CASE 46

An 18-year-old man is brought to the emergency center by the emergency medical service after being involved in a serious motor vehicle accident. On physical examination, the patient has normal vital signs but he cannot move and has no sensation in either of the lower extremities, which are flaccid and without reflexes. Magnetic resonance imaging (MRI) reveals a fracture of the lower spine with significant swelling of and injury to the spinal cord.

◆ Which tracts are responsible for direct cortical control of spinal motor systems?

◆ What is the muscle response during the reverse myotatic reflex?

◆ What do group Ia afferents detect?
ANSWERS TO CASE 46: LOWER MOTOR SYSTEM

Summary: An 18-year-old man is involved in a motor vehicle accident with resulting lower extremity paralysis.

◆ **Cortical control of motor neurons:** By the lateral and ventral corticospinal tracts.

◆ **Response during reverse myotatic reflex:** Relaxation of muscle that had been strongly contracting.

◆ **Group Ia afferents detect:** Muscle stretch.

**CLINICAL CORRELATION**

Transections of the central nervous system (CNS) above and within the spinal cord have different effects on motor reflexes. When a transection takes place along the spinal cord, there is loss of voluntary movements and conscious sensation below the level of the transection. The muscles that are innervated below the lesion initially become flaccid and lose reflex responses during spinal shock. Depending on the location of the lesion, various other physical signs may be present, including urinary and/or fecal incontinence, decreased heart rate and pressure, and respiratory failure. Spasticity and hyperreflexia develop later. Lesions of motor pathways above the spinal cord result in muscle rigidity and hyperreflexia without a period of flaccid paralysis. Decerebrate and decorticate posturing may develop with transections above the spinal cord.

**Objectives**

1. Understand the role of motor neurons and motor units.
2. Know about spinal reflexes.
3. Describe the descending control of spinal motor systems.

**Definitions**

- **Motor unit:** A single motor neuron plus all of the muscle fibers that it connects synaptically to.
- **Motor neuron pool:** The complete set of all motor neurons innervating a single muscle.
- **Homonymous muscle:** The same muscle as that containing afferents evoking a reflexive contraction of the muscle (eg, reflexive contraction of the same extensor muscle that is stretched).
- **Muscle tone:** The force with which a muscle resists being lengthened, equivalent to stiffness.
Spasticity: Abnormal increase in muscle tone, often associated with enhanced stretch (deep tendon) reflexes.

Spinal interneurons: Spinal neurons which excite or inhibit other neurons in the spinal cord via axons that do not leave the spinal cord.

DISCUSSION

Alpha motor neurons in the spinal cord have their cell bodies in the ventral horn and send their axons out the ventral roots to innervate axial, flexor, or extensor muscles in the trunk, limbs, and digits. Motor neurons that innervate muscles in the head and neck have their cell bodies in motor nuclei in the brainstem and send their axons out the cranial nerves. All the muscle fibers innervated by a single motor neuron constitute a motor unit. Motor units vary in size from two or three fibers in the fingers, where fine control is important, to more than a thousand fibers in antigravity muscles of the leg. Each action potential in an alpha motor neuron evokes a single twitch in each fiber in the motor unit. Large sustained contractions require the temporal summation of trains of high-frequency twitches. Small motor units are recruited more readily than are large motor units because the size of the motor neuron soma is proportional to the size of the motor unit, and smaller somata are more sensitive to their synaptic inputs than are larger somata (size principle). Motor neurons that innervate fast-twitch fibers fire at higher frequencies than do motor neurons that innervate slow-twitch fibers.

