

CHAPTER 15

REFLEXES

If you look at every textbook of neurophysiology and neurology in the library, you will not find many that give a definition of the term **reflex**. It is difficult to define the term in a way that includes everything we call a reflex, yet says anything that allows one to decide if any particular event is a reflex. We will follow suit in a sense and define a reflex as "a relatively stereotyped movement or response elicited

by a stimulus applied to the periphery, transmitted to the central nervous system and then transmitted back out to the periphery." Most reflexes involve activities that are nearly the same each time they are repeated, but no activity of an organism is fixed and independent of either the state or the history of the organism. Most reflexes involve the simplest of neural circuits, some only two or a few neurons; but many, like the scratch-reflex in a dog, are so complicated that their organization remains a mystery. Most reflexes are "involuntary" in the sense that they occur without the person willing them to do so, but all of them can be brought under "voluntary" control. Some reflexes serve protective functions, like the eyeblink reflex. Some reflexes act as control systems to maintain homeostasis in some bodily systems.

There are a number of ways of classifying reflexes. One is in terms of the systems that receive the stimulus and give the response. There are **viscerovisceral reflexes**, for example the decrease in heart rate that follows distention of the carotid sinus; **viscerosomatic reflexes**, like the abdominal cramping that accompanies rupture of the appendix; **somatovisceral reflexes**, such as the vasoconstriction that results from cooling the skin; and **somatosomatic reflexes**, like the knee jerk that follows tapping the patellar tendon. Reflexes can also be classified in terms of the number of neurons or synapses between the primary afferent neuron and the motor neuron. We distinguish two types, the **monosynaptic reflex** and the much more common **multisynaptic** or **polysynaptic**

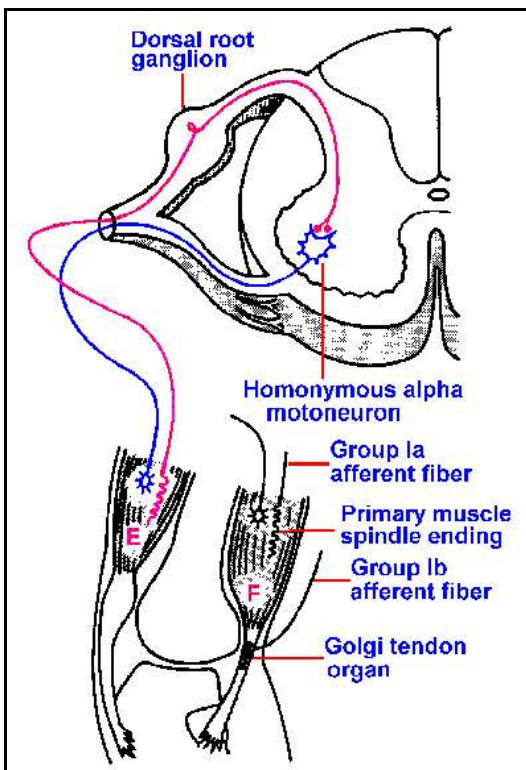


Figure 15-1. The tendon jerk reflex. A circuit diagram of the elements of the tendon jerk reflex: the muscle spindle, group Ia afferent fiber, alpha-motoneuron, and extrafusal muscle fiber. Note that this is a monosynaptic reflex. F and E indicate flexor and extensor muscles. (Schadé JP, Ford DH: *Basic Neurology*. Amsterdam, Elsevier, 1965)

reflex. The term multisynaptic implies that more than one synapse is involved, whereas polysynaptic usually implies that the pathway is of variable length, some parts disynaptic, some trisynaptic, etc.

The tendon jerk reflex

The simplest reflex is the monosynaptic reflex or the two-neuron reflex, an example of which is the **tendon jerk reflex** or **tendon tap reflex**, sometimes called the **myotatic reflex**. This is the reflex that is elicited by tapping the tendon just below the patella. The tap, applied to the tendons of the quadriceps muscles, stretches the muscles and their muscle spindles. A brisk tap excites the group Ia afferent fibers, because of their velocity sensitivity, ultimately causing the muscle to contract. The neural circuit for this reflex is shown in Figure 15-1¹. The group Ia afferent neuron enters the spinal cord through the dorsal root, penetrates into the ventral horn, and then synapses on an α -motoneuron. (This is the only synapse in the pathway within the spinal cord, thus the reflex is monosynaptic.) The axon of the α -motoneuron then exits the spinal cord through the ventral root and innervates the extrafusal fibers of the muscle from which the group Ia afferent fiber originated, i.e., the **homonymous muscle**. Note that the drawing shows only one neuron of each type, afferent and efferent, but that one represents many. For example, the cat soleus muscle contains about 50 group Ia afferent fibers and each soleus α -motoneuron appears to have a synaptic connection with each one of those 50 group Ia afferent fibers. A brief tap on the tendon will therefore activate many of the group Ia

afferent fibers, producing contraction of many of the soleus muscle fibers.

Tapping the tendon of the rectus femoris muscle of the quadriceps group produces a brief stretch of that muscle that acts as a powerful stimulus for the group Ia afferent fibers of the muscle, causing them to give a brief, synchronous discharge. Each discharge, after propagating down the group Ia axon to its termination, produces an EPSP in the rectus α -motoneurons. Because there are many EPSPs from many group Ia afferent fibers occurring nearly simultaneously in some α -motoneurons, the membrane potentials reach critical firing level (by spatial summation) with hypopolarization to spare, and the motoneurons discharge action potentials. The action potentials travel out by way of the ventral root to the muscle and, because the neuromuscular junction is an obligatory synapse, the muscle contracts. The contraction in turn causes the spindle to be unloaded or shortened passively, its equatorial region to relax, the group Ia afferent fiber to turn off, and the muscle to relax. This is the tendon jerk reflex.

