

The Motor System: Lecture 1

Spinal motor system

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NOTE: All slides and lecture notes for the motor lectures of Dr. Shadmehr are available at:
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OBJECTIVES: To provide an overview of the function of the main neural structures that contribute to control of action; to focus on the spinal motor system and its anatomy and physiology; introduce the state of art of neural prosthetics; and describe neural mechanisms of proprioception and feedback control of action.

The human motor system produces action. It controls goal-directed movement by selecting the targets of action, generating a motor plan, and coordinating the generation of forces needed to achieve those objectives. Genes encode a great deal of the information required by the motor system — especially for actions involving locomotion, orientation, exploration, ingestion, defense, aggression, and reproduction — but every individual must learn and remember a great deal of motor information during his or her lifetime. Some of that information rises to conscious awareness, but much of it does not. Here we will focus on the motor system of humans, drawing on information from other primates and other mammals, as necessary.

Major components of the motor system

Slide 1. The central nervous system that vertebrates evolved comprises six major components: the spinal cord, medulla, pons, midbrain, diencephalon and telencephalon, the last five of which compose the brain. In a different grouping, the hindbrain (medulla and pons), the midbrain, and the forebrain (telencephalon plus diencephalon) constitute the brain. Taken together, the midbrain and hindbrain make up the brainstem. All levels of the central nervous system participate in motor control. However, let us take the simple act of reaching to pick up a cup of coffee to illustrate the function of the various components of the motor system.

Parietal cortex: processes visual information and proprioceptive information to compute location of the cup with respect to the hand. Sends this information to the motor cortex

Motor cortex: using the information regarding the location of the cup with respect to the hand, it computes forces that are necessary to move the arm. This computation results in commands that are sent to the brainstem and the spinal cord.

Brainstem motor centers: sends commands to the spinal cord that will maintain the body's balance during the reaching movement.

Spinal cord: Motor neurons send the commands received from the motor cortex and the brainstem to the muscles. During the movement, sensory information from the limb is acquired and transmitted back to the cortex. Reflex pathways ensure stability of the limb.

Cerebellum: This center is important for coordination of multi-joint movements, learning of movements, and maintenance of postural stability.

Basal ganglia: This center is important for learning of movements, stability of movements, initiation of movements, emotional, and motivational aspects of movements.

Spinal cord is comprised of segments, each controlling a group of muscles

Slide 2. The spinal cord comprises four major divisions. From rostral to caudal, these are called: cervical, thoracic, lumbar, and sacral. Cervix is the Latin word for neck. The cervical spinal segments intervene between the pectoral (or shoulder) girdle and the skull. Thorax means chest (or breast plate). Lumbar refers to the loins. Sacral, the most intriguing name of all, refers to some sort of sacred bone.

In mammals, the cervical spinal cord has eight segments; the thoracic spinal cord has 12; and the lumbar and sacral cord both have five. The parts of the spinal cord that receive inputs from and control the muscles of the arms (more generally, forelimbs) and legs (more generally, hindlimbs) show enlargements associated with an increasing number and size of neurons and fibers: the cervical enlargement for the arms and the lumbar enlargement for the legs. Each segment is labeled and numbered according to its order, from rostral to caudal, within each general region of spinal cord. Thus, the first cervical segment is abbreviated C1 and together the eight cervical segments can be designated as C1–C8.

Slide 3. In each spinal segment, you find a ring of white matter surrounding a central core of gray matter. White matter is so called because the high concentration of myelin in the fiber pathways gives them a lighter, shiny appearance relative to regions with many cell bodies. The spinal gray matter bulges at the dorsal and ventral surfaces to form the dorsal horn and ventral horn, respectively.

The cord has two major systems of neurons: descending and ascending. In the descending group, the neurons control both smooth muscles of the internal organs and the striated muscles attached to our bones. The descending pathway begins in the brain, which sends electrical signals to specific segments in the cord. Motor neurons in those segments then convey the impulses towards their destinations outside the cord.

