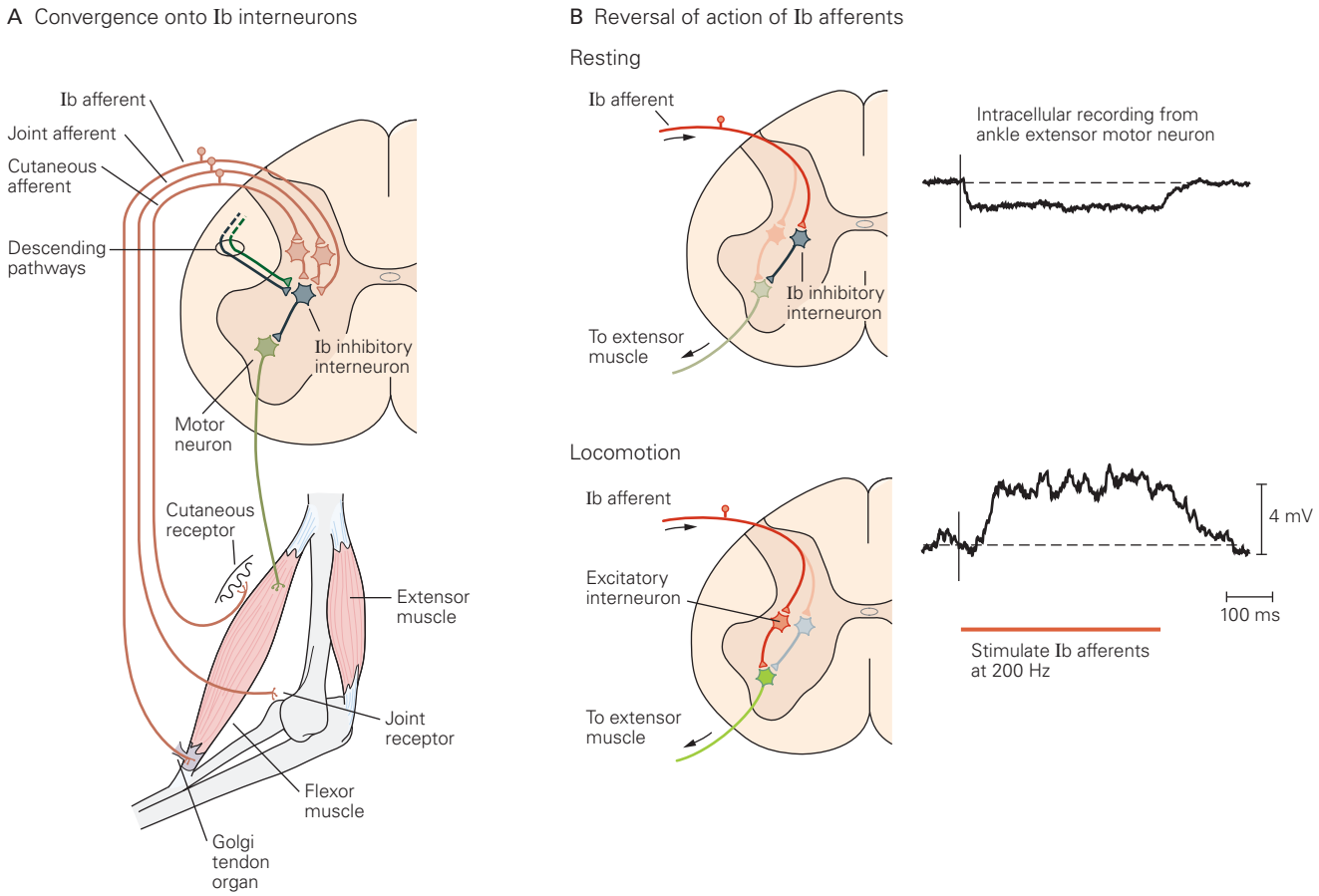

Figure 32–8 The reflex actions of Ib sensory fibers from Golgi tendon organs are modulated during locomotion.

A. The Ib inhibitory interneuron receives input from tendon organs, muscle spindles (not shown), joint and cutaneous receptors, and descending pathways.

B. The action of Ib sensory fibers on extensor motor neurons is reversed from inhibition to excitation when walking is initiated. When the animal is resting, stimulation of Ib fibers from the ankle extensor muscle inhibits ankle extensor motor neurons through Ib inhibitory interneurons, as shown by the hyperpolarization in the record. During walking, the Ib inhibitory interneurons are inhibited while excitatory interneurons that receive input from Ib sensory fibers are facilitated by the command system for walking, thus opening a Ib excitatory pathway from the Golgi tendon organs to motor neurons.

fast withdrawal only at the wrist and elbow, whereas touching a very hot stove invariably leads to a forceful contraction at all joints, leading to rapid withdrawal of the entire limb. The duration of the reflex usually increases with stimulus intensity, and the contractions produced in a flexion reflex always outlast the stimulus.

Because of the similarity of the flexion-withdrawal reflex to stepping, it was once thought that the flexion reflex is important in producing contractions of flexor muscles during walking. We now know, however, that a major component of the neural control system for walking is a set of intrinsic spinal circuits that do not require sensory stimuli (Chapter 33). Nevertheless, in mammals, the intrinsic spinal circuits that control walking share many of the interneurons involved in flexion reflexes.