Many of the homonymous α -motoneurons are not discharged by the Ia afferent fiber input, but have EPSPs evoked in them that do not achieve the critical firing level. The excitability of the motoneuron is therefore increased. This group of excited neurons is called the **subliminal fringe**. The presence of the subliminal fringe accounts for enhancement of the reflex response under certain circumstances, for example with the Jendrassik maneuver. In the Jendrassik maneuver, the fingers of the two hands are locked together and one hand pulls against the other while the tendon tap reflex is evoked. The reflex evoked is stronger than in the absence of the

¹ In this and the next three figures, E and F stand for extensor and flexor muscles.

maneuver. (Interestingly, mental arithmetic and a number of other activities will do the same thing!) During the Jendrassik maneuver activity, originating perhaps in the cervical enlargement of the spinal cord or some other rostral center, descends the spinal cord to excite α -motoneurons. This activity by itself does not cause the α -motoneurons to discharge or the muscle to contract, but when added to the subthreshold excitation of the subliminal fringe caused by the tap-induced muscle stretch, it causes the neurons in the subliminal fringe to discharge. The reflex contraction will therefore be larger than normal. There may also be some influence of increased γ -motoneuron activity, increasing the sensitivity of the primary spindle endings, but this influence should be small because the stimulus for the reflex is very brief.

The value of the stretch reflex mechanism may not be clear at first, but some reflection may clarify its role in motor control. It is unlikely that muscles undergo such rapid stretches very often, with the possible exception of when a person jumps off a wall or jumps up and down on a pogo stick. However, in these instances, the rapid stretch of the rectus femoris that occurs when the feet or the pogo stick contract the ground causes a reflex contraction that helps prevent the gluteus from being overly bruised.

Usually, the postural muscles experience relatively slow, sustained stretches and the anti-gravity muscles, of which the quadriceps is an example, are pulled upon by gravitational forces. This steady force sets up a sustained discharge in each group Ia afferent fiber, but the discharges in different fibers are not synchronized as they are when the tendon is tapped. In addition, longer, larger stretches are able to excite secondary

muscle spindle receptors which also have connections with homonymous α -motoneurons, di- and trisynaptic ones. These longer, larger stretches therefore activate the α -motoneurons by both monosynaptic and polysynaptic reflex pathways. The resulting reflex contraction of the muscle is called the **stretch reflex**. The polysynaptic effects are not seen in the tendon tap reflex for two reasons: (1) the brief stretch does not excite secondary spindle receptors and (2) the brief input over the polysynaptic pathways arrives after the monosynaptic input and finds the α -motoneurons in their refractory periods and therefore cannot cause them to discharge again.

In controlling posture, the asynchronous discharge in mono- and polysynaptic pathways induced by gravitational forces on muscles sums in the α -motoneuron with other activity from within the CNS to produce a contraction that just balances the gravitational force. If an additional force is applied, stretching the muscle, additional tension is developed by the stretch reflex to counteract that force. In this way, the stretch reflex serves as a mechanism for maintaining an upright body orientation under a variety of load conditions; the mechanism is automatic ("unconscious") and fast (19-24 msec for the quadriceps in man).

In addition to the monosynaptic connections of the group Ia afferent fibers with the homonymous α -motoneurons (e.g., rectus femoris Ia with rectus α -motoneuron), there are also monosynaptic connections with synergistic α -motoneurons, those innervating muscles that act in the same way at the same joint, but the effects are not as strong in the **synergists** as they are in the homonymous α -motoneurons. Thus, the rectus group Ia afferent fibers also excite the

vastus α -motoneurons, though not as strongly as they do the rectus α -motoneurons. Fewer of the synergistic α -motoneurons actually discharge, and the subliminal fringe is larger than for homonymous α -motoneurons.

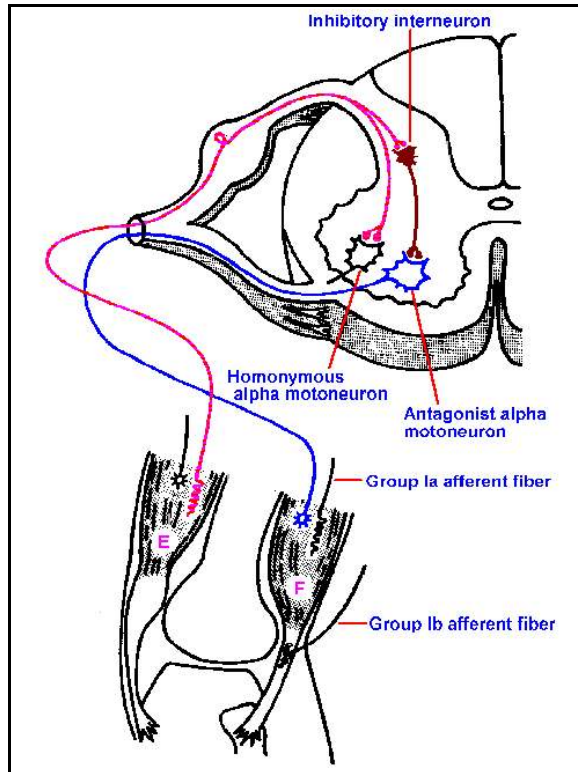


Figure 15-2. Reciprocal innervation. A circuit diagram showing the collateral of the group Ia afferent fiber synapsing on an inhibitory interneuron that synapses on the alpha-motoneuron of the antagonist muscle. F and E indicate flexor and extensor muscles. (Schadé JP, Ford DH: *Basic Neurology*. Amsterdam, Elsevier, 1965)

Most skeletal muscles exhibit a tendon tap reflex, but the reflex is strongest in the **antigravity** or **physiological extensor muscles**. This makes some sense in light of the discussion of the last paragraph. Note that physiological extensors are not necessarily anatomical extensors. The biceps brachii are a case in point; they are

anatomical flexors of the elbow but they are physiological extensors, moving the forearm against gravity.

It is clinically important to note that these reflexes involve only one or two segments of the spinal cord. In fact, the spinal cord can be cut above and below these segments, and the reflexes will still occur. For this reason, testing such reflexes cannot be used as an indicator of the condition of the brain or even other segments of the spinal cord.