The ascending group is the sensory pathways, sending sensory signals received from the limbs and muscles and our organs to specific segments of the cord and to the brain. These signals originate with specialized transducer cells, such as cells in the skin that detect pressure.

The cell bodies of the neurons are in a gray, butterfly shaped region of the cord (gray matter). The ascending and descending axon fibers travel in a surrounding area known as the white matter. It's called white matter because the axons are wrapped in myelin, a white insulating material.

Both the gray matter and the white matter house glial cells. These cells help the neurons survive and work effectively. The glial cells support the myelin that wraps around the axons. The myelin insulates the axons and allows for fast transmission of electrical signals with little or no loss in signal strength.

Spinal cord injury results in paralysis

Slide 4. If a fall or impact dislocates the spinal column, the vertebral bones can crush the spinal cord. The damaged might be limited to the gray matter in which case the fiber pathways running past the damaged segment, in the spinal cord's white matter, would remain intact. In this case, the muscles controlled by the motor neurons in this segment of the cord will be paralyzed, but others would not. For instance, if only the gray matter at the C8 was affected, the hands would be paralyzed but walking would remain normal. While no signals would go to, or be received from, the muscles connected to C8, the axons conveying signals up and down the surrounding white matter would keep working.

In contrast, if the spinal dislocation has severely damages the white matter as well, then the communication pathways from the brain to the cord levels below the level of the injury would be lost. In the case of the C8 level injury, the person would become paralyzed in the hands and lower limbs, and would lose control over the lower body.

After spinal cord injury, a chain of events dramatically exacerbates the initial loss of function. While the initial injury is likely to damage a small region of the spinal cord, in the next few hours the area of damage grows, invading neighboring segments of gray matter and eventually ballooning and destroying the white matter as well. This second wave starts with hemorrhaging from broken blood vessels in the gray matter. The result is a fluid filled region, or a cyst, that locally swells the spinal cord. The swelling puts pressure on healthy axons and other vessels, preventing delivery of oxygen and nutrients, and causing many of them to starve to death.

Meanwhile, the injured neurons release a flood of glutamate. Normally, the same cells release minute amounts of this neurotransmitter to excite other cells, but in this injury-related process, the large amounts of glutamate released overexcite the neighboring neurons and lead to their death. The term necrosis refers to the cell loss due to this mechanism, which is termed excitotoxicity.

The same high level of glutamate is also lethal to myelin-producing glia cells that support the axons that travel near the damaged region. The myelin insulates the axons and allows them to conduct action potentials between neighboring segments at a faster rate than is possible without myelin. The death of the glia cells results in the demyelination of healthy axons, making them unable to conduct impulses in a normal way. These axons are now useless for all practical purposes because even if they conduct action potentials, the timing and sequence of signal is highly abnormal. A few days after the initial event, myelin producing glia cells in spinal segments three or four segments away begin to die. The reason for this "mass suicide", or apoptosis, is currently poorly understood. A few weeks later, a wall of glia cells forms around the cyst, inhibiting regrowth of the severed axons.

Progress is being made in a number of fronts to counter the events that follow spinal cord injury. Research (currently mostly in rats) has identified a drug that is effective in limiting swelling and inflammation in the spinal cord. Other drugs have been found to inhibit release of glutamate, while still others inhibit its uptake. Apoptosis-inhibiting agents have been identified and have shown promise in rats.

Neural prosthetics electrically stimulate muscles to overcome paralysis

Slide 5. The most common type of spinal cord injury in humans is at the level of C5–C6. Damage to these segments of the cord leaves the individual with paralyzed finger muscles as well as an inability to activate the elbow extensor muscle, named triceps. This is because the motor neurons that serve some shoulder joint muscles (e.g.,

deltoid) and some elbow joint flexors (biceps, brachialis, and brachioradialis) reside between C4 and C6, while motor neurons serving the elbow extensor muscles (e.g., triceps) and all finger muscles reside at C6 and lower. Therefore, the motor neurons are not able to send impulses to the elbow extensor and finger muscles. However, the muscles themselves are still capable of contracting if there was some other way for the central nervous system to activate them.