Convergence of Sensory Inputs on Interneurons Increases the Flexibility of Reflex Contributions to Movement

The Ib inhibitory interneuron is not the only interneuron that receives convergent input from many different sensory modalities. An enormous diversity of sensory information converges on interneurons in the spinal cord, enabling them to integrate information from muscle, joints, and skin.

Interneurons activated by groups I and II sensory fibers have received special attention. It was thought

for some time that excitatory and inhibitory interneurons activated by group II fibers could be distinguished from those activated by group Ib afferents, but it is now believed that this distinction has to be abandoned and that groups I and II fibers converge on common populations of interneurons that integrate force and length information from the active muscle and thereby help coordinate muscle activity according to the length of the muscle, its activity level, and the external load.

Sensory Feedback and Descending Motor Commands Interact at Common Spinal Neurons to Produce Voluntary Movements

As pointed out by Michael Foster in his 1879 physiology textbook, it must be an "economy to the body" that the will should make use of the networks in the spinal cord to generate coordinated movements "rather than it should have recourse to an apparatus of its own of a similar kind." Research in the subsequent 140 years has confirmed this conjecture.

The first evidence came from intracellular recordings of synaptic potentials elicited in cat spinal motor neurons by combined and separate stimulation of sensory fibers and descending pathways. When separate stimuli are reduced in intensity to just below threshold for evoking a synaptic potential, combining the stimulations at appropriate intervals makes the synaptic potential reappear. This provides evidence of convergence of the sensory fibers and the descending pathways onto common interneurons in the reflex pathway (see Figure 13–14). Direct recordings from spinal interneurons have confirmed this, as have noninvasive Hoffmann reflex tests in human subjects (Figure 32–9).

Direct evidence that sensory feedback helps to shape voluntary motor commands through spinal reflex networks in humans comes from experiments in which sensory activity in length- and force-sensitive afferents has suddenly been reduced or abolished. This can be done by suddenly unloading or shortening a muscle during a voluntary contraction. The short latency of the consequent reduction in muscle activity can only be explained by sensory activity through a reflex pathway that directly contributes to the muscle activity.

Muscle Spindle Sensory Afferent Activity Reinforces Central Commands for Movements Through the Ia Monosynaptic Reflex Pathway

Stretch reflex pathways can contribute to the regulation of motor neurons during voluntary movements and during maintenance of posture because they form closed feedback loops. For example, stretching a muscle increases activity in spindle sensory afferents, leading to muscle contraction and consequent shortening of the muscle. Muscle shortening in turn leads to decreased activity in spindle afferents, reduction of muscle contraction, and lengthening of the muscle.

The stretch reflex loop thus acts continuously—the output of the system, a change in muscle length, becomes the input—tending to keep the muscle close to a desired or reference length. The stretch reflex pathway is a negative feedback system, or *servomechanism*, because it tends to counteract or reduce deviations from the reference value of the regulated variable.

In 1963, Ragnar Granit proposed that the reference value in voluntary movements is set by descending signals that act on both alpha and gamma motor neurons. The rate of firing of alpha motor neurons is set to produce the desired shortening of the muscle, and the rate of firing of gamma motor neurons is set to produce an equivalent shortening of the intrafusal fibers of the muscle spindle. If the shortening of the whole muscle is less than what is required by a task, as when the load is greater than anticipated, the sensory fibers increase their firing rate because the contracting intrafusal fibers are stretched (loaded) by the relatively greater length of the whole muscle. If shortening is greater than necessary, the sensory fibers decrease their firing rate because the intrafusal fibers are relatively slackened (unloaded) (Figure 32–10A).

In theory, this mechanism could permit the nervous system to produce movements of a given distance without having to know in advance the actual load or weight being moved. In practice, however, the stretch reflex pathways do not have sufficient control over motor neurons to overcome large unexpected loads. This is immediately obvious if we consider what happens when we attempt to lift a heavy suitcase that we believe to be empty. Automatic compensation for the greater-than-anticipated load does not occur. Instead, we have to pause briefly to plan a new movement with much greater muscle activation.

Strong evidence that alpha and gamma motor neurons are co-activated during voluntary human movement comes from direct measurements of the activity of the sensory fibers from muscle spindles. In the late 1960s, Åke Vallbo and Karl-Erik Hagbarth developed microneurography, a technique for recording from the largest afferent fibers in peripheral nerves. Vallbo later found that during slow movements of the fingers the large-diameter Ia fibers from spindles in the contracting muscles increase their rate of firing even when the muscle shortens as it contracts (Figure 32–10B). This occurs because the gamma motor neurons, which have