Reciprocal innervation

When the extensor muscle contracts during such a reflex, there is usually a relaxation of its **antagonists**, the flexor muscles crossing the same joint. If this did not occur, the reflex movement could be resisted and diminished by the force of the antagonist muscle². The neural mechanism underlying this relaxation of the antagonist muscles is shown in Figure 15-2. The group Ia afferent fiber, after entering the spinal cord, gives off a collateral branch that synapses on an interneuron. This interneuron, in turn, synapses on the antagonist α -motoneuron, in the case of our previous example, a hamstring motoneuron. Its effect on the hamstring α -motoneuron is inhibitory, i.e., it causes an IPSP in the α -motoneuron that is ultimately manifested as a relaxation of the muscle. This is **reciprocal inhibition**. Notice that this is a polysynaptic reflex pathway.

It is a general principle that anything that

² Actually, movements that require precise control of muscle activity involve co-contraction of the antagonist muscles at a joint, but the force produced by one of them must be greater than that produced by the other for the joint to move.

has an excitatory (or inhibitory) influence on an α -motoneuron also inhibits (or excites) the α -motoneurons of its antagonist muscle. This is the **principle of reciprocal innervation**. Thus, for example, excitation of the hamstring α -motoneurons by group Ia afferent fibers is accompanied by inhibition of quadriceps α -motoneurons. Reciprocal inhibition is a specific example of the more general principle of **reciprocal innervation**.

Dale's principle

At this point, it is appropriate to bring up the reason for interneurons in pathways such as that in Figure 15-2. **Dale's principle** (formulated by Sir Henry Dale) states that a single neuron synthesizes only one transmitter substance. Therefore according to the principle, if a neuron secretes acetylcholine at one of its terminals, it secretes acetylcholine at all of its terminals. Sir John Eccles has extended this notion to say that the effect of the transmitter released by a single fiber is the same at all its terminals. This would imply that the net result of activity in all group Ia afferent fibers is excitation and only excitation. Thus, if it is necessary to have inhibition in a pathway involving group Ia afferent fibers, then an inhibitory interneuron must be interposed.

Some neurons make, or at least contain, more than one of the putative transmitter substances discussed in Chapter 13. However, it is not known whether all of the substances made by a neuron are actually used as transmitter substances by that neuron. For example, some neurons contain both GABA and somatostatin or GABA and some neuropeptide. Many peptides are thought to be neuromodulators rather than traditional transmitter substances. Therefore, it is not clear that this is a

violation of Dale's principle. On the other hand, the idea that the effect of a transmitter substance is everywhere the same is not tenable. It is now known that certain neurons in *Aplysia californica*, the sea slug, secrete acetylcholine at synapses with two different postsynaptic cells, and, in one cell, the transmitter substance evokes an EPSP and, in the other, an IPSP. Thus, the action of the transmitter substance on a neuron depends upon that neuron and the receptors or channels it possesses not on the transmitter substance or the neuron that released it. Whether this behavior is also a characteristic of mammalian neurons is not yet certain. Nevertheless, the usual approach in neurophysiology has been to interpose an interneuron whenever the sign of the effect changes, whether there is direct evidence of an interneuron or not.

The flexion reflex

If you have ever touched a hot object or stepped on a sharp object and withdrawn your hand or foot, you have experienced a **flexion reflex**, a **nocifensive reflex**, or a **withdrawal reflex**, all terms describing the same event. The protective result of this reflex is obvious; it quickly removes the part of the body from the vicinity of the offending object by contracting the appropriate muscles, usually flexors, and relaxing extensor muscles (again, reciprocal innervation). The vigor of the reflex depends upon the strength of the stimulus. A weak pinch produces flexion of the foot; a slightly stronger one, flexion of both the foot and the leg; and a very strong one, flexion of foot, leg, and even hip. This spread of the reflex with stronger stimulation is called **irradiation**. The exact nature of the limb movement and the final position of the limb vary depending upon the site of stimulation.

This phenomenon is often called **local sign**. Because of local sign, the withdrawal of the limb from damaging stimuli is usually appropriate in both magnitude and direction.

The pathways for the flexion reflex are illustrated in Figure 15-3. The afferent limb (the part going to the spinal cord) of this reflex consists of nociceptors with A δ or C fibers and fibers of groups II, III, and IV of muscle. These are sometimes referred to collectively as the **flexor reflex afferent fibers**. They enter the spinal cord and synapse on interneurons, whose axons distribute to other interneurons that affect α -

motoneurons within the same and in different segments of the spinal cord. Notice that this is a polysynaptic reflex. Activity in the nociceptive afferent fibers in the common peroneal nerve serving the first and second toe leads to discharge of the peroneus α -motoneurons, which, in turn, leads to dorsiflexion of the foot. If the nociceptive activity is strong enough, it is able to activate other peroneus α -motoneurons, further increasing the flexion of the foot, and also to bring in α -motoneurons of synergistic muscles of the foot, as well as other related muscle groups, for example the hamstrings, to lift the leg. This may involve transmission to spinal segments other than the segment of entry.