Slide 6. A recent approach tries to get around this problem by implanting a stimulator on the muscles that are paralyzed and a sensor on muscles that are functioning normally. For example, to control the finger muscles of the right hand, a motion sensor is implanted on the left shoulder and stimulating electrodes are implanted on the muscles of the right hand. Because of the level of damage to the spinal cord, some control of the shoulder muscles is spared. For example, the patient can elevate his left shoulder. This movement is transduced by the sensor and sent to a control unit. The control unit stimulates the flexor muscles of the paralyzed hand, allowing the patient to close his or her hand. With this device, the patient can learn to make finger to thumb grasps and, for example, lift a glass of water. Electronics in the control unit provide further flexibility of control to the patient. For example, if the patient rapidly elevates the shoulder twice, the controller can “lock” the hand around the glass without having to maintain shoulder elevation.

Muscles are composed of fibers; each fiber is innervated by a single motor neuron's axon

Slide 8. Skeletal muscles develop from cells (technically, myoblasts) that fuse to form fibers 10–100 μm in diameter, extending on the order of a meter in length in large animals. Each muscle fiber is composed of bundles of *myofibrils* that house a collection of *filaments*, which in turn consist of *sarcomeres*. It is within these sarcomeres that muscle proteins *actin* and *myosin* form strands, and these strands provide the motile force of the muscle.

Each muscle fiber is innervated by an axon from a motor neuron. Action potentials in the motor neuron arrive at the synapse to the muscle fiber (called the neuromuscular junction) and release acetylcholine, resulting in the depolarization of the muscle fiber. This results in the release of calcium ions from the sarcoplasmic reticulum, exposing the myosin-attachment site; myosin attaches to actin, and they slide relative to each other to shorten the muscle and generate force. When depolarization ends, Ca is pumped back into the sarcoplasmic reticulum and relaxation occurs.

Slide 9. The motor neurons reside in the ventral portion of the gray matter in the spinal cord (called the ventral horn). Motor neurons that innervate a muscle are situated close to each other in the ventral horn. For example, motor neurons that innervate the biceps are all close to each other in the spinal cord. They collect into a cigar shaped region that stretches two to four spinal segments.

Slide 10. MNs innervating a single muscle are called a *motor pool*. In the ventral horn, there are two groups of motor pools. In the medial part of the ventral horn, the motor pools innervate axial muscles (e.g., neck and back). In other words, these MNs control muscles that help us stand up and maintain posture. In the lateral part of the ventral horn, motor pools innervate limb muscles (distal muscles).

Slide 11. The motor unit: Motor unit refers to a single motor neuron and the muscle fibers it controls. Each mature muscle fiber is innervated by only one motor neuron, although each motor neuron innervates many muscle fibers. The muscle fibers that are controlled by a single MN are not usually adjacent.

The number of fibers in a motor unit varies according to function. Muscles that contribute to fine movements, like those acting on the eye or the fingers, usually have a small number of muscle fibers per motor unit. Example: a motor unit in the ocular muscles (of the eye) may have a membership of 3 to 6 muscle fibers. In the gastrocnemius, there are thousands of muscle fibers per motor unit. Large motor neurons have many branches and have a large motor unit.

Slide 12. Polio and post-polio syndrome. The poliovirus invades the motor neurons, leaving intact adjacent nerve cells. Recently, poliovirus receptors at the neuromuscular junction have been identified. These receptors allow the virus to enter the motor axon and then to migrate to the motor neuron's cell body. The infected cell either overcomes the virus or dies. If it dies, the muscle cell is paralyzed. The motor neurons that survive develop new terminal axon sprouts that reinnervate orphaned muscle cells. A single motor neuron may grow sprouts to innervate up to 10 times more muscle cells that it did originally, creating a giant motor unit. Therefore, total quantity of muscle fibers that belong to a single motor unit increases, though the number of motor units decrease. Consequently, the twitch tension and action potentials recorded by stimulating a single motor unit are far larger than normal. The system is not static however. In a process called remodeling, the motor unit is constantly both losing old sprouts and growing new ones. At 30 or 40 years after onset of the disease, the enlarged motor units begin to break down, causing muscle weakness. Current hypothesis is that this is due to an overuse of the few remaining motor neurons during the previous decades, i.e., a kind of metabolic exhaustion that leads to an inability to regenerate new axon sprouts to replace degenerated ones.