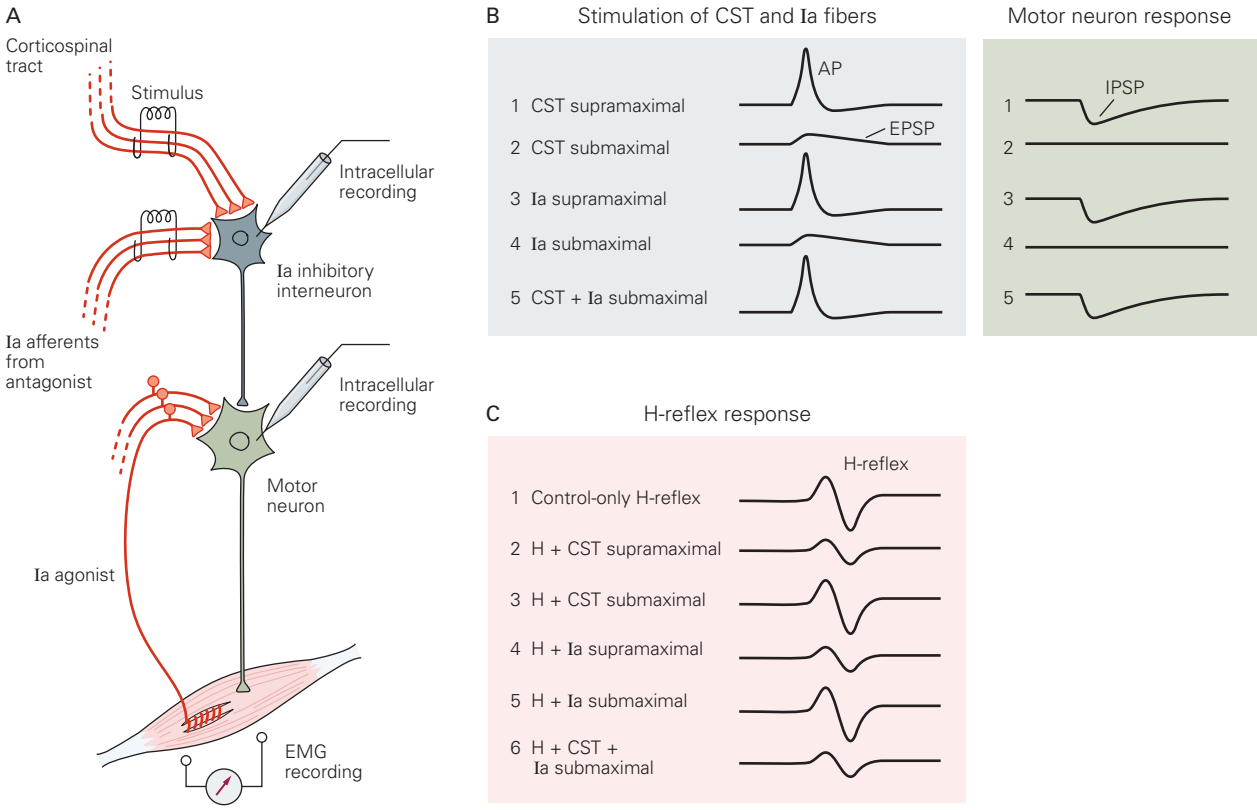

Figure 32–9 The spatial summation technique demonstrates how signals from descending inputs and spinal networks are integrated. This technique was introduced originally for investigation of spinal circuits in the cat in the 1950s, but it is also the basis of later investigations of the human spinal mechanisms of motor control. It relies on the spatial summation of synaptic inputs (see Figure 13–14), as illustrated here using the reciprocal Ia inhibitory pathway and corticospinal tract (**CST**).

A. The diagram shows the experimental setups for testing for convergence of excitatory reciprocal Ia and corticospinal pathways onto Ia inhibitory interneurons in the spinal cord.

B. In acute experiments on the cat spinal cord, supramaximal stimuli were applied separately to corticospinal fiber tracts (**1**) and Ia axons (**3**); each stimulus elicited an inhibitory postsynaptic potential (**IPSP**) in the motor neuron. Next, the intensities of the two stimuli were reduced to just submaximal levels, at which point each pathway failed to elicit an IPSP in the motor neuron (**2**, **4**). Then, when the two sets of submaximal stimuli were paired, they elicited an IPSP in the motor neuron (**5**), leading to the conclusion that the two input pathways converge on the same interneurons. This was confirmed by direct recording from a Ia inhibitory interneuron. (**AP**, action potential).

C. In humans, direct intracellular recording from interneurons and motor neurons is not possible, but recording of H-reflexes (Box 32–4, Figure 32–4) and transcutaneous stimulation of the corticospinal tract have provided indirect evidence for convergence similar to that demonstrated in cats (see part **B**). The electromyogram (**EMG**) record of the H-reflex provides a measure of the excitability of the spinal motor neurons (**1**). When the CST and antagonist Ia fibers were stimulated separately at supramaximal levels, the H-reflex amplitude was diminished due to the compound IPSPs elicited in the motor neurons (**2**, **4**). Next, the stimuli to these two excitatory pathways to the inhibitory interneurons were reduced until neither stimulus alone elicited a reduction in amplitude of the H-reflex (**3**, **5**). Then, the two submaximal stimuli were timed to produce synchronous subthreshold excitatory postsynaptic potentials (**EPSPs**) in the inhibitory interneurons (**6**). Because this protocol caused suppression of the H-reflex, one may conclude that the CST and Ia afferents converge on the same Ia inhibitory interneurons.

direct excitatory connections with spindles, are coactivated with alpha motor neurons.

Furthermore, when subjects attempt to make slow movements at a constant velocity, the firing of the Ia fibers mirrors the small deviations in velocity in the trajectory of the movements (sometimes the muscle shortens quickly and at other times more slowly). When the velocity of flexion increases transiently, the rate of firing in the fibers decreases because the muscle is shortening more rapidly and therefore exerts less tension on the intrafusal fibers. When the velocity decreases, firing increases because the muscle is