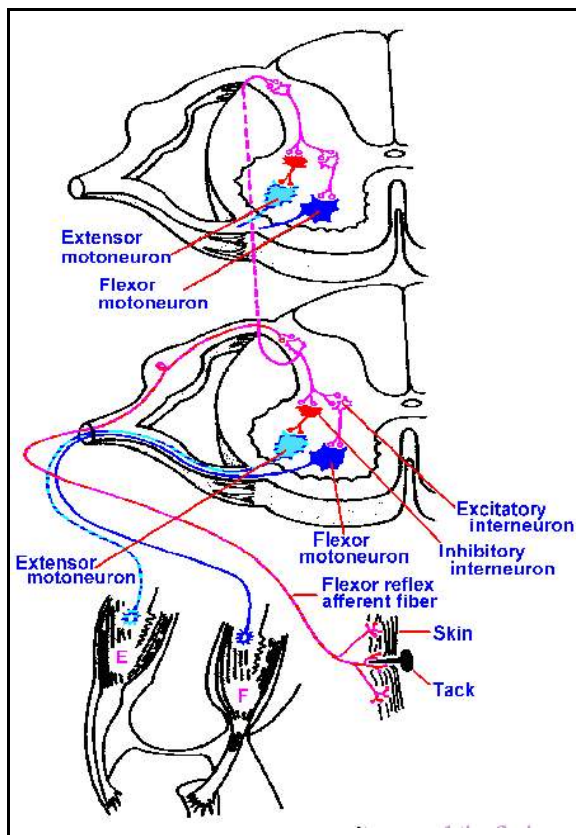


Figure 15-3. Flexion reflex. A circuit diagram of the flexion reflex showing afferent fibers from skin, interneurons, and flexor α -motoneurons in two spinal cord segments. Note that some interneurons are intersegmental. F and E indicate flexor and extensor muscles. (Schadé JP, Ford DH: *Basic Neurology*. Amsterdam, Elsevier, 1965)

The crossed-extension reflex

If protection of the limb requires it to be elevated, then the rest of the body is imperiled by removal of the support the limb normally provided, unless some compensation is made. The reflex contraction of flexor muscles on one side of the body is always accompanied by contraction of the extensor muscles of the contralateral limb. This gives increased antigravity support on the contralateral side to hold the body upright and is called the **crossed-extension reflex**. The circuit for this reflex is illustrated in Figure 15-4. The flexor reflex afferent fibers also synapse on interneurons that decussate (cross the midline) and terminate on contralateral extensor α -motoneurons. This pathway is polysynaptic and purely excitatory. In addition, there is the usual reciprocal inhibitory effect on the contralateral flexor α -motoneurons.

Autogenic inhibition

Activity in the group Ib afferent fibers,

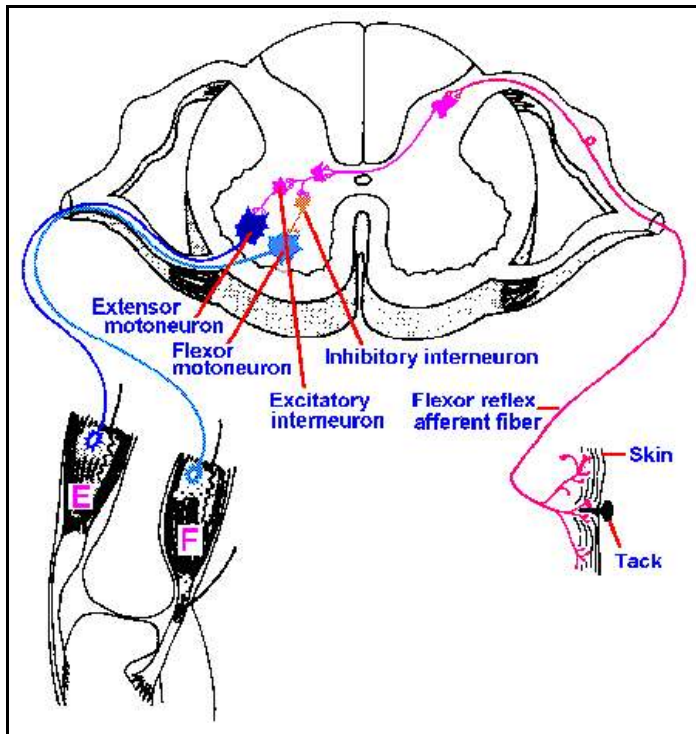


Figure 15-4. Crossed-extension reflex. Circuit diagram showing branching of the flexor reflex afferent fibers and their termination on an interneuron that decussates and terminates on contralateral extensor α -motoneurons. The circuit for the contralateral reciprocal inhibition of flexor α -motoneurons is also shown. F and E indicate flexor and extensor muscles. (Schadé JP, Ford DH: *Basic Neurology*. Amsterdam, Elsevier, 1965)

associated with Golgi tendon organs, inhibits the homonymous α -motoneurons. The inhibition is disynaptic involving one interneuron and two synapses between the afferent fiber and the motoneuron. The effect is called **autogenic inhibition** (sometimes written autogenetic inhibition). Like the group Ia excitation, group Ib inhibition is exerted not only on the homonymous α -motoneurons, but also on the α -motoneurons of synergistic muscles. Reciprocal innervation is also present here, with group Ib activity exciting the α -motoneurons of the antagonist muscles.

It was originally thought that this inhibition served only to protect the muscle

from being injured when contracting against too heavy a load. This concept came from two sources: (1) the supposed high threshold of group Ib fibers to tension (not true for *developed* tension) and (2) the existence of a clasp-knife reflex. The clasp-knife reflex exists only in certain pathological conditions, e.g., "upper motoneuron" disease. Under these conditions, there is an extensor spasticity that resists any attempt to flex the limbs, especially the arms. If the arm is gradually, forcibly flexed by someone other than the patient, a point will be reached when the resistance to flexion suddenly melts away, and the limb collapses easily into full flexion, reminiscent of the action of a pocket or clasp-knife. This is the **clasp-knife reflex** or the **lengthening reaction**. It was thought formerly to be mediated by autogenic inhibition and to occur near the threshold for group Ib activation by increased muscle tension. Now it is thought that it occurs at the point where autogenic inhibition is great enough to overcome the stretch

reflex excitation, that is, when the membrane potential of the extensor motoneuron falls below the critical firing level and the muscle relaxes. Actually, Golgi tendon organ activity cannot entirely account for the clasp-knife reflex. As the spastic muscle is lengthened, the group Ia discharge continually increases and, because the tension is increasing, the group Ib discharge also increases. The result, at the α -motoneuron, is excitation from the group Ia fiber input and inhibition from the group Ib fiber input, the inhibition finally taking dominance and silencing the α -motoneuron. The muscle relaxes. This accounts for the relaxation of the spastic muscle, but it does

not account for the failure of its immediate return. When the muscle relaxes, the tension falls, and the group Ib fiber discharge ceases. This should decrease the autogenic inhibition and allow group Ia fiber input to reestablish the spasticity, but the muscle stays relaxed until the joint is extended again, whereupon the spasticity reappears. Recent evidence suggests that group Ia fibers from muscle spindles also contribute to autogenic inhibition (Fetz EE, Jankowska E, Johannisson T, Lipski J: *J Physiol (Lond)* **293**:173-195, 1979). Because the muscle does not shorten, but lengthens when the spasticity disappears, the group Ia fibers will discharge even more briskly, increasing both excitation and autogenic inhibition of the α -motoneurons. Apparently, the inhibition predominates.

