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An Overall View

DURING PURPOSEFUL MOVEMENTS the central nervous system uses information from a vast array of sensory receptors to ensure that the pattern of muscle activity suits the purpose. Without this sensory information movements tend to be imprecise, and tasks requiring fine coordination in the hands, such as buttoning one's shirt, are impossible.

Charles Sherrington was among the first to recognize the importance of sensory information in regulating movements. In 1906 he proposed that simple reflexes—stereotyped movements elicited by activation of receptors in skin or muscle—are the basic units for movement. He further posited that complex sequences of movements can be produced by combining simple reflexes. This view guided motor physiology for much of the 20th century.

The view that reflexes are automatic, stereotyped movements in response to stimulation of peripheral receptors arose primarily from laboratory studies of reflexes in animals with central nervous system lesions. Once investigators began to measure reflexes in intact animals engaged in normal behavior, ideas about reflexes changed. We now know that reflexes are flexible, that under normal conditions they can be adapted to a task. The prevalent view today is that reflexes are integrated by centrally generated motor commands into complex adaptive movements.

In this chapter we consider the principles underlying the organization and function of reflexes, focusing on spinal reflexes. The sensory stimuli for spinal reflexes arise from receptors in muscles, joints, and skin, and the neural circuitry responsible for the motor response is entirely contained within the spinal cord.

Reflexes Are Adaptable to Particular Motor Tasks

A good example of the adaptability of reflexes is how certain reflexes change in response to stretching the wrist muscles. When a person is kneeling or standing the stretched muscles contract, but muscles in other limbs also contract to prevent a loss of balance. Interestingly, the reflex response of the elbow extensor of the *opposite* arm depends on how that arm is being used.

If the arm is used to stabilize the body by holding the edge of a table, a large excitatory response in the elbow extensor muscle resists the forward sway of the body. If the arm is instead holding an unsteady object such as a cup of tea, reflex inhibition of the elbow extensors prevents movement of the cup (Figure 35–1A).

Another example of adaptability is the reflex of finger and thumb flexor muscles in response to stretching thumb muscles. If a subject rhythmically taps the tips of the index finger and thumb to each other, and flexion of the thumb is resisted, a short-latency reflex response is produced in *both* the finger and thumb flexor muscles. As a result, the reflex in the finger flexor muscle produces a larger flexion movement of the finger to compensate for the reduced flexion of the thumb and ensure the performance of the intended task (Figure 35–1B). If the subject is simply making rhythmic thumb movements, a reflex response is produced only in the thumb flexor muscle.

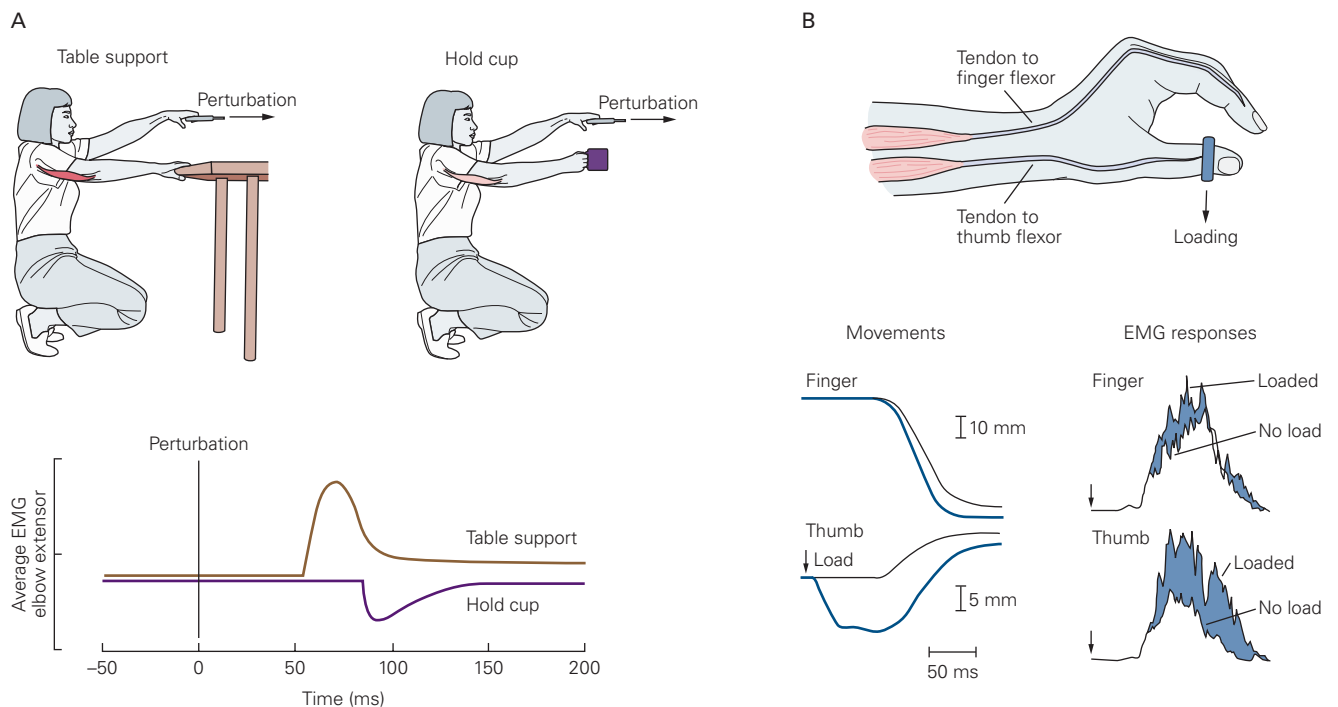


Figure 35–1 Reflex responses are often complex and can change depending on the task.

A. Perturbation of one arm causes an excitatory reflex response in the contralateral elbow extensor muscle when the contralateral limb is used to prevent the body from moving forward by grasping a table. The same stimulus produces an inhibitory response in the muscle when the contralateral hand holds a filled cup. (Adapted, with permission, from Marsden et al. 1981.)

B. Loading the thumb during a rhythmic sequence of finger-to-thumb pinching movements produces a reflex response in the finger muscle as well as the thumb muscle. The additional movement of the finger ensures that the pinching movement remains accurate. The blue area in the electromyogram (EMG) records indicates the reflex response. (Adapted, with permission, from Cole et al. 1984.)

A third example of adaptability is the conditioning of the flexion-withdrawal reflex. Flexion-withdrawal can be associated with an auditory tone by classical conditioning (see Chapter 65). A subject is asked to place the palmar surface of an index finger on an electrode. A mild electrical shock is then paired with an audible tone. As expected, after only a few pairings the tone alone will elicit the withdrawal reflex. What exactly has been conditioned? Is it the contraction of a fixed group of muscles or the behavioral *act* that withdraws the finger from the noxious stimulus?

This question can be answered by having the subject turn his or her hands over after conditioning is complete, so that now the dorsal surface of the finger is in contact with the electrode. Most subjects will withdraw their fingers from the electrode when the tone is played, even though this means that the opposite muscles now contract. Thus, the conditioned response is not merely a stereotyped set of muscle contractions but the elicitation of an appropriate behavior.

Three important principles are illustrated by these examples. First, neural signaling in reflex pathways is adjusted according to the motor task. The state of the reflex pathways for any task is referred to as the *functional set*. Exactly how a functional set is established for most motor tasks is largely unknown, and unraveling the underlying mechanisms is one of the challenging areas of contemporary research on motor systems. Second, sensory input from a localized source generally produces coordinated reflex responses in several muscles at once, some of which may be distant from the stimulus. Third, supraspinal centers play an important role in modulating and adapting spinal reflexes, even to the extent of reversing movements when appropriate.

To understand the neural basis for reflexes and how they are modified for a particular task, we must first have a thorough knowledge of how reflex pathways are organized in the spinal cord. Although under normal conditions descending central commands directly shape spinal reflexes, many qualitative features of spinal reflexes are maintained after complete transection of the spinal cord, a condition that isolates the spinal circuits from the brain.

Spinal Reflexes Produce Coordinated Patterns of Muscle Contraction

Cutaneous Reflexes Produce Complex Movements That Serve Protective and Postural Functions

A familiar example of a spinal reflex is the flexion-withdrawal reflex, in which a limb is quickly withdrawn

from a painful stimulus. Flexion-withdrawal is a protective reflex in which a discrete stimulus causes all the flexor muscles in that limb to contract coordinately. We know that this is a spinal reflex because it persists after complete transection of the spinal cord.

The sensory signal activates divergent polysynaptic reflex pathways. One excites motor neurons that innervate flexor muscles of the stimulated limb, whereas another inhibits motor neurons that innervate the limb's extensor muscles (Figure 35–2A). Excitation of one group of muscles and inhibition of their antagonists—those that act in the opposite direction—is what Sherrington called *reciprocal innervation*, a key principle of motor organization that is discussed later in this chapter.

The reflex can produce an opposite effect in the contralateral limb, that is, excitation of extensor motor neurons and inhibition of flexor motor neurons. This *crossed-extension reflex* serves to enhance postural support during withdrawal of a foot from a painful stimulus. Activation of the extensor muscles in the opposite leg counteracts the increased load caused by lifting the stimulated limb. Thus, flexion-withdrawal is a complete, albeit simple, motor act.

Although flexion reflexes are relatively stereotyped, both the spatial extent and the force of muscle contraction depend on stimulus intensity. Touching a stove that is slightly hot may produce moderately 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 a 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 (see Chapter 36). Nevertheless, in mammals the intrinsic spinal circuits that control walking share many of the interneurons that are involved in flexion reflexes.