Amyotrophic Lateral Sclerosis. In ALS, also called Lou Gehrig's disease, there is slow degeneration of alpha-motor neurons in the spinal cord, and eventually neurons in the motor cortex. Individuals show progressive weakness and die within 5 years of onset. There is currently no effective therapy.

Motor stroke: In this condition, there is no denervation of muscles because the spinal motor neurons remain intact and the damage is to the neurons in the brain. Consequently, it is difficult for the patient to recruit all available motor units in a voluntary contraction, resulting in weakness. In a stroke, motor unit discharge is often abnormally low.

Extrafusal muscle fibers are innervated by α motor neurons

Slide 13. There are two types of muscle fibers: *extrafusal fibers*, which attach to tendons, which in turn attach to the skeleton, and *intrafusal fibers*, which attach to the extrafusal fibers. Extrafusal fibers produce the force that acts on bones and other structures. Intrafusal fibers also produce force, but they are much smaller than extrafusal fibers and the level of force that they produce is negligible in comparison. Instead, intrafusal fibers play a sensory role. They contain muscle spindles which, innervated by *muscle spindle afferents*, provide the central nervous system with information about muscle length.

There are two major types of motor neurons. α motor neurons innervate large, extrafusal muscle fibers that generate force. γ motor neurons innervate intrafusal muscle fibers that house the muscle's sensory system.

Force of contraction depends on rate of stimulation

Slide 14. In response to a single impulse to the muscle, the muscle produces a force which is called a *twitch*. Whereas the impulse might be 1 to 3 msec in duration, the twitch is 10 to 100 msec. When the rate of impulses is low, the twitches have time to relax. When the rate of stimulation is high, the twitches fuse and the force in the muscle sums, until you reach a state where you can't distinguish individual twitches. This is called *tetanus*.

Rigor mortis. Muscles activation increases muscle stiffness, but so does death, which leads to *rigor mortis*. Death causes depolarization of the muscle fibers because the eventual depletion of ATP stops the sodium-potassium pump upon which normal cell polarization depends and the hydrolysis of ATP by myosin, upon which detachment from actin depends. In death as in action, attachment of myosin to actin causes stiffening of the muscle fibers.

Force of contraction depends on muscle length

Slide 15. Like a spring, muscles produce a restoring force when they are stretched beyond their "resting length". This is called the passive force. When the muscle is stimulated, the contractile elements shorten and the muscle starts producing force at a much shorter length. This force depends on the length of the muscle: there is a region of muscle length for which the active force produced is maximum. This is because it is at this length for which there is maximum overlap between the thick and thin filaments. When the sarcomere is stretched too much or relaxes too much, there is insufficient overlap and active force drops off.

To rapidly move a limb, antagonistic muscles are activated in sequence

Slide 16. Muscles can only pull, they cannot push. They are organized about a joint in antagonistic pairs. In order to move a limb, the agonist muscle (the muscle that will be shortened) needs to be activated. In order to move quickly, the agonist muscle needs to be strongly activated. This will accelerate the limb. To quickly decelerate a movement, the antagonist muscle will need to be activated. Finally, to maintain the limb at the final position, the agonist muscle needs to be activated again. This 3-burst pattern of activity (agonist-antagonist-agonist) is often observed when fast movements take place.

Slide 17. The CNS uses this three-burst pattern of activity for making rapid movements. This figure shows an example of a rapid wrist flexion.