Muscle length and tension are monitored by proprioceptive sensory (afferent) neurons, whose cell bodies are in dorsal root ganglia and which have peripheral terminals in specialized sensory structures within the muscle. Muscle length properties are sensed by terminals that coil around thin intrafusal fibers in muscle spindles. These muscle fibers, which regulate sensory sensitivity, lie in parallel with the thicker extrafusal skeletal muscle fibers that do the effective contractile work. Group Ia afferents in the spindle detect both the absolute muscle length and the rate of change in length of the intrafusal fiber. Group II afferents monitor static length of the intrafusal fiber. Muscle tension properties are detected in the Golgi tendon organs, which lie in series with the muscle fibers and their tendons. Group Ib afferents terminate in these organs and monitor the force exerted by extrafusal muscle contraction or passive stretch. By exciting interneurons that inhibit the same or homonymous (synergistic) alpha motor neurons, the Ib afferents trigger the reverse (or inverse) myotatic reflex, which protects the muscle from excessive contraction. In contrast, the stretch (also known as the deep tendon or myotatic) reflex is initiated when Ia afferents detect stretch of the intrafusal fibers. These afferents make monosynaptic excitatory synaptic connections to alpha motor neurons, innervating the same or homonymous muscle. The Ia afferent pathway drives a homeostatic system in which imposed stretch automatically elicits a compensatory contraction—an arrangement that contributes to balance, posture, and muscle tone. The sensitivity of this system is maintained
during active contraction of surrounding extrafusal fibers by the coactivation of a second type of motor neuron along with the alpha motor neurons. These **gamma motor neurons** excite the intrafusal muscles and prevent them from becoming flaccid when the parallel extrafusal muscles contract. The activity of the gamma motor neurons, and hence the sensitivity of the stretch reflex, can be adjusted by several descending pathways from the brain.

Numerous motor patterns and reflexes are mediated by spinal circuits without the need for input from higher centers. In addition to the stretch and reverse myotatic reflexes, an important reflex is the **withdrawal (flexor) reflex** of a limb triggered by the activation of somatic nociceptor terminals in the limb. These sensory neurons activate excitatory interneurons which in turn activate flexor motor neurons that innervate the ipsilateral limb. In addition, the nociceptors activate inhibitory interneurons that produce **reciprocal inhibition** of the extensor motor neurons which innervate the same limb. Interneurons also activate a second reflex, the **crossed-extensor reflex**, which extends the contralateral leg to provide balance and support during leg flexion. During defensive arm flexion, the crossed-extensor reflex causes a protective extension of the contralateral arm. Another reflex is the **scratch reflex**, which involves repetitive contraction and relaxation of muscles in the arm and hand that are generated by interneuronal circuits within the spinal cord. Groups of interneurons within the spinal cord also generate the complex patterns of motor output responsible for **locomotion**.

The circuits underlying these reflexes and motor patterns are used as building blocks for more complex behaviors that are controlled hierarchically by higher brain structures. Descending input to spinal motor circuits comes by several pathways. One is the **lateral corticospinal tract (pyramidal tract)**, which carries commands for conscious, voluntary movements from upper motor neurons in the primary motor cortex to lower motor neurons in the spinal cord that control distal muscles. Similarly, **lower motor neurons that control proximal muscles** receive commands through the **ventral corticospinal tract**. Upper motor neurons that control muscles in the face and head send their axons into another pyramidal tract, the **corticobulbar tract**, to synapse with lower motor neurons in brainstem motor nuclei. A second major pathway to the spinal cord is the **ventromedial pathway**, which consists of four tracts from brainstem regions that are involved in posture and locomotion: the vestibulospinal, tectospinal, pontine reticulospinal, and medullary reticulospinal tracts. Direct and indirect input to spinal motor neurons also comes from the cerebellum and basal ganglia.

Although **upper motor neurons excite lower motor neurons**, as well as interneurons, much of the descending input to spinal motor neurons is **inhibitory**. Damage to descending pathways at any level above a specific spinal segment (lumped together as upper motor neuron lesions) removes these excitatory and inhibitory influences and thus produces profound effects on reflexes mediated by that segment. These effects include some **weakness** (because of decreased excitation of alpha motor neurons and some interneurons), **increased**
muscle tone (rigidity), and prominent spasticity, which is characterized by hyperactive stretch reflexes and increased resistance to rapid muscle stretch. Mechanisms of spasticity are unclear but appear to involve disinhibition of spinal neurons, including interneurons and gamma motor neurons. Transection of the spinal cord causes a period of spinal shock for a few weeks in which all spinal reflexes below the transection are reduced severely or abolished (flaccid paralysis). This probably results from sudden interruption of descending facilitatory influences. Reflexes then gradually recover and eventually become exaggerated, perhaps because of disinhibition, denervation supersensitivity of motor neurons and interneurons, and/or sprouting of additional afferent terminals. Spinal transection also interrupts autonomic pathways, and this can result in urinary and/or fecal incontinence, increased or decreased heart rate and blood pressure, and respiratory failure. Lesions of peripheral nerves that transect motor neuron axons cause flaccid paralysis and a total loss of voluntary and reflex responses from the denervated muscles, spontaneous twitch-like fasciculations, and muscle atrophy. If the entire nerve is not transected, peripheral axons sometimes can regenerate so that many months after nerve injury the reflexes recover partially or (rarely) completely.