The Stretch Reflex Resists the Lengthening of a Muscle

Perhaps the most important—certainly the most studied—spinal reflex is the *stretch reflex*, a lengthening contraction of a muscle. Stretch reflexes were originally thought to be an intrinsic property of muscles. But early in the last century Liddell and Sherrington showed that they could be abolished by cutting either the dorsal or the ventral root, thus establishing that

A Polysynaptic pathways (flexion reflex)

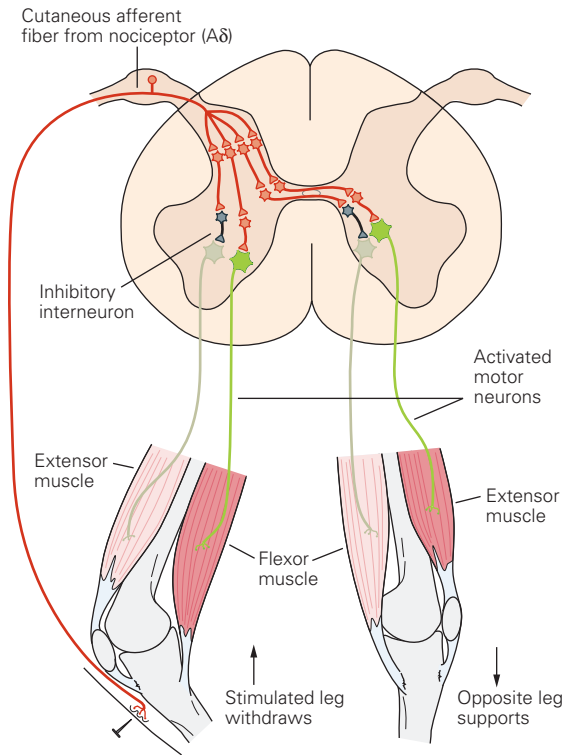
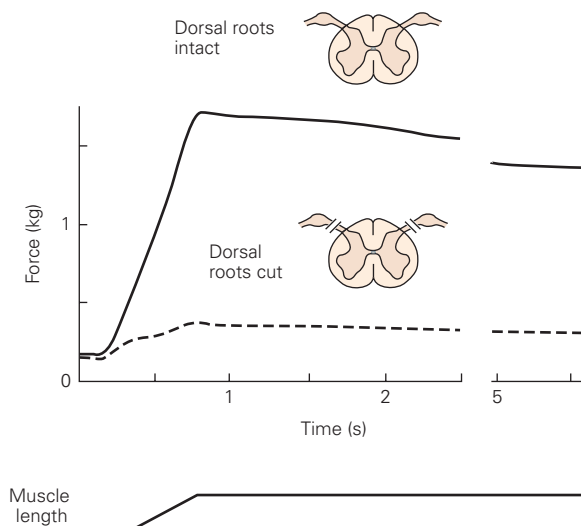
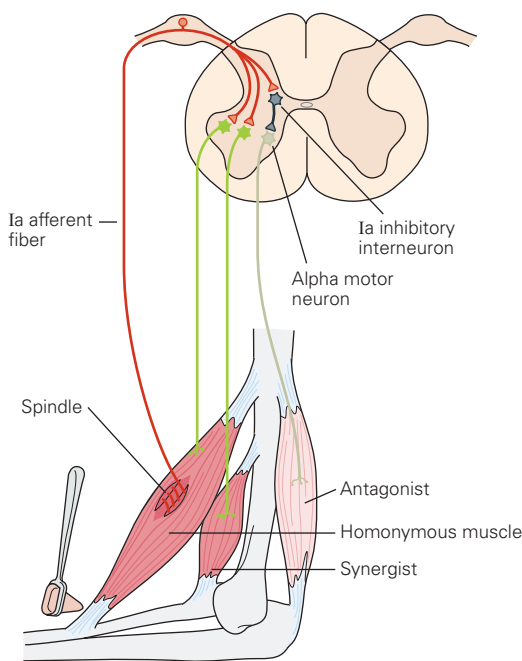


Figure 35-2 Spinal reflexes involve coordinated contractions of numerous muscles in the limbs.

A. Polysynaptic pathways in the spinal cord mediate flexion and crossed-extension reflexes. One excitatory pathway activates motor neurons that innervate ipsilateral flexor muscles, which withdraw the limb from noxious stimuli. Another pathway simultaneously excites motor neurons that innervate contralateral extensor muscles, providing support during withdrawal of the limb. Inhibitory interneurons ensure that the motor neurons supplying antagonist muscles are inactive during the reflex response. (Adapted, with permission, from Schmidt 1983.)

B. Monosynaptic pathways mediate stretch reflexes. Afferent axons from muscle spindles make excitatory connections on two sets of motor neurons: alpha motor neurons that innervate the same (homonymous) muscle from which they arise and motor neurons that innervate synergist muscles. They also act through interneurons to inhibit the motor neurons that innervate antagonist muscles. When a muscle is stretched by a tap with a reflex hammer, the firing rate in the afferent fiber from the spindle increases. This leads to contraction of the same muscle and its synergists and relaxation of the antagonist. The reflex therefore tends to counteract the stretch, enhancing the spring-like properties of the muscles. The records on the right demonstrate the reflex nature of contractions produced by muscle stretch in a decerebrate cat. When an extensor muscle is stretched it normally produces a large force, but it produces a very small force (**dashed line**) after the sensory afferents in the dorsal roots have been severed. (Adapted, with permission, from Liddell and Sherrington 1924.)

B Monosynaptic pathways (stretch reflex)



these reflexes require sensory input from muscle to spinal cord and a return path to muscle (Figure 35–2B).

We now know that the receptor that senses the change of length is the muscle spindle (Box 35–1) and that the type Ia axon from this receptor makes direct

excitatory connections with motor neurons. (The classification and nomenclature of sensory fibers from muscle are discussed in Box 35–2.) The afferent axon also connects to interneurons that inhibit the motor neurons that innervate antagonist muscles, another

Box 35–1 Muscle Spindles

Muscle spindles are small encapsulated sensory receptors that have a spindle-like or fusiform shape and are located within the fleshy part of a muscle. Their main function is to signal changes in the length of the muscle within which they reside. Changes in length of muscles are closely associated with changes in the angles of the joints that the muscles cross. Thus muscle spindles are used by the central nervous system to sense relative positions of the body segments.

Each spindle has three main components: (1) a group of specialized *intrafusal* muscle fibers with central regions that are noncontractile; (2) sensory fibers that terminate in the noncontractile central regions of the intrafusal fibers; and (3) motor axons that terminate in the polar contractile regions of the intrafusal fibers (Figure 35–3A).

When the intrafusal fibers are stretched, often referred to as “loading the spindle,” the sensory nerve endings are also stretched and increase their firing rate. Because muscle spindles are arranged in parallel with the *extrafusal* muscle fibers that make up the main body of the muscle, the intrafusal fibers change in length as the whole muscle changes. Thus, when a muscle is stretched, activity in the sensory endings of muscle spindles increases. When a muscle shortens, the spindle is unloaded and the activity decreases.

The intrafusal muscle fibers are innervated by *gamma* motor neurons, which have small-diameter myelinated axons, whereas the extrafusal muscle fibers are innervated by *alpha* motor neurons, with large-diameter myelinated axons. Activation of gamma motor neurons causes shortening of the polar regions of the intrafusal fibers. This in turn stretches the central region from both ends, leading to an increase in firing rate of the sensory endings or to a greater likelihood that the sensory endings will fire in response to stretch of the muscle. Thus the gamma motor neurons adjust the sensitivity of the muscle spindles. Contraction of the intrafusal muscle fibers does not contribute significantly to the force of muscle contraction.

The structure and functional behavior of muscle spindles is considerably more complex than this simple description implies. When a muscle is stretched the change in length has two phases: a dynamic phase, the

period during which length is changing, and a static or steady-state phase, when the muscle has stabilized at a new length. Structural specializations within each component of the muscle spindles allow spindle afferents to signal aspects of each phase separately.

There are two types of intrafusal muscle fibers: nuclear bag fibers and nuclear chain fibers. The bag fibers can be divided into two groups, dynamic and static. A typical spindle has two or three bag fibers and a variable number of chain fibers, usually about five. Furthermore, the intrafusal fibers receive two types of sensory endings. A single Ia (large diameter) axon spirals around the central region of all intrafusal muscle fibers and serves as the primary sensory ending (Figure 35–3B). A variable number of type II (medium diameter) axons, located adjacent to the central regions of the static bag and chain fibers, serve as secondary sensory endings.

The gamma motor neurons can also be divided into two classes: Dynamic gamma motor neurons innervate the dynamic bag fibers, whereas the static gamma motor neurons innervate the static bag fibers and the chain fibers.

This duality of structure is reflected in a duality of function. The tonic discharge of both primary and secondary sensory endings signals the steady-state length of the muscle. The primary sensory endings are, in addition, highly sensitive to the velocity of stretch, allowing them to provide information about the speed of movements. Because they are highly sensitive to small changes, the primary endings rapidly provide information about sudden unexpected changes in length, which can be used to generate quick corrective reactions.

Increases in the firing rate of dynamic gamma motor neurons increase the dynamic sensitivity of primary sensory endings but have no influence on secondary sensory endings. Increases in the firing rate of static gamma motor neurons increase the tonic level of activity in both primary and secondary sensory endings, decrease the dynamic sensitivity of primary endings (Figure 35–3C), and can prevent the silencing of primary endings when a muscle is released from stretch. Thus the central nervous system can independently adjust the dynamic and static sensitivity of the different sensory endings in muscle spindles.

instance of reciprocal innervation. This inhibition prevents muscle contractions that might otherwise resist the movements produced by the stretch reflexes.

