Books. This prize-winning book presents a fascinating account of one of the mysteries of the motor system, how the great flu of the 1920s produced the Parkinsonism that developed as the aftermath of the "sleeping sickness." This story presents wonderful insights into the function of the motor system.

Porter, R., & Lemon, R. (1993). *Corticospinal function and voluntary movement*. Oxford: Clarendon Press. This book tells the story of the human brain's great pathway, the corticospinal tract.