In the normal person, it is certain that the Golgi tendon organ contributes to control of muscle activity over the whole range of movement, not just at its extremes. There is no reflex movement associated with stimulation of Golgi tendon organs in isolation, so that nothing can be said with certainty about their reflex function. Perhaps they do serve a protective function, but they may also be supplying tension information for complicated tension-maintaining reflexes or supplying inhibition at the appropriate moment to switch from flexion to extension movements in walking or running.

They may also play a role in increasing muscle force during fatigue. Thus, during fatigue the muscle produces less force, which reduces Golgi tendon organ activity. This leads to decreased inhibition and, therefore, increased activity of the homonymous α -motoneurons. The increased motoneuron activity will lead to greater force of contraction. The problem

with this scenario is that the resulting increased force should lead to increased Golgi tendon organ activity and, so, decreased force. How this complicated system is working is yet to be determined.

Other reflexes

There are a number of other reflexes commonly tested in the clinic, whose mechanism we do not understand thoroughly. The **extensor thrust reflex** involves extension of the lower limb when a tactile stimulus is applied to the plantar surface of the foot. This may play some role in walking or standing by maintaining contact with the surface. The **Babinski sign** is a reflex that is pathological in adults but normal in infants. Scraping the sole of the foot of a normal adult with a tongue depressor results in plantar flexion of the toes. In the infant and the spinal adult (with a spinal cord transection), the same stimulus leads to dorsiflexion of the toes. This pathological change (in adults) is called Babinski's sign, and, as we shall see, it is usually taken as a sign of pyramidal tract damage.

There are a great many other reflexes that could be mentioned here. We have already discussed reflex control of pupil diameter and lens shape in the eye and auditory sensitivity by the middle ear muscles and olivocochlear bundle. In addition, there are stretch receptors in the lungs, probably in the bronchi and bronchioles, that are activated by lung inflation. The activity of these receptors enters the medulla by way of the vagus nerve (Xth cranial nerve) to produce inhibition of inspiratory neurons. This effect, called the **Hering-Breuer reflex**, has the effect of terminating inspiration when a certain level of inflation has been attained. It is mediated

through the nucleus solitarius and the reticular formation.

There are other respiratory reflexes as well. For example, the stretch reflex and autogenic inhibition play a major role in control of the intercostal muscles during inspiration. The gamma loop appears to provide an important facilitation of discharges in α -motoneurons to the external intercostals in particular. A most important reflex pathway regulates the concentrations of oxygen and carbon dioxide in arterial blood by adjusting respiratory parameters. Chemoreceptors in the carotid and aortic bodies increase their discharge rates in response to a decrease in arterial oxygen concentration or an increase in arterial carbon dioxide concentration (or, alternatively, a decrease in pH). Sensory fibers from these receptors enter the brain stem through the glossopharyngeal nerve (IXth cranial nerve), where they make connections with cells whose activity results in an increase in respiratory volume and rate, with certain changes in peripheral vasculature, to elevate blood oxygen or decrease blood carbon dioxide levels. In addition, there are chemoreceptors in the CNS that are excited by elevation of the carbon dioxide of the cerebrospinal fluid or a decrease in its pH. Their activity results in the same changes in respiration, apparently without much change in the cardiovascular system.

There are also reflexes involving the heart. Baroreceptors (pressure receptors) in the carotid sinus, in the aortic arch, and perhaps elsewhere, increase their discharge rates in response to elevated blood pressure. This increase in activity leads reflexly to a decrease in sympathetic discharge, resulting in peripheral vasodilatation and in an increase in parasympathetic discharge

primarily through the vagus nerve, resulting in decreased heart rate. The net result is a decrease in blood pressure. Stimulation of high threshold (small diameter) primary afferent fibers in cutaneous and muscle nerves also leads to a reflex increase in blood pressure.

Vomiting is the result of a reflex arc involving receptors in the region of the pharynx, supplied by the IXth and Xth cranial nerves and also receptors in the medulla. The medullary receptors, located in a region with high permeability of the blood brain barrier, are sensitive to chemical agents in the blood. The efferent limb of the reflex involves somatic innervation of the abdominal muscles and autonomic innervation of the stomach (smooth) muscles and the cardiac sphincter. During vomiting, the cardiac sphincter relaxes, while the abdominal and stomach muscles contract, expelling the stomach contents through the esophagus.

Swallowing is also a reflex activity. The reflex is triggered by tactile stimulation of the mucosa of the palate, pharynx, and epiglottis. Receptors in these regions, perhaps free nerve endings, send their activity through the IXth cranial nerve, the superior laryngeal branch of the Xth cranial nerve, and perhaps also the Vth cranial nerve. The reflex pathway involves synapses in the nucleus solitarius and efferent fibers to the tongue in the hypoglossal nerve (XIIth cranial nerve) and to the palate, pharyngeal, and esophageal musculature through the IX, X and XIth cranial nerves from the nucleus ambiguus.