Sherrington developed an experimental model for investigating spinal circuitry that is especially valuable in

the study of stretch reflexes. He conducted his experiments on cats whose brain stems had been surgically transected at the level of the midbrain, between the superior and inferior colliculi. This is referred to as a *decerebrate preparation*. The effect of this procedure is to

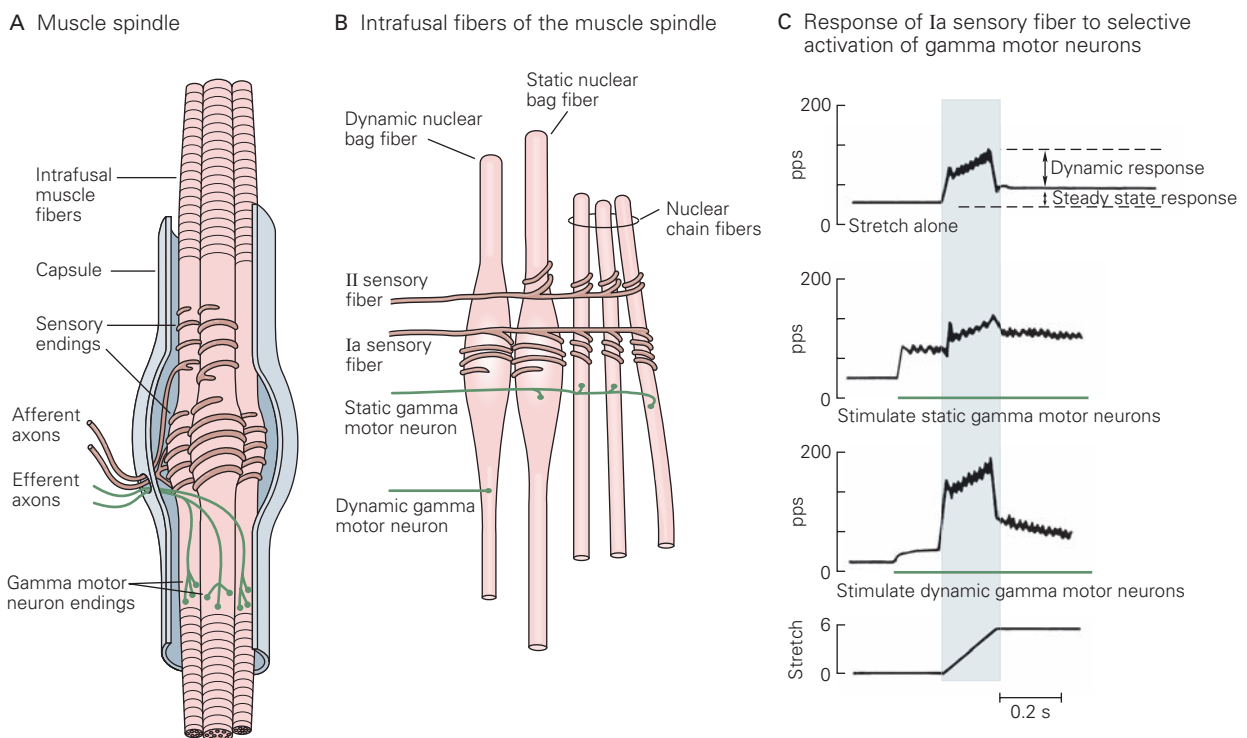


Figure 35-3 The muscle spindle detects changes in muscle length.

A. The main components of the muscle spindle are intrafusal muscle fibers, afferent sensory endings, and efferent motor endings. The intrafusal fibers are specialized muscle fibers with central regions that are not contractile. Gamma motor neurons innervate the contractile polar regions of the intrafusal fibers. Contraction of the polar regions pulls on the central regions of the intrafusal fiber from both ends. The sensory endings spiral around the central regions of the intrafusal fibers and are responsive to stretch of these fibers. (Adapted, with permission, from Hulliger 1984.)

B. The muscle spindle contains three types of intrafusal fibers: dynamic nuclear bag, static nuclear bag, and nuclear chain fibers. A single Ia sensory axon innervates all three types of fibers, forming a primary sensory ending. Type II sensory axons innervate the nuclear chain fibers

and static bag fibers, forming a secondary sensory ending. Two types of motor neurons innervate different intrafusal fibers. Dynamic gamma motor neurons innervate only dynamic bag fibers; static gamma motor neurons innervate various combinations of chain and static bag fibers. (Adapted, with permission, from Boyd 1980.)

C. Selective stimulation of the two types of gamma motor neurons has different effects on the firing of the Ia fibers from the spindle. Without gamma stimulation the Ia fiber shows a small dynamic response to muscle stretch and a modest increase in steady-state firing. When a static gamma motor neuron is stimulated, the steady-state response of the Ia fiber increases, but there is a decrease in the dynamic response. When a dynamic gamma motor neuron is stimulated, the dynamic response of the Ia fiber is markedly enhanced, but the steady-state response gradually returns to its original level. (Adapted, with permission, from Brown and Matthews 1966.)

Box 35–2 Classification of Sensory Fibers from Muscle

Sensory fibers are classified according to their diameter. Axons with larger diameters conduct action potentials more rapidly than do fibers of smaller diameters. Because each class of sensory receptors is innervated by fibers with diameters within a restricted range, this method of classification distinguishes to some extent the fibers that arise from the different types of receptor organs. The main groups of sensory fibers from muscle are listed in Table 35–1.

The organization of reflex pathways in the spinal cord has been established primarily by electrically stimulating the sensory fibers and recording evoked responses in different classes of neurons in the spinal cord. This method of activation has three advantages over natural stimulation. The timing of afferent input can be precisely established; the responses evoked in

motor neurons and other neurons by different classes of sensory fibers can be assessed by grading the strength of the electrical stimulus; and certain classes of receptors can be selectively activated.

The strength of the electrical stimulus required to activate a sensory fiber is measured relative to the strength required to activate the afferent fibers with the largest diameter because these fibers have the lowest threshold for electrical activation. The threshold of type I fibers is usually one to two times that of the largest afferents (with Ia fibers having, on average, a slightly lower threshold than Ib fibers). For most type II fibers the threshold is 2 to 5 times higher, whereas type III and IV have thresholds in the range of 10 to 50 times that of the largest afferents.

Table 35–1 Classification of Sensory Fibers from Muscle

Type	Axon	Receptor	Sensitive to
Ia	12–20 μm myelinated	Primary spindle ending	Muscle length and rate of change of length
Ib	12–20 μm myelinated	Golgi tendon organ	Muscle tension
II	6–12 μm myelinated	Secondary spindle ending	Muscle length (little rate sensitivity)
II	6–12 μm myelinated	Nonspindle endings	Deep pressure
III	2–6 μm myelinated	Free nerve endings	Pain, chemical stimuli, and temperature (important for physiological responses to exercise)
IV	0.5–2 μm nonmyelinated	Free nerve endings	Pain, chemical stimuli, and temperature

disconnect the rest of the brain from the spinal cord, thus blocking sensations of pain as well as interrupting normal modulation of reflexes by higher brain centers. A decerebrate animal has stereotyped and usually heightened stretch reflexes, making it is easier to examine the factors controlling their expression.

Without control by higher brain centers, descending pathways from the brain stem powerfully facilitate the neuronal circuits involved in the stretch reflexes of extensor muscles. This results in a dramatic increase in extensor muscle tone that sometimes suffices to support the animal in a standing position. In normal animals, owing to the balance between facilitation and inhibition,

stretch reflexes are weaker and considerably more variable in strength than those in decerebrate animals.

Local Spinal Circuits Contribute to the Coordination of Reflex Responses

The Stretch Reflex Involves a Monosynaptic Pathway

The neural circuit responsible for the stretch reflex was one of the first reflex pathways to be examined in detail. The physiological basis of this reflex was examined by

measuring the latency of the response in ventral roots to electrical stimulation of dorsal roots. When the Ia sensory axons innervating the muscle spindles were selectively activated, the reflex latency through the spinal cord was less than 1 ms. This demonstrated that the Ia fibers make direct connections on the alpha motor neurons, for the delay at a single synapse is typically 0.5 ms to 0.9 ms (Figure 35–4B).

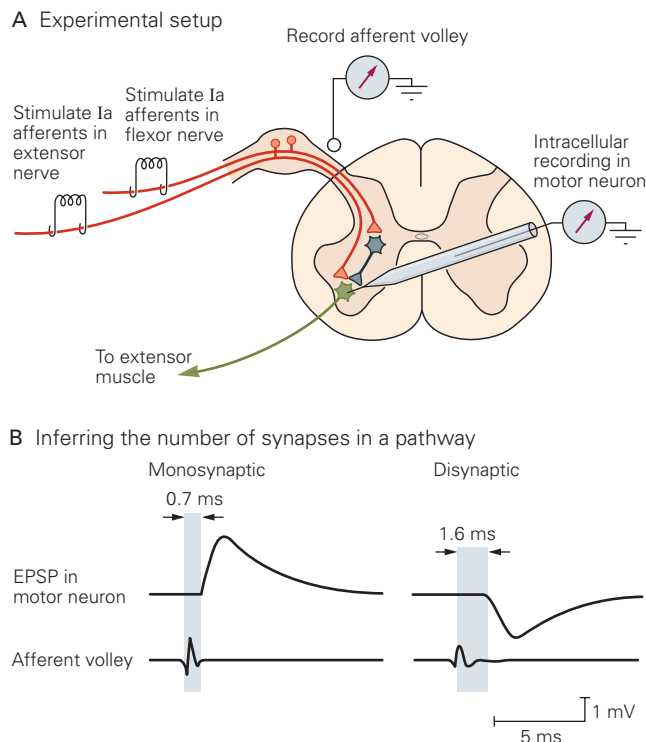


Figure 35–4 The number of synapses in a reflex pathway can be inferred from intracellular recordings.

A. An intracellular recording electrode is inserted into the cell body of a spinal motor neuron that innervates an extensor muscle. Stimulation of Ia sensory fibers from flexor or extensor muscles produces a volley of action potentials at the dorsal root.

B. Left: When Ia fibers from an extensor muscle are stimulated, the latency between the recording of the afferent volley and the excitatory postsynaptic potential in the motor neuron is only 0.7 ms. Because this is approximately equal to the duration of signal transmission across a single synapse, it can be inferred that the excitatory action of the stretch reflex pathway is monosynaptic. **Right:** When Ia fibers from an antagonist flexor muscle are stimulated, the latency between the recording of the afferent volley and the inhibitory postsynaptic potential in the motor neuron is 1.6 ms. Because this is approximately twice the duration of signal transmission across a single synapse, it can be inferred that the inhibitory action of the stretch reflex pathway is disynaptic.