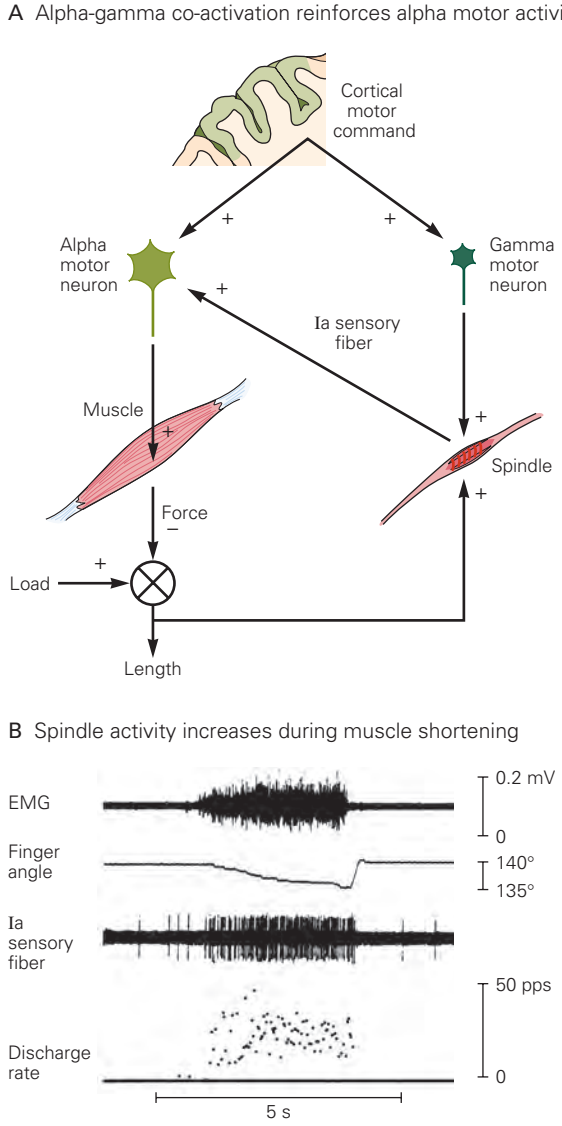

Figure 32–10 Co-activation of alpha and gamma motor neurons.

A. Co-activation of alpha and gamma motor neurons by a cortical motor command allows feedback from muscle spindles to reinforce activation in the alpha motor neurons. Any disturbance during a movement alters the length of the muscle and thus changes the activity in the sensory fibers from the spindles. The changed spindle input to the alpha motor neuron compensates for the disturbance.

B. The discharge rate in the Ia sensory fiber from a spindle increases during slow flexion of a finger. This increase depends on alpha-gamma co-activation. If the gamma motor neurons were not active, the spindle would slacken, and its discharge rate would decrease as the muscle shortened. (**EMG**, electromyogram; **PPS**, pulses/s) (Adapted, with permission, from Vallbo 1981.)

shortening more slowly, and therefore, the relative tension on the intrafusal fibers increases. This information can be used by the nervous system to compensate for irregularities in the movement trajectory by exciting the alpha motor neurons.

Modulation of Ia inhibitory Interneurons and Renshaw Cells by Descending Inputs Coordinate Muscle Activity at Joints

Reciprocal innervation is useful not only in stretch reflexes but also in voluntary movements. Relaxation of the antagonist muscle during a movement enhances speed and efficiency because the muscles that act as prime movers are not working against the contraction of opposing muscles.

The Ia inhibitory interneurons receive inputs from collaterals of the axons of neurons in the motor cortex that make direct excitatory connections with spinal motor neurons. This organizational feature simplifies the control of voluntary movements, because higher centers do not have to send separate commands to the opposing muscles.

It is sometimes advantageous to contract both the prime mover and the antagonist at the same time. Such *co-contraction* has the effect of stiffening the joint and is most useful when precision and joint stabilization are critical. An example of this phenomenon is the co-contraction of flexor and extensor muscles of the elbow immediately before catching a ball. The Ia inhibitory interneurons receive both excitatory and inhibitory signals from all of the major descending pathways (Figure 32–11A). By changing the balance of excitatory and inhibitory inputs onto these interneurons, supraspinal centers can modulate reciprocal inhibition of muscles and enable co-contraction, thus controlling the relative amount of joint stiffness to meet the requirements of the motor act.

The activity of spinal motor neurons is also regulated by another important class of inhibitory interneurons, the *Renshaw cells.* Excited by collaterals of the axons of motor neurons and receiving significant synaptic input from descending pathways, Renshaw cells make inhibitory synaptic connections with several populations of motor neurons, including the motor neurons that excite them, as well as Ia inhibitory interneurons (Figure 32–11B). The connections with motor neurons form a negative feedback system that regulates the firing rate of the motor neurons, whereas the connections with the Ia inhibitory interneurons regulate the strength of inhibition of antagonistic motor neurons, for instance in relation to co-contraction of antagonists. The distribution of projections from Renshaw