Temperature regulation (see also Chapter 17) is a complicated reflex activity, involving both peripheral thermoreceptors and central thermoreceptors located in the anterior hypothalamus and perhaps

elsewhere. The reflex pathways are not well understood, but they involve hypothalamic activation of the autonomic system, resulting in peripheral vasoconstriction, piloerection, and shivering, if heat needs to be generated or conserved, and in sweating, increased respiration, and peripheral vasodilatation, if heat needs to be lost in maintaining body core temperature at around 37°C.

Micturition is a reflex activity that depends upon stretch receptors located in series with smooth muscle cells of the bladder wall. This mode of attachment allows the receptors to discharge both when the muscles are stretched and when they contract. Fibers from these receptors are carried in the pelvic nerves to the S₂-S₄ spinal cord segments. Somatic afferent fibers to these same segments play a role in the reflex as well. Bladder emptying is produced by contraction of the detrusor muscle of the bladder and also muscles of the abdominal wall, with relaxation of the internal (smooth muscle) and external (striated muscle) sphincters. Reflex emptying can be accomplished with an isolated sacral spinal cord, provided that the sacral roots, especially ventral root S₃, are intact, but usually the reflex is under strong control from supraspinal centers.

The reflex emptying of the colon, i.e., defecation, is accomplished in a way similar to micturition. Here, the receptors are located in series with smooth muscle of the rectal wall. Tactile receptors in the skin and anus also facilitate defecation, as does the simultaneous occurrence of a micturition reflex. Emptying is under parasympathetic control from the S₂-S₄ spinal cord segments, producing contraction of the smooth muscle lining the rectum and relaxation of the sphincters. Evacuation is also assisted by contraction of the diaphragm and abdominal

muscles with the glottis closed (Valsalva's maneuver), increasing the pressure in the abdominal cavity.

There are also sexual reflexes whose organization is partially understood. The afferent fibers for penile erection and ejaculation reflexes arise from the genitalia and course through branches of the pudendal nerve, again, to spinal cord segments S₂-S₄, although many other stimuli can elicit sexual responses under appropriate circumstances. Many of these other stimuli must act through higher centers. Both somatic and autonomic efferent fibers are involved in producing erection, with parasympathetic fibers producing vascular engorgement and somatic motoneurons to the bulbocavernosus and ischiocavernosus muscles producing contraction that compresses the venous outflow from erectile tissues. Sympathetic fibers also play a poorly understood role; bilateral sympathectomy reduces or eliminates both erections and ejaculations. Sympathetic, parasympathetic and somatic motor activity are all involved in producing ejaculation.

These are a few of the many reflexes mediated by the nervous system. Some are mediated by the spinal cord and can occur when the spinal cord is disconnected from the brain, but some require intact connections with the brain stem or diencephalon for their operation. All, however, are under the influence of cortical and subcortical structures in their normal operation.

The function and utility of reflexes

Many of the reflexes are artifacts of the way we stimulate the body (although many, like the eye blink reflex, are essential to protect body parts from injury and many play specific regulatory roles). However,

they tell us two useful things: (1) how the nerve circuits of the spinal cord or brain stem are put together and (2) the condition of various parts of these circuits. The first piece of information is useful to the neurophysiologist, who ultimately wants to know how the nervous system works. However, we will see in the next chapter that many reflexes are suppressed or modified during phases of the stepping cycle in order to prevent muscles from contracting at the wrong times. The state of a reflex circuit may be highly variable depending upon the condition of a person, e.g., awake, asleep, comatose; upon what he is doing, e.g., walking, running, thinking; upon his position in space or posture; and upon other factors. Therefore, the reflex pattern at any one time may not be a good indicator of the neural circuits available in the nervous system or of how they behave under a variety of conditions.

The second piece of information is useful to the clinician, who wants to know what is wrong with a patient. An example may help to show how the reflexes are used clinically. A patient complains of **paresis** or weakness of the right leg. There are three possible causes for this paresis: (1) he is suffering from disease of descending tracts having to do with leg function; (2) he is suffering from disease of the α -motoneurons themselves; or (3) something is wrong with the muscle. A normal tendon tap reflex rules out the last two alternatives, because the α -motoneurons and muscle must be functional to give a normal reflex.

Reflex pathways as control systems

One way to think of the function of

reflexes is as **control systems**³, systems that regulate the various parameters of muscle contraction. The essential feature of this type of system is the more-or-less continuous flow of information from the element controlled back to the device that controls it. This is called **feedback**. An everyday example of feedback is the continuous information your eyes give you about the course your automobile is taking. Without visual information, you are likely to run off the road before you can detect errors in your steering. With visual feedback, small corrections can be made in your steering before you leave the road.

A convenient way of illustrating a control system is to use a block diagram such as that shown in Figure 15-5. In a block diagram, boxes indicate the components of the system, the name of the box indicates the operation performed by the box, and arrows indicate the direction of flow of signals in the system. The terms indicated in parentheses refer to the car example. The **controlled system** has an input that can be varied by a **controller**. This input interacts with influences from outside the system, called **disturbances**, in producing the **output** of the system, which is the parameter to be controlled. In the automobile example, the input is the position of the steering wheel, the output is the position of the car on the road, and a dis-

³ These same principles will also apply to regulation of a number of physiological parameters such as blood pressure, body temperature and body weight. A complete description of all possible applications is beyond the scope of this chapter. For further discussions, see Guyton AC et al. *Circ Res* **35**:159-176, 1974 or Jaros GG et al. *Ann Biomed Engn* **8**:103-141, 1980.

turbance might be a sudden gust of wind that forces the car off its intended course. The **sensor** is a device that measures the output of the system and its measurement is the **feedback signal** to the **error detector**. The feedback signal is compared with the **control signal** (the signal that specifies the intended output) by the error detector, which, when it finds a difference between the two signals, sends an **error signal** to the controller to increase its output and reduce the amount of error. The actual output is brought closer to the intended output, the new output is again sensed by the sensor, and a new correction is made, bringing the

system even closer to the intended output, and so on. In our car example, the error detector, sensor, and controller are all within the human operator. He senses the position of the car with his eyes (sensor), compares that with the course of the road (control signal), and, if they are not the same (an error exists), uses his muscles (controller) to turn the steering wheel to bring the car (controlled system) back to the appropriate position on the road (output). When the wind gusts, the car deviates from the course that would ordinarily be determined by the position of the wheel; the driver senses this and corrects for the disturbance.