The pattern of connections of Ia fibers to motor neurons can be shown directly by intracellular recording. Ia fibers from a muscle excite not only the motor neurons innervating the same (*homonymous*) muscle, but also those innervating other (*heteronymous*) muscles with a similar mechanical action. The Ia fibers also form inhibitory connections with the alpha motor neurons innervating antagonistic muscles through the *Ia inhibitory interneurons*. This disynaptic inhibitory pathway is the basis for reciprocal innervation: When a muscle is stretched, its antagonists relax.

Ia Inhibitory Interneurons Coordinate the Muscles Surrounding a Joint

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 involved in the stretch reflex are also used to coordinate muscle contraction during voluntary movements. These interneurons receive inputs from collaterals of axons descending from neurons in the motor cortex that make direct excitatory connections with spinal motor neurons. This organizational feature simplifies the control of voluntary movements, for higher centers do not have to send separate commands to the opposing muscles.

Sometimes it is advantageous to contract 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 35–5A). By changing the balance of excitatory and inhibitory inputs onto these interneurons, supraspinal centers can reduce 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, Renshaw cells make inhibitory synaptic connections with several populations of motor neurons, including the motor neurons that excite them and the Ia inhibitory interneurons (Figure 35–5B). The connections with motor neurons form a negative

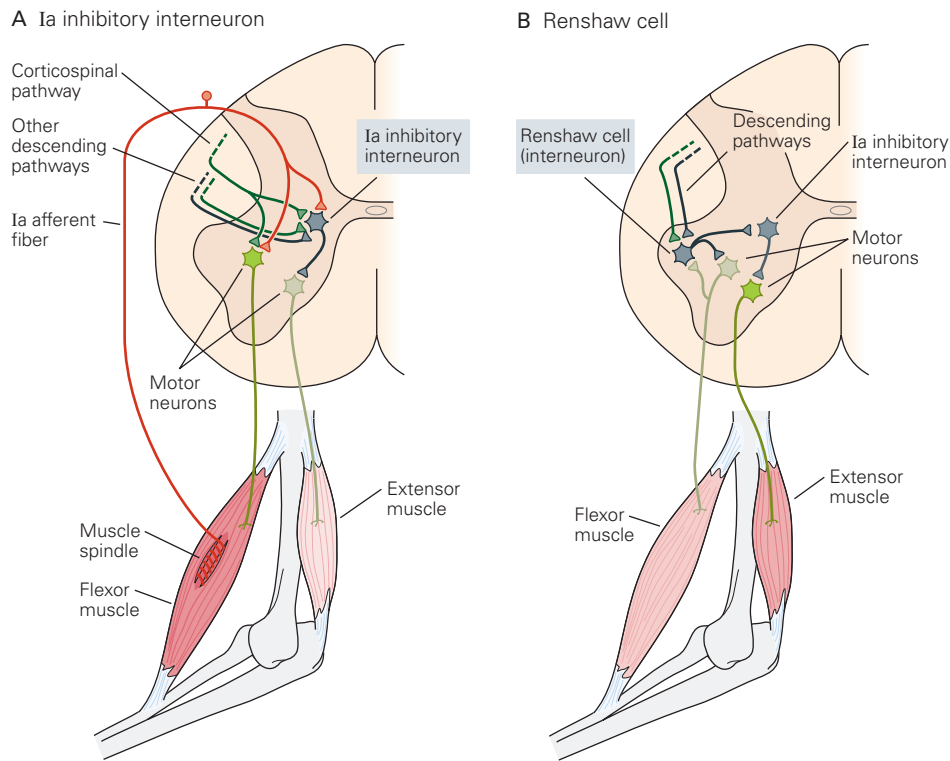


Figure 35-5 Inhibitory spinal interneurons coordinate reflex actions.

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. Renshaw cells are spinal interneurons that produce 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 around a joint.

feedback system that may help stabilize the firing rate of the motor neurons, whereas the connections with the Ia inhibitory interneurons may regulate the strength of inhibition of antagonistic motor neurons. In addition, Renshaw cells also receive significant synaptic input from descending pathways and distribute inhibition to task-related groups of motor neurons and Ia interneurons. It is therefore likely that Renshaw cells help establish the pattern of signaling in divergent Ia sensory pathways according to the motor task.

Divergence in Reflex Pathways Amplifies Sensory Inputs and Coordinates Muscle Contractions

In all reflex pathways in the spinal cord the sensory neurons form divergent connections with a large number of target neurons through extensive axonal

branching. The flexion-withdrawal reflex, for example, involves extensive divergence within the spinal cord. Stimulation of a small number of sensory axons from a small area of skin is sufficient to cause contractions of widely distributed muscles and thus to produce a coordinated motor pattern.

Lorne Mendell and Elwood Henneman used a computer enhancement technique called *spike-triggered averaging* to determine the extent to which the action potentials in single Ia fibers are transmitted to a population of spinal motor neurons. They found that individual Ia axons make excitatory synapses with all homonymous motor neurons innervating the medial gastrocnemius of the cat. This widespread divergence effectively amplifies the signals of individual Ia fibers, leading to a strong excitatory drive to the muscle from which they originate (*autogenic excitation*).

The Ia axons in reflex pathways also provide excitatory inputs to many of the motor neurons innervating synergist muscles (up to 60% of the motor neurons of some synergists). Although widespread, these connections are not as strong as the connections to homonymous motor neurons. The strength of these connections varies from muscle to muscle in a complex way according to the similarity of the mechanical actions of the synergists. We have already noted that, in the control of voluntary movements, descending pathways make use of reciprocal inhibition of antagonists in the stretch reflex. A similar convergence principle holds for the activation of motor neurons innervating synergist muscles. Thus stretch reflex pathways provide a principal mechanism by which the contractions of different muscles can be linked in voluntary as well as reflex actions.

Convergence of Inputs on Ib Interneurons Increases the Flexibility of Reflex Responses

Thus far we have considered reflex pathways as though each included only one type of sensory fiber. But an enormous diversity of sensory information converges on interneurons in the spinal cord.

The *Ib inhibitory interneuron* is one of the best-studied interneurons that receive extensive convergent input. Its principal input is from Golgi tendon organs, sensory receptors that signal the tension in a muscle (Box 35–3), and it makes inhibitory connections with homonymous motor neurons. As one might expect from this connectivity, stimulation of tendon organs or their Ib afferent fibers in passive animals produces disynaptic inhibition of homonymous motor neurons (*autogenic inhibition*). However, stimulation of Ib afferents in active animals does not always inhibit homonymous motor neurons. Indeed, we shall see in the next section that stimulation of tendon organs may in certain conditions excite homonymous motor neurons.

One reason that the reflex actions of the sensory axons from tendon organs are complex in natural situations is that the Ib inhibitory interneurons also receive input from the muscle spindles, cutaneous receptors, and joints (Figure 35–7A). In addition, they receive both excitatory and inhibitory input from various descending pathways.

Golgi tendon organs were first thought to have a protective function, preventing damage to muscle, for it was assumed that they always inhibited homonymous motor neurons and that they fired only when tension in the muscle was high. But we now know that these receptors signal minute changes in muscle tension, thus

providing the nervous system with precise information about the state of a muscle's contraction.

The convergence of sensory input from tendon organs, cutaneous receptors, and joint receptors onto interneurons that inhibit motor neurons may allow for precise spinal control of muscle force in activities such as grasping a delicate object. Additional input from cutaneous receptors may facilitate activity in the Ib inhibitory interneurons when the hand reaches an object, thus reducing the level of muscle contraction and permitting a soft grasp.

Finally, like the Ia fibers from muscle spindles, the Ib fibers from tendon organs form widespread connections with motor neurons that innervate muscles acting at different joints. Therefore the connections of the afferent fibers from tendon organs with the Ib inhibitory interneurons are part of spinal reflex networks that regulate movements of whole limbs.

Central Motor Commands and Cognitive Processes Can Alter Synaptic Transmission in Spinal Reflex Pathways

Both the strength and the sign of synaptic transmission in spinal reflex pathways can be altered during behavioral acts. For example, in humans the strength of the monosynaptic reflex declines as we progress from standing to walking to running. It does so because stiffness increases naturally as muscle force increases, and thus reflexes are not needed.

Another example of a change in sign occurs in the activity of Ib sensory axons during walking. As we have seen, in passive animals the Ib 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 35–7B). This phenomenon is called *state-dependent reflex reversal*.

Transmission in spinal reflex pathways can also be modified in association with higher cognitive functions. Examples are increases in the tendon jerk reflex in the soleus muscle of humans while imagining pressing a foot pedal, and modulation of the Hoffmann reflex in arm and leg muscles when subjects observe grasping and walking movements, respectively. The latter findings indicate that the mirror-neuron system identified in cortical networks (see Chapter 38) influences neuronal systems in the spinal cord. Furthermore, intracellular recordings from monkeys engaged in normal behavior have demonstrated that the intention

Box 35-3 Golgi Tendon Organs

Golgi tendon organs are slender encapsulated structures approximately 1 mm long and 0.1 mm in diameter located at the junction between skeletal muscle fibers and tendon. Each capsule encloses several braided collagen fibers connected in series to a group of muscle fibers.

Each tendon organ is innervated by a single Ib axon that branches into many fine endings inside the capsule; these endings become intertwined with the collagen fascicles (Figure 35-6A).

Stretching of the tendon organ straightens the collagen fibers, thus compressing the Ib nerve endings and causing them to fire. Because the nerve endings are so closely associated with the collagen fibers, even very small stretches of the tendons can compress the nerve endings.

Whereas muscle spindles are most sensitive to changes in length of a muscle, tendon organs are most

sensitive to changes in muscle tension. Contraction of the muscle fibers connected to the collagen fiber bundle containing the receptor is a particularly potent stimulus to a tendon organ. The tendon organs are thus readily activated during normal movements. This has been demonstrated by recordings from single Ib axons in humans making voluntary finger movements and in cats walking normally.