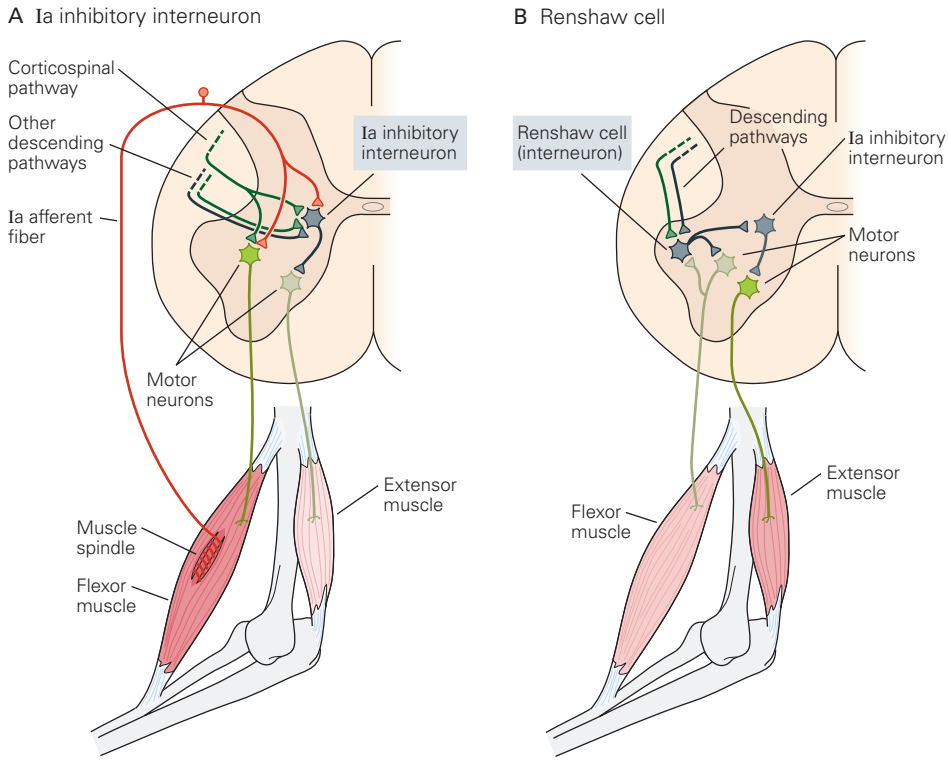

Figure 32–11 Inhibitory spinal interneurons coordinate reflex actions.

A. The Ia inhibitory interneuron regulates contraction in antagonist muscles in stretch reflex circuits through its divergent contacts with motor neurons. In addition, the interneuron receives excitatory and inhibitory inputs from corticospinal and other descending pathways. A change in the balance of these supraspinal signals allows the interneuron to coordinate co-contractions in antagonist muscles at a joint.

B. The Renshaw cell produces recurrent inhibition of motor neurons. These interneurons are excited by collaterals from motor neurons and inhibit those same motor neurons. This negative feedback system regulates motor neuron excitability and stabilizes firing rates. Renshaw cells also send collaterals to synergist motor neurons (not shown) and Ia inhibitory interneurons that synapse on antagonist motor neurons. Thus, descending inputs that modulate the excitability of the Renshaw cells adjust the excitability of all the motor neurons that control movement around a joint.

cells to different motor nuclei also facilitate that muscle activity is coordinated in functional synergies during movement.

Transmission in Reflex Pathways May Be Facilitated or Inhibited by Descending Motor Commands

As we have seen, in an animal at rest, the Ib sensory fibers from extensor muscles have an inhibitory effect on homonymous motor neurons. During locomotion, they produce an excitatory effect on those same motor neurons because transmission in the disynaptic inhibitory pathway is depressed (Figure 32–8B), while at the same time transmission through excitatory interneurons is facilitated.

This phenomenon, called *state-dependent reflex reversal*, illustrates how transmission in spinal circuit is regulated by descending motor commands to meet the changing requirements during movement. By favoring transmission through excitatory pathways from the load-sensitive Golgi tendon organs, the descending motor commands ensure that feedback from the active muscles automatically facilitates the activation of the muscles, thereby greatly simplifying the task for supraspinal centers.

State-dependent reflex reversal has also been demonstrated in humans. Stimulation of skin and muscle afferents from the foot produces facilitation of muscles that lift the foot early in the swing phase, but suppresses activity of the same muscles late in the swing phase. Both effects make good functional sense. Early in the swing phase, positive feedback from the foot will help to lift the foot over an obstacle, whereas suppression of the same muscles in late swing will help to lower the foot quickly to the ground so that the obstacle may be passed using the opposite leg first.

Descending Inputs Modulate Sensory Input to the Spinal Cord by Changing the Synaptic Efficiency of Primary Sensory Fibers

In the 1950s and early 1960s, John C. Eccles and his collaborators demonstrated that monosynaptic excitatory postsynaptic potentials (EPSPs) elicited in cat spinal motor neurons by stimulation of Ia sensory fibers become smaller when other Ia fibers are stimulated. This led to the discovery in the spinal cord of several groups of GABAergic inhibitory interneurons that exert presynaptic inhibition of primary sensory neurons (Figure 32–12). Some interneurons inhibit mainly Ia sensory axons, whereas others inhibit mainly Ib axons or sensory fibers from skin.

The principal mechanism responsible for sensory inhibition is a depolarization of the primary terminal caused by an inward Cl^- current when GABAergic receptors on the terminal are activated. This depolarization inactivates some of the Na^+ channels in the terminal, so the action potentials that reach the synapse are reduced in size. The effect of this is that release of neurotransmitter from the sensory afferent is diminished.

When tested by stimulation of peripheral afferents, presynaptic inhibition is widespread in the spinal cord and affects primary afferents from all muscles in a limb. However, similar to other interneurons, the neurons responsible for presynaptic inhibition are also controlled by descending pathways, making possible a much more focused modulation of presynaptic inhibition in relation to movement. Presynaptic inhibition at the synapse of Ia axons with motor neurons of the muscles that are activated as part of a movement is reduced at the onset of movement. In contrast, presynaptic inhibition of Ia axons on motor neurons connected to inactive muscles is increased. One example of this selective modulation is increased presynaptic inhibition of Ia axons at their synapse with antagonist motor neurons, which explains part of the reduction of stretch reflexes in antagonist muscles at the onset of agonist contraction. In this way, the nervous system takes advantage of the widespread connectivity of Ia axons, using presynaptic inhibition to shape activity in the Ia afferent network to facilitate activation of specific muscles.