Slide 18. When there is damage to the brain, this simple pattern of three-burst activity is sometimes disturbed. A prominent example is in a condition called essential tremor. This condition is thought to be associated with damage to the cerebellum. Movements exhibit an oscillatory pattern. For example, in this figure data from a rapid wrist flexion is displayed. In both the normal individual and the patient, this rapid movement produces a slight overshoot. The wrist comes back toward the target mainly because of the activity in the wrist extensor, i.e., the antagonist burst. However, while in the normal individual, the movement is terminated with a second agonist burst that partially overlaps with the antagonist burst, in the patient the second agonist burst is delayed so that the action of the antagonist is essentially unopposed. This makes the wrist to significantly undershoot the target. The pattern

is repeated, with the result being an oscillation at the target. The extent of delay in the second agonist burst is correlated with the frequency of tremor.

Muscle fibers have varying contraction and fatigue properties

Slide 19. The exact content of the myosin molecule determines the functional characteristics of the muscle fiber. In adults, we see three varieties of the myosin molecule. These isoforms are designated as type I, IIa, and IIx. Type I fibers are known as slow fibers, while type II are fast fibers. The fibers are called slow and fast because of their rate of force production in response to a single action potential. When they are electrically stimulated, Type I fibers contract at about a tenth the speed of type IIx fibers (type IIa are somewhere in between).

Slide 20. Recall that a motor unit is made up of a single motor neuron and a collection of muscle fibers. There are 3 basic types of motor units: we categorize them based on their twitch contraction time and fatigability.

1) Fast fatiguing (FF). These motor units have a small contraction time and produce a high twitch tension, but tend to fade fast. FF motor units are composed of muscle fibers with relatively large diameter and are supplied by fast conducting, large diameter axons from large motor neurons.

2) Fast, fatigue resistant (FR). These motor units have an intermediate contraction time and can maintain force longer.

3) Slow non-fatiguing (S). These motor units have a long contraction time and show little or no loss of force with repeated stimulation. S type motor units are composed of muscle fibers with small diameter, and are supplied by slow conducting, small diameter axons from small motor neurons.

Different muscles have different proportions of motor units: for example, the diaphragm muscle is composed almost entirely of S type motor units, while gastrocnemius, which is sometimes used for jumping, has a larger proportion of FR and FF units.

Muscle fibers change based on their history of activation

Slide 21. A muscle fiber cannot split to form a new fiber. So a muscle can become bulkier only if the individual fibers become thicker. This happens by addition of new myofibrils. The process starts when there is additional stress at the tendons, for example, when one lifts weights. This triggers signaling proteins to activate genes that cause muscle fibers to make more contractile proteins (chiefly myosin). But activity (or lack of) can also change a muscle fiber's type.

A leg muscle like quadriceps in the thigh in a typical person is made up of about equal number of type I and type II fibers. If a person suffers a spinal cord injury and is paralyzed in the lower limbs, the lack of use results in loss of tissue, as is expected, but also a surprising change in the muscle types. The paralyzed individual experiences a sharp decrease of the relative amount of slow myosin, whereas the amount of fast myosin increases. Ten years after paralysis, an individual may have little or no slow type muscles in their quadriceps. The neural input from the spinal cord may be essential for expression of the slow type myosin isoform. Without it, the myosin's default state is the fast type. This might account for the different ratio of slow to fast fibers in marathon runners and sprinter.

There is change in muscle simply due to aging. By age 50, 10% of muscle mass is lost. At 80, loss is at 50%. Motor neurons can also die. Motor neuron death is particularly prevalent among large motor neurons that supply the type II fibers. In an elderly individual, proportion of fibers tends to be more of the slow type.

Grading of muscle force is accomplished through recruitment and stimulation frequency

Slide 22.

1. During voluntary contraction, as force requirements increase, new motor neurons are recruited.
2. As force requirements increase, the frequency of activation of already recruited motor neurons increases.

Slide 23. Motor units that are recruited early tend to have slow contraction time and produce small peak force. Motor units that are recruited later tend to have faster contraction time and larger peak force. The motor neurons that are recruited early will control slow (type I) muscle fibers. The motor neurons that are recruited late will innervate fast (type II) muscle fibers.