Studies in anesthetized animal preparations have shown that the average level of activity in the population of tendon organs in a muscle is a good index of the total force in a contracting muscle (Figure 35-6B). This close agreement between firing frequency and force is consistent with the view that the tendon organs continuously measure the force in a contracting muscle.

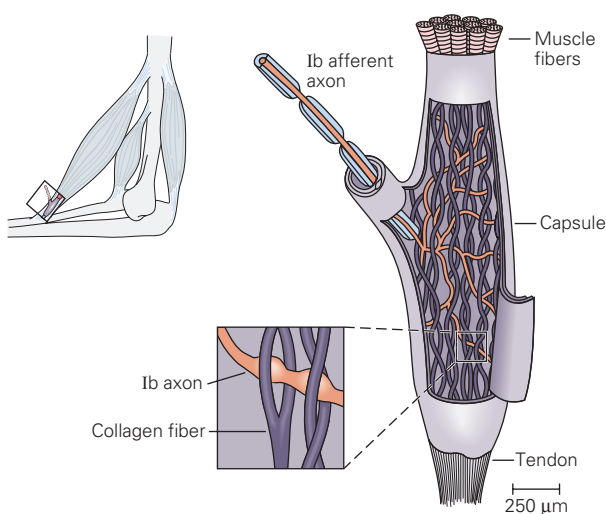


Figure 35-6A When the Golgi tendon organ is stretched (usually because of contraction of the muscle), the Ib afferent axon is compressed by collagen fibers (see inset) and its rate of firing increases. (Adapted, with permission, from Schmidt 1983; inset adapted, with permission, from Swett and Scholtz 1975.)

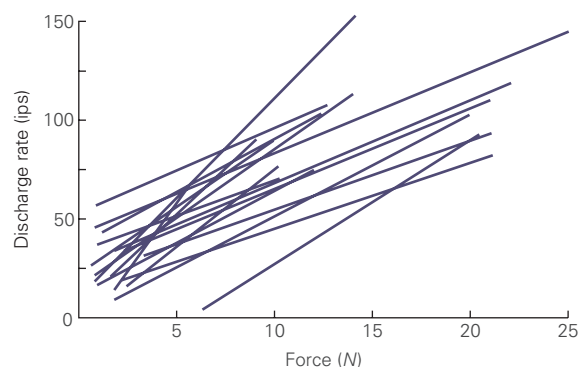


Figure 35-6B The discharge rate of a population of Golgi tendon organs signals the force in a muscle. Linear regression lines show the relationship between discharge rate and force for Golgi tendon organs of the soleus muscle of the cat. (Adapted, with permission, from Crago et al. 1982.)

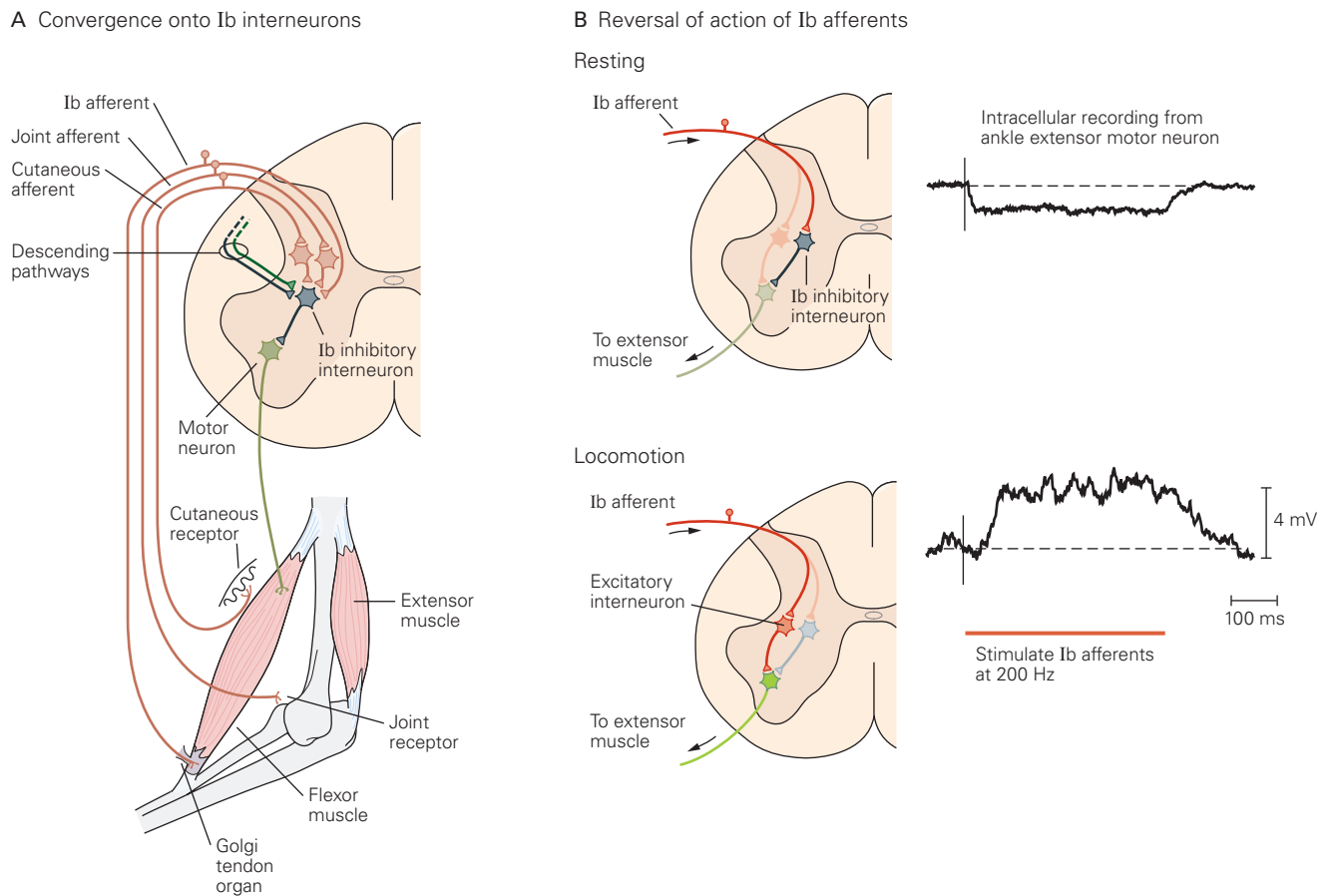


Figure 35-7 The reflex actions of Ib afferent fibers from Golgi tendon organs.

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.

to make a movement modifies activity in interneurons in the spinal cord and alters the transmission in spinal reflex pathways.

Central Neurons Can Regulate the Strength of Spinal Reflexes at Three Sites in the Reflex Pathway

As noted earlier, the force of a reflex can vary, although the sensory stimulus remains constant. This variability in reflex strength is possible because synaptic transmission in spinal reflex pathways can be modified at three possible sites: alpha motor neurons, interneurons

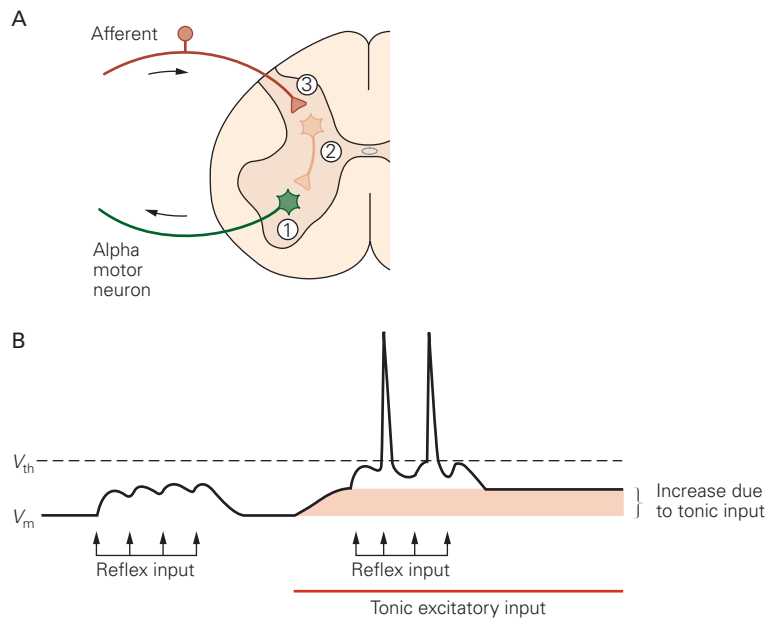
in all reflex circuits except monosynaptic pathways with Ia afferent fibers, and the presynaptic terminals of the afferent fibers (Figure 35-8A).

All three sites receive inputs from neurons in motor centers in the brain stem and cerebral cortex as well as other regions of the spinal cord. Signals from these higher-level neurons regulate the strength of reflexes by changing the background (tonic) level of activity at any of the three sites in the spinal reflex pathway. For example, an increase in tonic excitatory input to the alpha motor neurons moves the membrane potential of these cells closer to threshold so that

Figure 35–8 The strength of a spinal reflex can be modulated by changes in synaptic transmission in the reflex pathway.

A. A reflex pathway can be modified at three sites: alpha motor neurons (1), interneurons in polysynaptic pathways (2), and afferent axon terminals (3). Transmitter release from the primary afferent fibers is regulated by presynaptic inhibition (see Figure 12–16).

B. An increase in tonic (background) excitatory input to a motor neuron depolarizes the neuron to a level that enables an otherwise ineffective reflex input (left) to initiate action potentials. The reflex input is represented by a series of excitatory postsynaptic potentials. (V_{th} , threshold voltage; V_m , membrane potential.)



even the slightest reflex input will more easily activate the motor neurons (Figure 35–8B). Another mechanism for modulating the strength of reflexes is to change the physiological properties of motor neurons and perhaps interneurons. Activity in descending monoaminergic systems can alter the properties of motor neurons so they either discharge at a much higher rate in response to the same synaptic input or remain active following a brief excitatory input (see Chapter 34).