Presynaptic inhibition provides a mechanism by which the nervous system can reduce sensory feedback predicted by the motor command, while allowing unexpected feedback access to the spinal motor circuit and the rest of the nervous system. In line with this function, presynaptic inhibition of Ia sensory axons from muscle spindles generally increases during

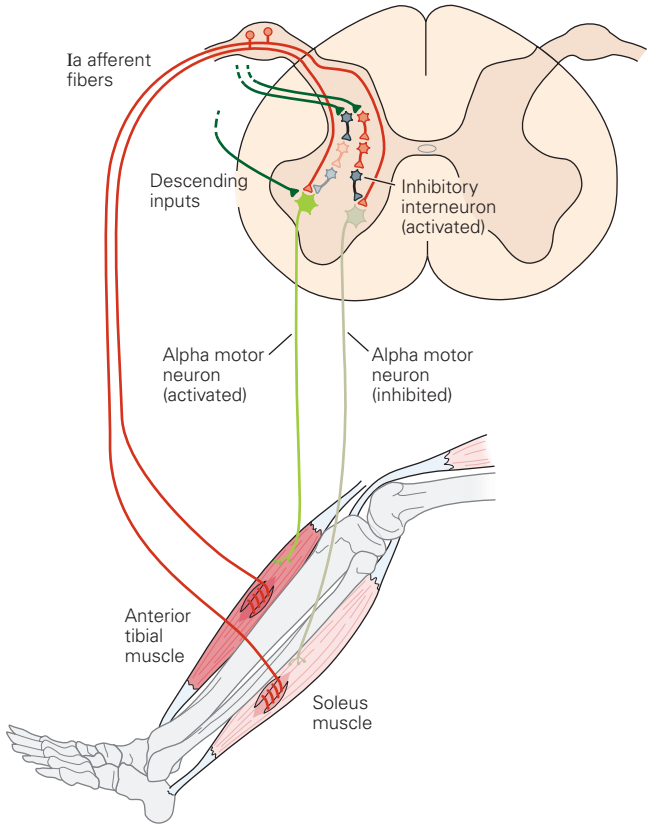

Figure 32–12 Selective modulation of primary sensory axon terminals by descending presynaptic inhibitory inputs contributes to generation of coordinated limb movements. Inhibitory interneurons (**blue**) activated by descending inputs can have either pre- or postsynaptic effects. Some interneurons releasing the inhibitory neurotransmitter γ-aminobutyric acid (GABA) form axo-axonic synapses with the primary sensory fibers. The principal inhibitory mechanism involves activation of GABAergic receptors on the terminals of the presynaptic Ia sensory axons, resulting in depolarization of the terminals and reduced transmitter release. Such presynaptic inhibition is widely distributed in the spinal cord. Stimulation of Ia sensory fibers from one flexor muscle may elicit presynaptic inhibition of both flexor and extensor Ia axon terminals on motor neurons innervating muscles throughout the limb. However, several different populations of interneurons mediating presynaptic inhibition exist, which allows a very specific regulation of presynaptic inhibition in relation to voluntary movements. Interaction of sensory inputs with descending motor commands in the corticospinal tract may thus *decrease* presynaptic inhibition of Ia axon terminals on agonist motor neurons (eg, anterior tibial motor neurons) and at the same time *increase* presynaptic inhibition of Ia terminals on antagonist motor neurons (eg, soleus motor neurons). Regulation of presynaptic inhibition may thus simultaneously facilitate the sensory feedback to the activation of agonist motor neurons and at the same time diminish the risk that stretch of the antagonist muscles will elicit a stretch reflex that would counteract the movement.

movements that are highly predictable, such as walking and running.

Finally, presynaptic inhibition may help stabilize the execution of movements by preventing excessive sensory feedback and associated self-reinforcing oscillatory activity.

Part of the Descending Command for Voluntary Movements Is Conveyed Through Spinal Interneurons

In cats as well as most other vertebrates, the corticospinal tract has no direct connections to spinal motor neurons; all the descending commands have to be channeled through spinal interneurons that are also part of reflex pathways. Humans and Old World monkeys are the only species in which corticospinal neurons make direct connections with the spinal motor neurons in the ventral horn of the spinal cord. Even in these species, a considerable fraction of the corticospinal tract fibers terminate in the intermediate nucleus on spinal interneurons, and the corticospinal fibers that terminate on motor neurons also have collaterals that synapse on interneurons. A considerable part of each descending command for movement in the corticospinal tract therefore has to be conveyed through spinal interneurons—and integrated with sensory activity—before reaching the motor neurons.

Propriospinal Neurons in the C3–C4 Segments Mediate Part of the Corticospinal Command for Movement of the Upper Limb

In the 1970s, Anders Lundberg and his collaborators demonstrated that a group of neurons in the C3–C4 spinal segments of the cat spinal cord send their axons to motor neurons located in more caudal cervical segments (Figure 32–13). Since the neurons in the C3–C4 segments project to motor neurons that innervate a range of forelimb muscles controlling different joints, and receive input from both skin and muscles throughout the forelimb, they are named *propriospinal neurons*. In addition to sensory input from skin and muscle afferents, the C3–C4 propriospinal neurons are activated by collaterals from the corticospinal tract and thereby relay disynaptic excitation from the motor cortex to the spinal motor neurons.