Slide 24. We noted earlier that the distribution of muscle fibers in a leg muscle depends on its history of use. Extensive use of a limb can skew distribution of fibers in a muscle toward the slow type. During voluntary contraction of a muscle that has a large proportion of slow fibers, a large proportion of motor units will be recruited. In this experiment, the authors tested this idea by recording the activity from the first interosseus (FDI) muscle of the dominant and non-dominant hands of a few healthy volunteers as they produced a voluntary contraction. In each volunteer, they randomly sampled from the motor units of this muscle and plotted the percentage of motor units that came on when the muscle was producing small vs. large amounts of force. They found that in the dominant hand, a

larger proportion of motor units were recruited to produce a small amount of force than in the non-dominant hand. This is consistent with the idea that because the dominant hand is used more, it tends to have a larger proportion of slow fibers, and therefore a larger proportion of motor units that are recruited early during a voluntary contraction.

Muscle receptors measure length and force of the muscle

Slide 25. Based on the diameter of the afferent fiber, muscle receptors are divided into groups I and II. Group I has the larger fiber diameter. Groups Ia and II wrap themselves around interfusal muscle fibers. They are sensitive to length changes. Group Ib afferents innervate the junction between extrafusal muscle fibers and the tendon. They are sensitive to force changes in the muscle.

Muscle spindles afferents measure length, Golgi tendon afferents measure force

Slide 26. Spindle is a term used to describe a group of fine intrafusal muscle fibers that are tapered at the end and have a fluid-filled capsule at the center. Within the capsule the muscular elements are entwined with afferent fibers (from the group Ia and II afferents). Intrafusal fibers are innervated by gamma motor neurons. Spindle afferents respond to changes in muscle length.

The tendon area is innervated by a different kind of afferents, called the Ib afferents. These afferents are also called the Golgi tendon afferents. They respond to changes in muscle force.

The gamma motor neuron system controls spindle sensitivity. When the extrafusal muscle fibers shorten due to activation from a motor neuron, the intrafusal fibers can go slack. This would lead to a sudden loss of firing in the spindle afferents, resulting in a loss of information regarding muscle length. To prevent this, the CNS activates the gamma motor neurons during contraction to maintain the tension in the intrafusal fibers.

Our sense of limb position is mainly via muscle spindles

Slide 27. Because muscle spindles are sensitive to muscle length, it is reasonable that the brain would rely on their discharge to estimate where our limbs are, that is, proprioception. To investigate this, a subject was seated with her elbows on a table and had eyes closed. The subject was instructed to try to match the angle of the right elbow with the left arm. To investigate how the subject is estimating the position of the right elbow, a mechanical vibrator (100 Hz) was positioned immediately over the tendons at a point just above the right elbow. This vibration strongly stimulates the spindle afferents. During the stimulation, the subject moved her left arm (the tracking arm) to a more extended position. As the stimulation stopped, the tracking arm was quickly flexed back to its original position. This suggests that the brain perceived the stimulation as an extension of the biceps on the right arm, and therefore extended the left elbow to match its perceived position.

Spindle afferents excite motor neurons of the same muscle

Slide 28. Ia fibers, coming from the spindles, terminate in the part of the spinal cord where the motor neurons are (ventral horn). They have monosynaptic excitatory connections on alpha motor neurons of the same muscle. A single Ia afferent sends afferents to nearly all of the MNs in the same muscle. They make excitatory synaptic input to inhibitory interneurons acting on alpha motor neurons of antagonist muscles.

Golgi tendon afferents inhibit motor neurons of the same muscle

Slide 29. Group Ib fibers, coming from the Golgi tendon organs, terminate in the regions of the spinal cord where interneurons are located. These interneurons inhibit the alpha motor neurons that go to the same muscle.

The stretch reflex can be tested by suddenly extending a muscle

Slide 31. The sudden stretching of a muscle, for example through tapping of the tendon with a hammer, results in lengthening of the muscle spindle afferents. This produces action potentials in the afferents. The afferent neuron makes synapses on the motor neurons of the stretched muscle, exciting these motor neurons and contracting the muscle.