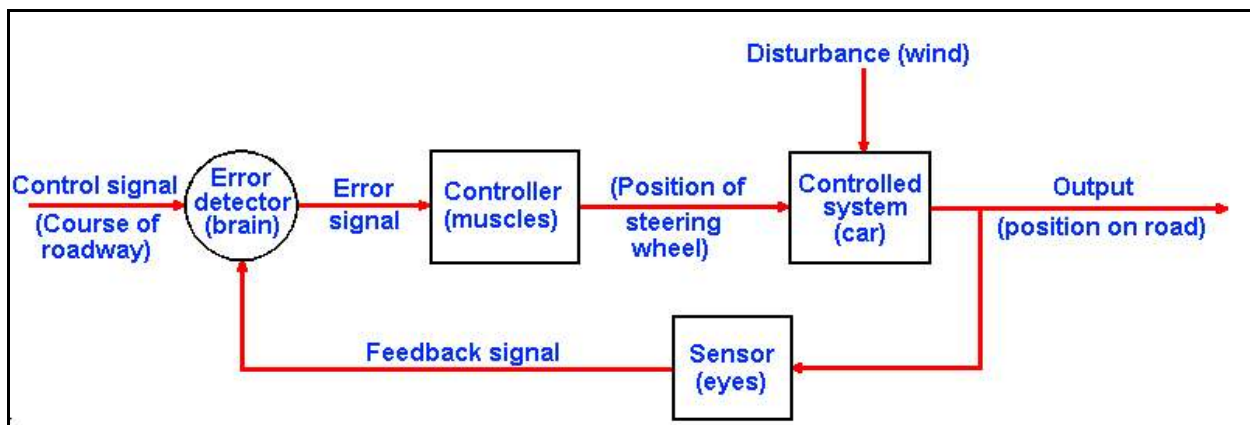


Figure 15-5. Block diagram of a generalized feedback control system. The operation performed by each component is indicated by its name. Arrows indicate the unidirectional flow of information. Any feedback control system can be drawn in this form. Terms indicated in parentheses refer to the car analogy used in the text. (Houk J, Henneman E: Feedback control of muscle: introductory concepts. In Mountcastle VB [ed]: *Medical Physiology, 13th ed., Vol. 1*. St. Louis, Mosby, 1974)

It is possible to view the control of the driver's muscles in the same way. The muscle spindle receptors and their reflex connections form a feedback system to control muscle length, as shown in the block diagram of Figure 15-6. In this case, the control signal is a command from some

supraspinal center to maintain the biceps muscle at some fixed length in order to hold a chair at a fixed distance off the floor. This signal is fed to spinal neurons, which are the error detectors, with the α -motoneurons as the controller that signals the muscle (controlled system) to contract, producing

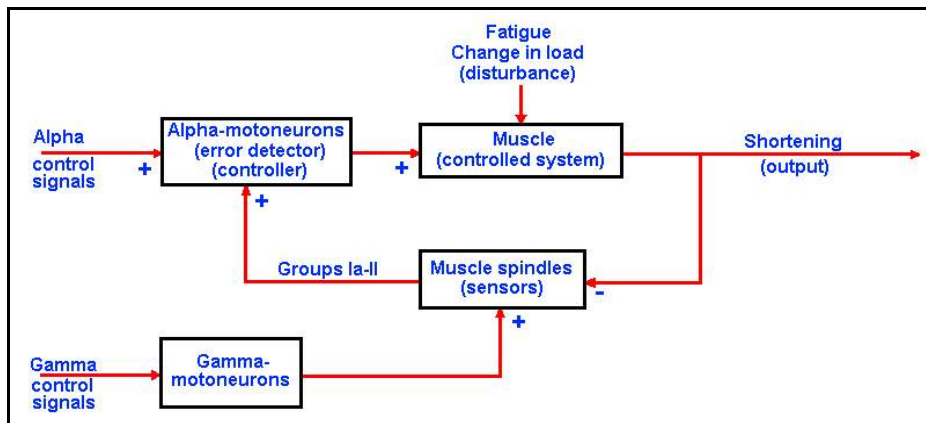


Figure 15-6. Block diagram of the control system involving the stretch reflex. This system controls muscle length. (Houk J, Henneman E: Feedback control of muscle: introductory concepts. In Mountcastle VB [ed]: *Medical Physiology*, 13th ed., Vol. 1. St. Louis, Mosby, 1974)

enough force to decrease its length (output) and lift the chair off the floor. The muscle spindle endings (length and velocity sensors) sense the length of the muscle and transmit its length (feedback signal) back to the α -motoneuron, where it is compared with the control signal. If the length of the muscle (or the height of the chair) is not as intended, a new error signal is generated, commanding more or less force and so on, around the system loop until the actual and intended muscle lengths are the same. Small deviations from the length specified by the control signal, for example due to fatigue, are sensed and quickly corrected in the same way.

Suddenly, someone drops a heavy book onto the chair and a disturbance of the system is produced by the sudden downward movement of the chair and the lengthening of the muscle. The spindle receptors sense the increased length and signal this increase to the α -motoneurons, which detect the error and command more force to be generated by the muscle and the load to be lifted back to the intended height. In this manner, the system helps to maintain the length of the

muscle in the face of changes in the load being lifted. Other disturbances are, of course, possible. The lifter's other hand could touch a hot object, evoking a flexion reflex and a crossed-extension reflex, or the ground could suddenly tilt due to an earthquake, yet the length of the muscle will be maintained constant

until a new control signal comes to the α -motoneuron. Suppose a new control signal orders the chair to be held higher. This new control signal will produce a large error signal, because it is quite different from the feedback signal. As a consequence, the controller will increase its output, resulting in more force that decreases the error. There will be a continuous cycling around the loop until the error is reduced to zero, and the new intended length is achieved. Notice that the γ -motoneurons affect the sensitivity of the sensors, so that any input that excites the γ -motoneurons will result in an increase in the feedback signal because of the increase in spindle activity. The γ -motoneurons also receive their own control signals, perhaps independent of those to α -motoneurons, especially from cutaneous receptors and many supraspinal pathways, including the corticospinal, reticulospinal, and rubrospinal tracts. However, in general, pathways that influence α -motoneurons also influence γ -motoneurons (**alpha-gamma coactivation**), with the striking exception that group Ia afferent fibers do not influence γ -motoneurons.