Reflex strength can be changed quickly to adapt to the requirements of specific tasks. Intracellular recordings suggest that presynaptic inhibition of the Ia fibers from muscle spindles is particularly important for producing these changes. For example, during locomotion the level of presynaptic inhibition is rhythmically modulated; this action presumably modulates the strength of reflexes during the different phases of the gait cycle (see Chapter 36).

Gamma Motor Neurons Adjust the Sensitivity of Muscle Spindles

Activity of muscle spindles may be modulated by changing the level of activity in the gamma motor neurons, which innervate the intrafusal muscle fibers of muscle spindles (see Box 35–1). This function of gamma motor neurons, often referred to as the fusimotor system, can be demonstrated by selectively stimulating the alpha and gamma motor neurons under experimental conditions.

When only alpha motor neurons are stimulated, the firing of the Ia fiber from the muscle spindle pauses during contraction of the muscle because the muscle is shortening and therefore unloading (slackening) the spindle. However, if gamma motor neurons are activated at the same time as alpha motor neurons, the pause is eliminated. The contraction of the intrafusal fibers by the gamma motor neurons keeps the spindle under tension, thus maintaining the firing rate of the Ia fibers within an optimal range for signaling changes in length, whatever the actual length of the muscle (Figure 35–9). This *alpha-gamma co-activation* thus stabilizes the sensitivity of the muscle spindles and is used in many voluntary movements.

In addition to the axons of gamma motor neurons, axon collaterals of alpha motor neurons sometimes innervate the intrafusal fibers. Axons that innervate both intrafusal and extrafusal muscle fibers are referred to as *beta* axons. Beta axon collaterals provide the equivalent of alpha-gamma coactivation. Beta innervation in spindles exists in both cats and humans, although it is unquantified for most muscles.

The beta fusimotor system's forced linkage of extrafusal and intrafusal contraction highlights the importance of the independent fusimotor system (the gamma motor neurons). Indeed, in lower vertebrates, such as amphibians, beta efferents are the only source of intrafusal innervation. Mammals have evolved a mechanism that frees muscle spindles from complete dependence on the behavior of their parent muscles. In principle

this uncoupling allows greater flexibility in controlling spindle sensitivity for different types of motor tasks.

This conclusion is supported by recordings in spindle sensory afferents during a variety of natural movements in cats. The amount and type of activity in gamma motor neurons are set at steady levels, which vary according to the specific task or context. In general, activity levels in both static and dynamic gamma

motor neurons (see Figure 35–3B) are set at progressively higher levels as the speed and difficulty of the movement increase. Unpredictable conditions, such as when the cat is picked up or handled, lead to marked increases in activity in dynamic gamma motor neurons and thus increased spindle responsiveness when muscles are stretched. When an animal is performing a difficult task, such as walking across a narrow beam,

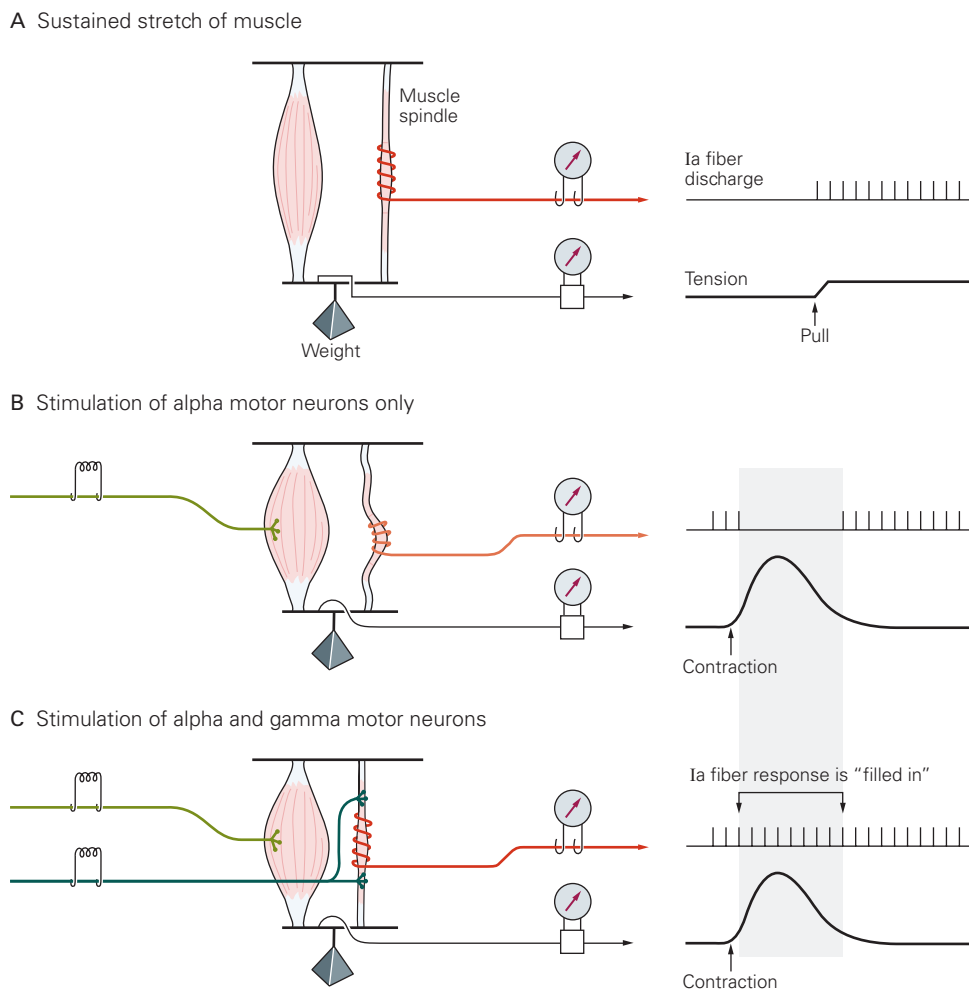


Figure 35–9 Activation of gamma motor neurons during active muscle contraction maintains muscle spindle sensitivity to muscle length. (Adapted, with permission, from Hunt and Kuffler 1951.)

A. Sustained tension elicits steady firing in the Ia sensory fiber from the muscle spindle (the two muscle fibers are shown separately for illustration only).

B. A characteristic pause occurs in the discharge of the Ia fiber when the alpha motor neuron is stimulated, causing a brief contraction of the muscle. The Ia fiber stops firing because the spindle is unloaded by the contraction.

C. Gamma motor neurons innervate the contractile polar regions of the intrafusal fibers of muscle spindles (see Figure 35–3A). If a gamma motor neuron is stimulated at the same time as the alpha motor neuron, the spindle is not unloaded during the contraction. As a result, the pause in discharge of the Ia sensory fiber that occurs when only the alpha motor neuron is stimulated is “filled in” by the response of the fiber to stimulation of the gamma motor neuron.

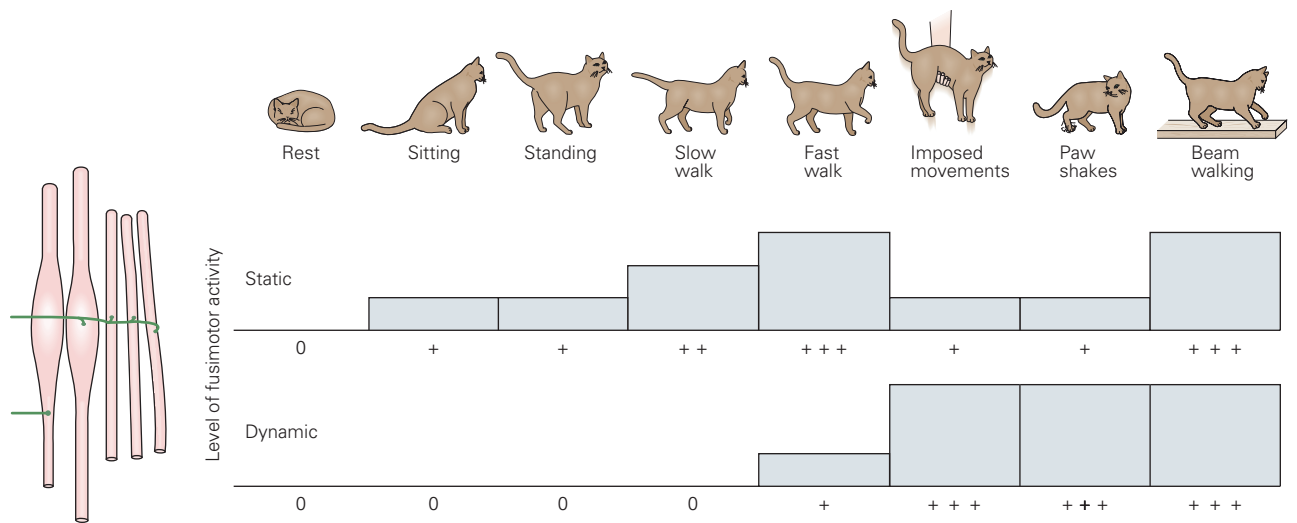


Figure 35-10 The level of activity in the fusimotor system varies with the type of behavior. Only static gamma motor neurons are active during activities in which muscle length changes slowly and predictably. Dynamic gamma motor

neurons are activated during behaviors in which muscle length may change rapidly and unpredictably. (Adapted, with permission, from Prochazka et al. 1988.)

both static and dynamic gamma activation are at high levels (Figure 35-10).

Thus the nervous system uses the fusimotor system to fine-tune muscle spindles so that the ensemble output of the spindles provides information most appropriate for a task. The task conditions under which independent control of alpha and gamma motor neurons occurs in humans have not yet been clearly established.

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 (see Chapter 36). Proprioceptive signals can also

contribute to the regulation of motor activity during voluntary movements, as has been shown in recent 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.

Reflexes Involving Limb Muscles Are Mediated Through Spinal and Supraspinal Pathways

Reflexes involving the limbs are mediated by multiple pathways acting in parallel through spinal and supraspinal pathways. Consider the response evoked by a sudden stretch of a flexor muscle of the thumb. This response has two discrete components. The first, the M1 response, is generated by the monosynaptic connection of muscle spindle afferents to the spinal motor neurons. The second, the M2 response, is also a reflex because its latency is shorter than the voluntary reaction time (Figure 35-11A).