Stretch reflex has both a short latency and a long-latency component

Slide 32. When the limb is suddenly perturbed, the spinal reflexes respond. This response cannot be voluntarily controlled. However, a later response is also observed which can be voluntarily controlled. This long-latency response is believed to have a cortical origin.

The short-loop: mono-synaptic activation of motorneurons by the Ia afferents. When you stretch a muscle, spindles afferent neurons increase their activity, resulting in excitatory input onto the motorneurons, and activation of the muscle that was elongated.

Slide 33. The long-loop: In humans, particularly for the muscles of the arm, wrist and fingers, there is a second pathway through which a stretch can result in a compensating activity in the motoneurons. This pathway begins with the Ia afferents, goes up the spinal cord through the dorsal column-medial lemniscal pathway, reaches the thalamus, then the somatosensory and motor cortex, and then comes back down to the spinal cord through the cortico-spinal tract. The latency of this loop is about 70 ms. However, unlike the short-loop, its activity is programmable by the brain: voluntarily we can change the response to a stretch (if the stretch is expected, we can ignore it, or respond vigorously).

There are long delays associated with transfer of information from the spinal cord to the cortex and back

Slide 34. In this slide, we have the results of an experiment where the ankle of a healthy volunteer was suddenly stretched. The experimenter is recording with a small surface electrode from the scalp, about the region of the cortex where somatosensory cortex lies. In response to the stretch, an “evoked” potential at the scalp is recorded. This potential reflects the activity of neurons in the somatosensory cortex. Notice that the delay from the stretch to the onset of the evoked potential is about 45ms. Next, the experimenter stimulates the scalp around the region of the motor cortex. This activates neurons in the motor cortex, activates the corticospinal tract, and then activates the motoneurons in the spinal cord. The result is muscle contraction, which is picked up with EMG electrodes. The delay from activation of the motor cortex to the activation of muscles is about 30ms. Therefore, the fastest possible loop time from the spinal cord to the cortex and back is about 95ms. Now on the bottom row of this figure, you see the EMG on the ankle muscle when it is suddenly stretched. Notice that there are three peaks. Only the last peak can be due to activity in the spinal-cortex-spinal loop (long loop). The first two peaks are due to pathways within the spinal cord or brainstem.

A stroke in the brainstem can disrupt the long-latency stretch reflex on the ipsilateral arm

Slide 35. The thumb flexor in a patient with a lesion in the right brain-stem (due to stroke) is suddenly stretched. In this patient, the stroke has resulted in a complete loss of proprioception (sense of limb position) from the right arm and hand. In response to the stretch at time 0, we see the EMG record from the left (middle trace) and right (bottom trace) hand. The EMG response at 25 msec is due to the short loop stretch reflex. The response at 40-55 msec is due to the long-loop stretch reflex. Note that in the right hand, the short-loop EMG response is present, but the long-loop response is missing.

Movements can take place even without sensory afferents

The major role for our reflexes is to respond when there is an unexpected perturbation. They tend to change the activations of motor neurons so to stabilize the limb. However, to what extent are these reflexes important for generation of voluntary movements where there are no perturbations? To answer this question, we can look at patients who have a neurodegenerative disease the specifically destroys the spindle and Golgi tendon organ afferents.

Large fiber sensory neuropathy: In large fiber sensory neuropathy, the large afferent fibers degenerate (Ia and Ib fibers). Patients can't sense the position of their limb, nor can they detect its motion. They need to see their limb in order to be able to make accurate movements. They feel pain and temperature on the limbs because these afferent fibers are not affected

Slide 36. Here we have the records for a rapid thumb flexion in a normal individual and in a patient with large fiber sensory neuropathy affecting his arm and hand. We see the 3-phase EMG pattern in the normal subject, as well as in the patient. Therefore, the origin of the EMG pattern is likely through direct descending commands to the alpha-motor neurons and not through a feedback loop.

Slide 37. However, the brain still needs sensory information from the hand in order to generate the appropriate pattern of muscle activity. We need to know where the hand is before we can accurately move it. In the case of the neuropathy patient, he needs to see where the hand is. Now if we were to cover the hand after the flexion of the thumb has been made, the finger drifts.