It has been suggested that some movements are initiated, not by control signals to α -motoneurons, but by control signals to γ -motoneurons. An increase in γ -motoneuron activity would result in a shortening of the intrafusal muscle fibers and an increase in the discharge of group Ia afferent fibers. This, in turn, would lead to an increase in α -motoneuron activity, causing contraction of the extrafusal muscle fibers and shortening of the whole muscle. This mechanism is called the **follow-up length servo mechanism** of contraction, because the length of the spindle (at least the receptor region of the spindle) controls the length of the muscle, and the changes in the length of the muscle follow the changes in the length of the spindle. It is not likely that this is the only mechanism by which contractions are initiated because of the alpha-gamma coactivation from most sources. Contractions are probably initiated mainly by direct action on the α -motoneurons, but perhaps in some situations they are initiated by the follow-up length servo mechanism.

When a movement is initiated by α - γ coactivation, the coactivation can act as a servo-assistor. If the estimated load, as determined by the command signal, is correct, i.e., if the actual load equals the estimated load, the spindle discharge will remain constant and no further reflex α -motoneuron excitation will occur. If the actual load exceeds the estimated load, there will be a misalignment of the intra- and extrafusal fibers, a large spindle discharge, and an increased α -motoneuron discharge with increased tension. On the other hand, if the actual load is less than the estimated load, there will be a different misalignment of the intra- and extrafusal fibers and a reduced spindle discharge leading to a

reduced α -motoneuron activation. In any case, α - γ coactivation insures that the spindles will continue to act as length detectors even when the muscle shortens.

Summary

A monosynaptic reflex involves only two neurons and the one synapse between them in the central nervous system. The only monosynaptic reflexes known involve the primary muscle spindle or group Ia afferent neurons and the α -motoneurons of the muscle from which the afferent fibers originated and its close synergists. This is the anatomical substrate of the tendon tap reflex elicited by tapping the tendon of the muscle. In order for a muscle's contraction to cause joint rotation, its antagonists must relax or at least generate less tension. The mechanism for preventing co-contraction is reciprocal inhibition, in which the group Ia afferent fibers from the contracting muscle excite interneurons (one or more) whose ultimate effect on the antagonist α -motoneuron is inhibition. Dale's principle and Eccles' extension of the principle lead to the interposition of an inhibitory interneuron between the excitatory primary afferent fibers and the inhibited motoneuron, though there may be other ways of accomplishing this change in the "sign" of the effect. Reciprocal inhibition is an example of a polysynaptic reflex. Another example is the flexion reflex, started by stimulating cutaneous nociceptors and high threshold muscle afferent fibers and involving interneurons in several segments of the spinal cord and α -motoneurons of several flexor muscles. To compensate for the resulting flexion movement, the extensor muscles on the other side of the body are contracted in the crossed-extension reflex. This too is a polysynaptic reflex. Autogenic

inhibition involves disynaptic inhibition of homonymous α -motoneurons by group Ib afferent fibers (and also group Ia fibers) and usually shows up in pathological conditions such as decerebrate rigidity. Reflexes act to protect various parts of the body, and they serve as indicators of the way the nervous system is put together. Clinically, they are used to test the integrity of various parts of nervous system pathways. These same reflex circuits also function in control systems that continuously modulate the contraction of muscles to produce behavior.

Suggested Reading

1. Fetz EE, Jankowska E, Johannisson T: Autogenetic inhibition of motoneurons by impulses in group Ia muscle spindle afferents. *J. Physiol. (Lond)* **293**: 173-195, 1979.
2. Granit R: *The Basis of Motor Control*. New York, Academic Press, 1970.
3. Guyton AC, Coleman TG, Cowley AW Jr, Manning RD Jr, Norman RA Jr, Ferguson JD: A systems analysis approach to understanding long-range arterial blood pressure control and hypertension. *Circ Res* **35**:159-176, 1974.
4. Henneman E: Spinal reflexes and the control of movement. In Mountcastle VB [ed]: *Medical Physiology. 13th ed., Vol 1*, St. Louis, Mosby, 1974.
5. Homma S [ed]: *Understanding the Stretch Reflex, Vol. 44. Progress in Brain Research*. Amsterdam, Elsevier, 1976.
6. Houk J, Henneman E: Feedback control of muscle: Introductory concepts. In Mountcastle VB [ed]: *Medical Physiology, 13th ed.*, St. Louis, Mosby, 1974.
7. Hunt CC, Perl ER: Spinal reflex mechanisms concerned with skeletal muscle. *Physiol Rev* **40**:538-579, 1960.
8. Jaros GG, Guyton AC, Coleman TG: The role of bone in short-term calcium homeostasis: An analog-digital computer simulation. *Ann Biomed Engn* **8**:103-141, 1980.
9. Matthews PBC: The human stretch reflex and the motor cortex. *Trends in Neuroscience* **14**:87-90, 1991.
10. Matthews PBC: *Mammalian Muscle Receptors and their Central Actions*. Baltimore, Williams and Wilkins, 1972.
11. Prochazka A: Proprioceptive feedback and movement regulation. In: Rowell L, Sheperd JT (eds). *Handbook of Physiology: Regulation and Integration of Multiple Systems*, pp. 89-127. New York: American Physiological Society, 1996.