The M2 response has been observed in virtually all limb muscles. In the distal muscles the M2 responses are mediated by pathways that include the motor cortex, as shown in studies of patients with Klippel-Feil syndrome. In this unusual condition axons descending from neurons in the motor cortex bifurcate and make connections to homologous motor neurons on

both sides of the body. Thus, when the individual voluntarily moves the fingers of one hand, these movements are mirrored by movements of the fingers of the other (Figure 35–11B). Similarly, when the M2 component is evoked by stretching muscles of one hand, a response with the same latency is evoked in the corresponding muscle of the other hand, even though no

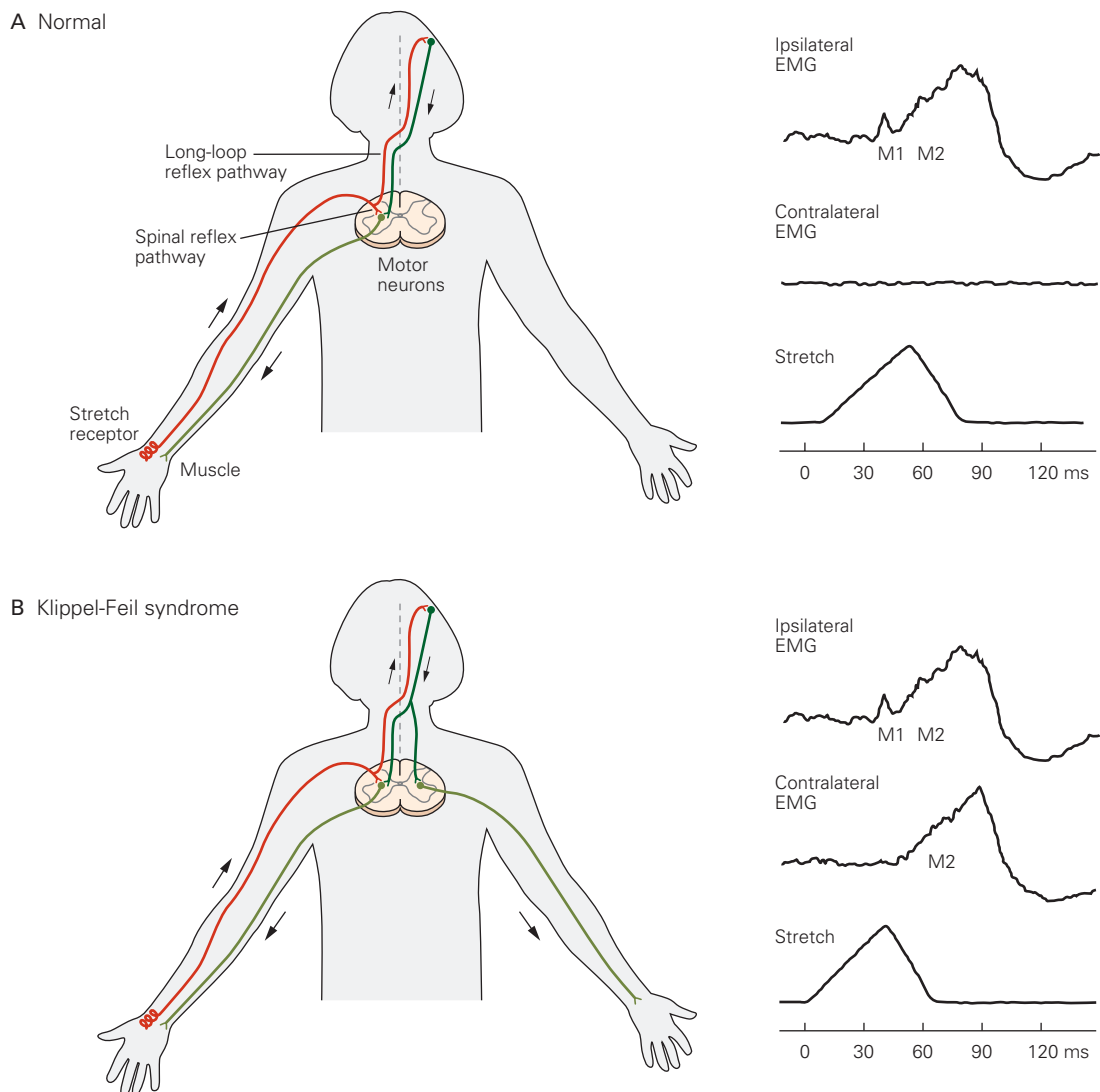


Figure 35–11 Reflexes of the limbs are mediated by spinal reflex pathways and long-loop pathways that involve the motor cortex. (Adapted, with permission, from Matthews 1991.)

A. In normal individuals a brief stretch of a thumb muscle produces a response that has two components. A short-latency response (M1) in the stretched muscle is controlled by the spinal reflex pathway, the monosynaptic connection between

muscle spindle afferents and spinal motor neurons. This is followed by a long-latency response (M2) controlled by a pathway that loops through the motor cortex.

B. In individuals with Klippel-Feil syndrome the M2 response is evoked bilaterally because neurons in the motor cortex activate motor neurons bilaterally.

M1 response has occurred in the other hand. The reflex pathway responsible for the M2 response must therefore traverse the motor cortex (Figure 35–11B).

Reflex responses mediated through the motor cortex and other supraspinal structures, the *long-loop reflexes*, have been investigated in numerous muscles in humans and other animals. The general conclusion is that long-loop reflexes via the cortex are of primary importance in regulating contractions in distal muscles, whereas subcortical reflex pathways are largely responsible for regulating the contractions of proximal muscles.

This type of organization is related to functional demands. Many tasks involving distal muscles require precise regulation by voluntary commands. By transmitting sensory signals to regions of cortex most involved in controlling voluntary movements, motor commands can be quickly adapted to the evolving needs of a task. More automatic motor functions, such as maintaining balance and producing gross bodily movements, can be efficiently executed largely through subcortical and spinal pathways.

Pioneering studies by Edward Evarts in the 1960s revealed that activity in approximately 50% of neurons in the motor cortex is modified in response to loading muscles during the voluntary maintenance of wrist position and during simple movements at the wrist. This early finding was initially interpreted as evidence that long-loop cortical reflexes are involved in compensating for change in loading conditions by functioning as online negative feedback controllers. However, subsequent studies on multijoint movements revealed complex response patterns in cortical neurons, many of which are not consistent with this concept. Moreover, the long delays in transmission in long-loop reflexes make these reflexes inappropriate for a direct role in load compensation because the potential delays would create instabilities.

Currently the function of proprioceptive input to the motor cortex in the volitional control of movement remains a puzzle. One contemporary notion is that proprioceptive input is integrated into internal models for estimating the state of the system (see Chapter 33). Another hypothesis is that proprioceptive inputs play a key role in motor cortical circuits that function as optimal feedback controllers, which use only those sensory signals that are required for attaining a specific goal.

Stretch Reflexes Reinforce Central Commands for Movements

Stretch-reflex pathways can contribute to regulation of motor neurons during voluntary movements and 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.

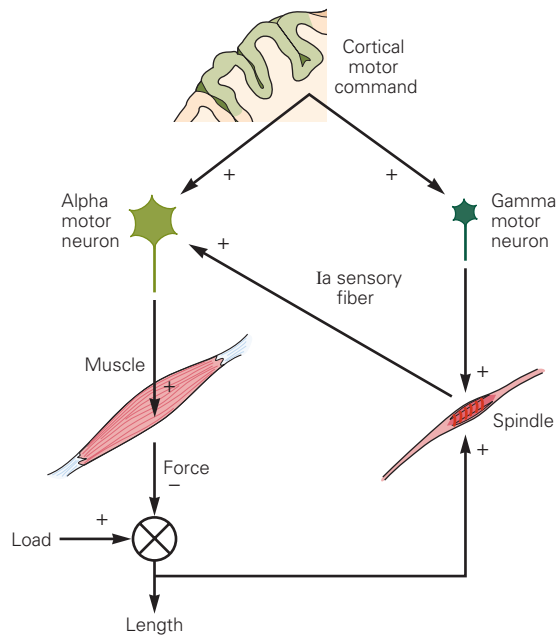
The stretch reflex loop thus acts continuously—the output of the system, a change in muscle length, becomes the input—tending to keep the muscle close to a desired or reference length. The stretch reflex is a negative feedback system, or *servomechanism*, because it tends to counteract or reduce deviations 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 that 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 35–12A).

In theory this mechanism could permit the nervous system to produce a movement 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. Thus, rather than providing compensation for large unexpected loads, monosynaptic and long-loop stretch reflex pathways may compensate for small changes in load and intrinsic irregularities in the muscle contraction, with the relative contribution of each pathway dependent on the muscle and the task.

Strong evidence that alpha and gamma motor neurons are co-activated during voluntary human movement has come from direct measurements of the activity of muscle spindles. In the late 1960s Åke Vallbo and Karl-Erik Hagbarth developed microneurography, a technique for recording from the largest afferent fibers

A Alpha-gamma co-activation reinforces alpha motor activity



B Spindle activity increases during muscle shortening

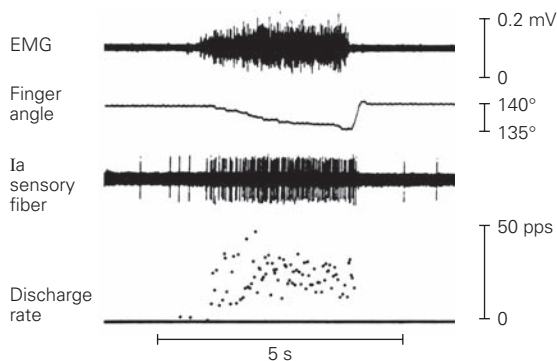


Figure 35–12 Alpha and gamma motor neurons are co-activated during voluntary movements.