COMPREHENSION QUESTIONS

[46.1] A motor unit that innervates only three muscle fibers is likely to innervate muscle in which of the following?
A. Back
B. Biceps
C. Bladder
D. Thigh
E. Thumb

[46.2] Which of the following effects is caused by activation of gamma motor neurons during active contraction of extrafusal muscle fibers?
A. Decreased magnitude of the stretch reflex
B. Increased force developed by the extrafusal muscle fibers
C. Increased sensitivity of Ib afferents
D. Increased summation of motor units
E. Maintained sensitivity of the Ia afferents during unexpected stretch

[46.3] Which of the following observations would suggest that an upper motor neuron lesion rather than a lower motor neuron lesion is present?
A. Fasciculations
B. Hyporeflexia
C. Profound weakness
D. Pronounced atrophy
E. Spasticity
Answers

[46.1]  **E.** Motor units are smallest in the parts of the body that have the most precise motor control, such as the thumb, fingers, and tongue.

[46.2]  **E.** Coactivation of gamma motor neurons with alpha motor neurons shortens the intrafusal muscle fibers during contraction of extrafusal fibers so that sensitivity of the Ia stretch receptors in the intrafusal fibers is maintained when unexpected stretch occurs during the extrafusal muscle contraction. This would increase rather than decrease the magnitude of any stretch reflex evoked during contraction (answer A) and would have no direct effect on extrafusal force development, the sensitivity of Ib afferents, or the summation of motor units (answers B, C, and D).

[46.3]  **E.** Upper motor neuron lesions produce spasticity, involving hyperactive stretch reflexes. Lower motor neuron lesions do not produce spasticity. Fasciculations are not induced by upper motor neuron lesions, and compared with lower motor neuron lesions, there is less dramatic weakness and muscle atrophy.
PHYSIOLOGY PEARLS

❖ Motor units that control fine, precise contractions by distal muscle are much smaller than motor units that control posture or massive contractions by proximal muscle.

❖ Group Ia afferents innervate muscle spindles organized in parallel with extrafusal muscle fibers that, when activated by increased stretch of the muscle, directly excite alpha motor neurons which cause contraction of the same and homonymous muscle fibers, thereby evoking the stretch reflex.

❖ Descending influences can adjust the sensitivity of the stretch reflex by adjusting the background activity of gamma motor neurons to produce contraction of intrafusal muscle and maintain relatively constant tension on Ia afferent terminals in the spindle during extrafusal muscle contractions.

❖ Local circuits composed of spinal afferents, spinal interneurons, and spinal motor neurons mediate a large number of behaviors that can operate relatively independently of the supraspinal circuits. These behaviors include the stretch, reverse myotatic, flexor, crossed-extensor, and scratch reflexes, as well as major components of locomotor patterns.

❖ Conscious, voluntary movements depend on commands to lower motor neurons in the spinal cord and/or brainstem from upper motor neurons in the primary motor cortex that are conveyed by axons in the lateral corticospinal, ventral corticospinal, and corticobulbar tracts.

❖ Descending influences on spinal motor activity related to posture, balance, orientation, and general muscle tone are conveyed from nuclei in the brainstem by axons in the vestibulospinal, tectospinal, pontine reticulospinal, and medullary reticulospinal tracts.

❖ Upper motor neuron lesions are associated with some weakness, increased basal muscle tone, and prominent spasticity and hyperreflexia, whereas lower motor neuron lesions are associated with profound weakness, decreased muscle tone, hyporeflexia, muscle fasciculations, and muscle atrophy.

REFERENCES