Subsequent experiments by Bror Alstermark in Sweden and Tadashi Isa in Japan have confirmed that similar propriospinal neurons also exist in the C3–C4 segments of the monkey spinal cord and are involved in mediating at least part of the motor command for reaching. Noninvasive experiments have also provided indirect evidence of the existence of C3–C4 propriospinal neurons in the human spinal cord. With the evolution of direct monosynaptic corticomotor connections in monkeys and humans, the corticospinal transmission through this disynaptic pathway may have become less important.

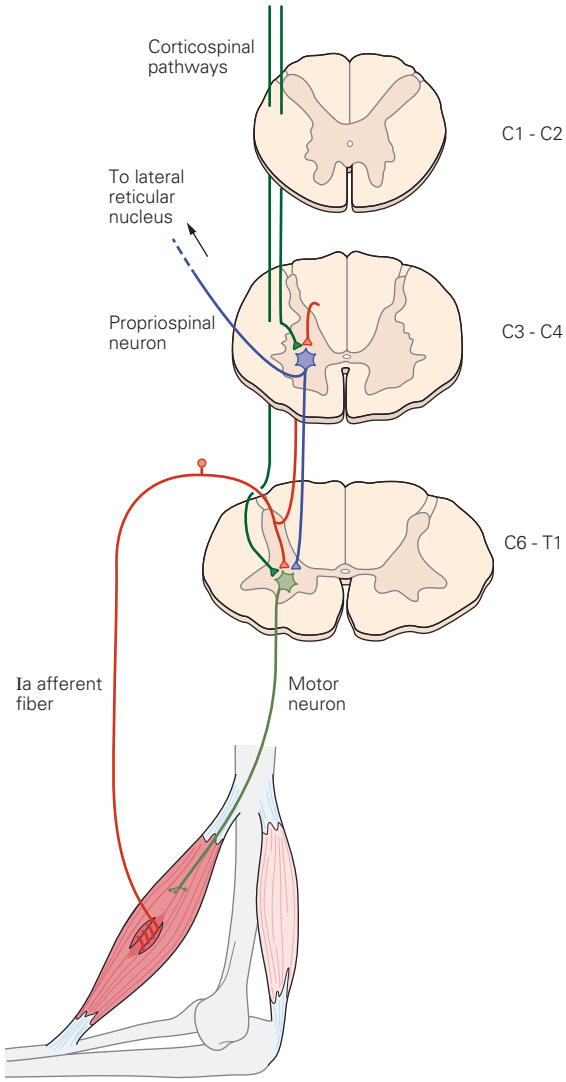

Figure 32–13 **Propriospinal neurons in spinal segments C3–C4 mediate part of the descending motor command to cervical motor neurons.** Some corticospinal fibers (**green**) send collaterals to propriospinal neurons in the C3–C4 segments (**blue**). These C3–C4 propriospinal neurons project to motor neurons located in more caudal cervical segments. They also receive excitatory input from muscle afferents and send collaterals to the lateral reticular nucleus.

Lumbar interneurons that receive input from groups I and II sensory axons from muscle also receive significant input from descending motor tracts and provide excitatory projections to spinal motor neurons. These interneurons thus convey part of the indirect motor command for voluntary movements to the spinal motor neurons that control leg muscles and may be a lumbar equivalent of the C3–C4 propriospinal neurons in the cervical spinal cord.

Neurons in Spinal Reflex Pathways Are Activated Prior to Movement

Synaptic transmission in spinal reflex pathways may change in response to the intention to move, independent of movement. Intracellular recordings from active monkeys have demonstrated that the intention to make a movement modifies activity in interneurons in the spinal cord and alters transmission in spinal reflex pathways. Similarly, in human subjects who have been prevented from contracting a muscle (by injection of lidocaine into the peripheral nerve supplying the muscle), the voluntary effort to contract the muscle still changes transmission in reflex pathways as if the movement had actually taken place.

In both humans and monkeys, spinal interneurons also change their activity well in advance of the actual movement. For example, in human subjects, Hoffmann reflexes elicited in a muscle that is about to be activated are facilitated fully 50 ms prior to the onset of contraction and remain facilitated throughout the movement. Conversely, reflexes in the antagonist muscles are suppressed. The suppression of stretch reflexes in the antagonist muscle prior to the onset of movement is an efficient way of preventing the antagonist from being reflexively activated when it is stretched at the onset of the agonist contraction.

Transmission in spinal reflex pathways can also be modified in connection with higher cognitive functions. Two examples are (1) an increase in the tendon jerk reflex in the soleus muscle of a human subject imagining pressing a foot pedal and (2) modulation of the Hoffmann reflex in arm and leg muscles while a subject observes grasping and walking movements, respectively.

Proprioceptive Reflexes Play an Important Role in Regulating Both Voluntary and Automatic Movements

All movements activate receptors in muscles, joints, and skin. Sensory signals generated by the body's own movements were termed *proprioceptive* by Sherrington, who proposed that they control important aspects of normal movements. A good example is the Hering-Breuer reflex, which regulates the amplitude of inspiration. Stretch receptors in the lungs are activated during inspiration, and the Hering-Breuer reflex eventually triggers the transition from inspiration to expiration when the lungs are expanded.

A similar situation exists in the walking systems of many animals; sensory signals generated near the end of the stance phase initiate the onset of the swing phase (Chapter 33). Proprioceptive signals can also contribute to the regulation of motor activity during voluntary movements, as shown in studies of individuals with sensory neuropathy of the arms. These patients display abnormal reaching movements and have difficulty in positioning the limb accurately because the lack of proprioception results in a failure to compensate for the complex inertial properties of the human arm.