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. Because any disturbance during a movement alters the length of the muscle and changes the activity in the muscle spindles, altering the spindle input to the alpha motor neuron compensates for the disturbance.

B. The discharge rate in the Ia sensory fiber of 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 (see Figure 35–9C). (Adapted, with permission, from Vallbo 1981.)

in peripheral nerves. Vallbo later showed 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 35–12B). This occurs because the gamma motor neurons, which have direct excitatory connections with spindles, are co-activated with alpha motor neurons.

Furthermore, when subjects attempt to make slow movements at a constant velocity, the firing of the Ia fibers mirrors small deviations in velocity in the trajectory of the movements (sometimes the muscle shortens quickly and 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 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.

Damage to the Central Nervous System Produces Characteristic Alterations in Reflex Response and Muscle Tone

Stretch reflexes can be evoked in many muscles throughout the body and 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. An electrical analog of the tendon jerk is the Hoffmann reflex (Box 35–4).

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. 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.

Box 35-4 The Hoffmann Reflex

The characteristics of the monosynaptic connections from Ia sensory fibers to spinal motor neurons in humans can be studied using an important technique introduced in the 1950s and based on early work by Paul Hoffmann. This technique involves electrically stimulating the Ia fibers in a peripheral nerve and recording the reflex response in the homonymous muscle. The response is known as the *Hoffmann reflex*, or H-reflex (Figure 35-13A).

The H-reflex is readily measured in the soleus muscle, an ankle extensor. The Ia fibers from the soleus and its synergists are excited by an electrode placed above the tibial nerve behind the knee. The response recorded from the soleus muscle depends on stimulus strength. At low stimulus strengths a pure H-reflex is evoked, for the threshold for activation of the Ia fibers is lower than the threshold for motor axons. Increasing the stimulus strength excites the motor axons innervating the soleus, producing two successive responses.

The first results from direct activation of the motor axons, and the second is the H-reflex evoked by stimulation of the Ia fibers (Figure 35-13B). These two components of the evoked electromyogram are called the M-wave and H-wave. The H-wave occurs later because it results from signals that travel to the spinal cord, across a synapse, and back again to the muscle. The M-wave, in contrast, results from direct stimulation of the motor axon innervating the muscle.

As the stimulus strength is increased still further, the M-wave continues to become larger and the H-wave progressively declines (Figure 35-13C). The decline in the H-wave amplitude occurs because action potentials in the motor axons propagate toward the cell body (antidromic conduction) and cancel reflexively evoked action potentials in the same motor axons. At very high stimulus strengths only the M-wave persists.

An interesting feature of the H-reflex is that its magnitude depends on motor experience. For example, it is low in highly trained ballet dancers and varies among different kinds of athletes. This strongly suggests that modification of spinal reflexes is an important process in learning motor skills.

Extensive studies of humans, monkeys, and rats by Jonathan Wolpaw and his colleagues have demonstrated that H-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 motor neuron properties. For down-conditioning they are dependent on corticospinal signals.

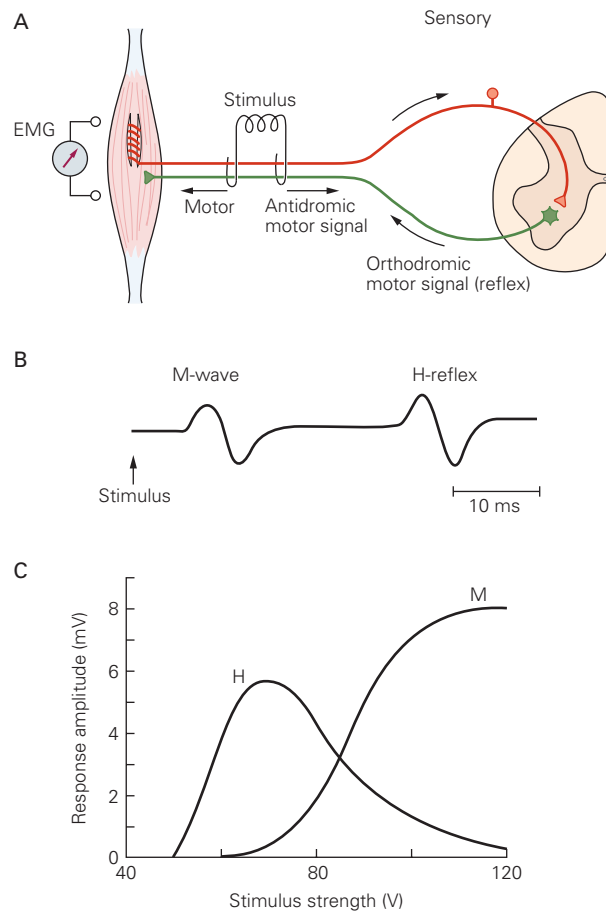


Figure 35-13 The Hoffmann reflex.

A. The Hoffmann reflex (H-reflex) is evoked by electrically stimulating Ia sensory fibers from muscle spindles in mixed nerves. The sensory fibers excite alpha motor neurons, which in turn activate the muscle. Muscle activation is detected by the electromyogram (EMG).

B. At intermediate stimulus strengths motor axons in the mixed nerve are excited in addition to the spindle afferents. Excitation of the motor neurons produces an M-wave that precedes the H-wave (H-reflex) in the EMG.

C. At low stimulus strengths only an H-wave is produced because only the spindle afferents are excited. As the stimulus strength increases, the magnitude of the H-reflex also increases, then declines, because the orthodromic motor signals generated reflexively by the spindle afferents are cancelled by antidromic signals initiated by the electrical stimulus in the same motor axons. At very high stimulus strengths only an M-wave is evoked. (Adapted, with permission, from Schieppati 1987.)

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

Muscle tone, 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. However, there is also a neural contribution to muscle tone; the feedback loop inherent in the stretch reflex pathway acts to resist lengthening of the muscle. The local neural circuits responsible for stretch reflexes provide the brain with a mechanism for adjusting muscle tone to suit different circumstances.

Because the strength of stretch reflexes is controlled by higher brain centers, disorders of muscle tone are frequently associated with lesions of the motor system, especially those that interfere with descending motor pathways. These conditions may involve either an abnormal increase in tone (*hypertonus*) or a decrease (*hypotonus*). The most common form of hypertonus is spasticity, which is characterized by hyperactive tendon jerks and an increase in resistance to rapid stretching of the muscle. A slowly applied stretch in a patient with spasticity elicits little resistance; as the speed of the stretch is increased, resistance to the stretch also rises progressively.

Thus spasticity is primarily a phasic phenomenon. An active reflex contraction occurs only during a rapid stretch; when the muscle is held in a lengthened position the reflex contraction subsides. In some patients, however, the hypertonus also has a tonic component; that is, the reflex contraction continues even after the muscle is no longer being lengthened.

The pathophysiology of spasticity is still unclear. It was long thought that hyperactivity of stretch reflexes in spasticity resulted from overactivity of the gamma motor neurons. Recent experiments have cast doubt on this explanation. Although gamma motor neurons may be overactive in some cases, changes in the background activity of alpha motor neurons and interneurons are probably more important. Especially important may be modifications in the properties of motor neurons that enable sustained firing in response to brief excitatory input (see Chapter 34).

Whatever the precise mechanism that produces spasticity, the effect is a strong facilitation of synaptic transmission in the Ia sensory fibers in the monosynaptic reflex pathway. Indeed, this can provide a mechanism for treatment. A common therapeutic procedure today is to mimic presynaptic inhibition in the terminals

of the Ia fibers by intrathecally administering the drug baclofen to the spinal cord. Baclofen is an agonist of GABA_B (γ -aminobutyric acid) receptors; binding of the drug to these receptors decreases the influx of Ca²⁺ into presynaptic terminals, reducing transmitter release.

Transection 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. 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 transection are reduced or completely suppressed. This condition is known as *spinal shock*. During the course of weeks and months spinal reflexes gradually return, often greatly exaggerated. For example, a light touch to the skin of the foot may elicit strong flexion withdrawal of the leg.

The mechanisms underlying spinal shock and recovery are poorly understood. The initial shock is thought to result from the sudden withdrawal of tonic facilitatory influence from the brain. Several mechanisms may contribute to the recovery, including sprouting of afferent sensory terminals and denervation supersensitivity owing to increased numbers of postsynaptic receptors.

Interestingly, the period of recovery from spinal shock is much shorter in animals than in humans. In nonhuman primates the recovery period is rarely more than a week; in cats and dogs it is only a few hours. The longer recovery period for humans presumably reflects the greater influence of supraspinal centers on spinal reflex circuits. This may in turn reflect the increased complexity of upright bipedal locomotion. Indeed, as we shall see in the next chapter, in humans with spinal cord injury the recovery of automatic locomotor patterns is slight compared with that of quadrupedal mammals.

An Overall View

Reflexes are coordinated, involuntary motor responses initiated by a stimulus applied to peripheral receptors. Some reflexes initiate movements to avoid potentially

hazardous situations, whereas others automatically adapt motor patterns to achieve or maintain a behavioral goal. The actual response evoked by a reflex depends on mechanisms that set the strength and pattern of responses according to the task and the behavioral state, or functional set. We know little about the details of these mechanisms, except that modification of synaptic transmission in spinal reflex pathways by descending signals from the brain is thought to be an important factor.

Many groups of interneurons in spinal reflex pathways are also involved in producing complex movements such as walking and transmitting voluntary commands from the brain. In addition, some components of reflex responses, particularly those involving the limbs, are mediated by supraspinal centers, such as brain stem nuclei, the cerebellum, and the motor cortex. Reflexes can be smoothly integrated into centrally generated motor commands because of the convergence of sensory signals onto spinal and supraspinal interneuronal systems involved in initiating movements. Establishing the details of these integrative events is one of the major challenges of contemporary research on reflex regulation of movement.

Because of the role of supraspinal centers in spinal reflex pathways, injury to or disease of the central nervous system often results in significant alterations in the strength of spinal reflexes. The pattern of changes provides an important aid to diagnosis of patients with neurological disorders.

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