Therefore, a primary function of proprioceptive reflexes in regulating voluntary movements is to adjust the motor output according to the changing biomechanical state of the body and limbs. This adjustment ensures a coordinated pattern of motor activity during an evolving movement and compensates for the intrinsic variability of motor output.

Spinal Reflex Pathways Undergo Long-Term Changes

Transmission in spinal reflex pathways is modulated not only to suit the immediate requirements of the movement but also to adapt the motor command to the motor experience of the individual. For example, transmission in the reciprocal Ia inhibitory pathway shows a gradual change when subjects improve their ability in coordinating agonist and antagonist contraction. Inactivity following long periods of bedrest or immobilization also results in changes in stretch reflexes and H-reflexes. Conversely, the soleus stretch reflex is low in highly trained ballet dancers and varies among different kinds of athletes.

Extensive studies of humans, monkeys, and rats by Jonathan Wolpaw and his colleagues have found that stretch reflexes can be operantly conditioned to either increase or decrease. The mechanisms underlying these changes are complex and involve alterations at multiple sites including changes in the properties of motor neurons. A general prerequisite for these changes is that corticospinal control of the spinal motor circuits must be intact.

Damage to the Central Nervous System Produces Characteristic Alterations in Reflex Responses

Stretch reflexes are routinely used in clinical examinations of patients with neurological disorders. They are typically elicited by sharply tapping the tendon of a muscle with a reflex hammer. Although the responses are often called tendon reflexes or tendon jerks, the receptor that is stimulated, the muscle spindle, actually lies in the muscle rather than the tendon. Only the primary sensory fibers in the spindle participate in the tendon reflex, for these are selectively activated by a rapid stretch of the muscle produced by the tendon tap.

Measuring alterations in the strength of the stretch reflex can assist in the diagnosis of certain conditions and in localizing injury or disease in the central nervous system. Absent or hypoactive stretch reflexes often indicate a disorder of one or more of the components of the peripheral reflex pathway: sensory or motor axons, the cell bodies of motor neurons, or the muscle itself (Chapter 57). Nevertheless, because the excitability of motor neurons is dependent on descending excitatory and inhibitory signals, absent or hypoactive stretch reflexes can also result from lesions of the central nervous system. Hyperactive stretch reflexes, conversely, always indicate that the lesion is in the central nervous system.

Interruption of Descending Pathways to the Spinal Cord Frequently Produces Spasticity

The force with which a muscle resists being lengthened depends on the muscle's intrinsic elasticity, or stiffness. Because a muscle has elastic elements in series and parallel that resist lengthening, it behaves like a spring (Chapter 31). In addition, connective tissue in and around the muscle may also contribute to its stiffness. These elastic elements may be pathologically altered following brain and spinal cord injury and thereby cause contractures and abnormal joint positions. However, there is also a neural contribution to the resistance of a muscle to stretch; the feedback loop inherent in the stretch reflex pathway acts to resist lengthening of the muscle.

Spasticity is characterized by hyperactive tendon jerks and an increase in resistance to rapid stretching of the muscle. Slow movement of a joint elicits only passive resistance, which is caused by the elastic properties of the joint, tendon, muscle, and connective tissues. As the speed of the stretch is increased, resistance to the stretch rises progressively. This phasic relation is what characterizes spasticity; an active reflex contraction occurs only during a rapid stretch, and when the muscle is held in a lengthened position, the reflex contraction subsides.

Spasticity is seen following lesion of descending motor pathways caused by stroke, injuries of the brain or spinal cord, and degenerative diseases such as multiple sclerosis. It is also seen in individuals with brain damage that occurs before, during, or shortly after birth, resulting in *cerebral palsy*.

Spasticity is not seen immediately following lesions of descending pathways, but develops over days, weeks, and even months. This parallels plastic changes at multiple sites in the stretch reflex circuitry. Sensory group Ia axons release more transmitter substance when active, and the alpha motor neurons change their intrinsic properties and their morphology (dendritic sprouting and denervation hypersensitivity) so that they become more excitable. Changes in excitatory and inhibitory interneurons that project to the motor neurons also take place and probably contribute to the increased excitability.

Whatever the precise mechanisms that produce spasticity, the effect is a strong facilitation of transmission in the monosynaptic reflex pathway. It is not the only reflex pathway affected by lesions of descending motor pathways. Pathways involving group I/II interneurons and sensory fibers from skin are also affected and exhibit the symptomatology observed in patients with central motor lesions. In the clinic, spasticity is therefore used in a broader sense and does not only relate to stretch reflex hyperexcitability. It is still debated whether reflex hyperexcitability contributes to the movement disorder following lesion of descending pathways or whether it may be a pertinent adaptation that helps to activate the muscles when descending input is diminished.

Lesion of the Spinal Cord in Humans Leads to a Period of Spinal Shock Followed by Hyperreflexia

Damage to the spinal cord can cause large changes in the strength of spinal reflexes. Each year, approximately 11,000 Americans sustain spinal cord injuries, and many more suffer from strokes. More than half of these injuries produce permanent disability, including impairment of motor and sensory functions and loss of voluntary bowel and bladder control. Approximately 250,000 people in the United States today have some permanent disability from spinal cord injury.

When the spinal cord is completely transected, there is usually a period immediately after the injury when all spinal reflexes below the level of the