The Motor System: Lecture 2
Descending Tracts

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OBJECTIVES: To consider the function of the corticospinal tract and how each cerebral hemisphere contributes to control of action; and to introduce the contribution of brainstem descending motor tracts to control of action.

Consider how you write your signature. Everyone has their unique signature. This uniqueness retains its character even if undertaken by different joints and muscles than the fingers: you may never have tried to write your signature with a pencil held in your mouth, but if you do, chances are that it will share essential characteristics of your hand written signature. This tells you that voluntary movements are represented at the highest levels not as a pattern of muscle activations, but as a kinematic pattern, i.e., a desired motion for the ``end-effector", in this case, the pencil, whether held in our mouth or by our fingers. The primary problem for motor control is to translate a desired motion to a pattern of muscle activations.

Refer to Slide 1. Handwriting retains its character even if undertaken with major use of three different joints: (A) the normal condition, using fingers and wrist; (B) using the elbow as fulcrum; (C) using the shoulder joint as fulcrum when writing with the outstretched and unsupported arm.

Refer to Slide 2. Consider how we perform a reaching movement. Our hand tends to follow a straight line, despite the fact the joints of our may follow a much more complicated path. For example, in this example a volunteer hold a lever in hand and moves it from target to target. Look at the movement from T2 to T6. The joint angles for the elbow and shoulder for this movement are shown in the 3rd column. Note how the elbow first flexes, then extends. However, the hand follows an approximately straight line and its speed (third row in part C) is a smooth, “bell” shaped profile. The reach involves a smooth, straight path for the hand, and a complicated path for the joints. This suggests that in our brain, we plan a simple movement for the hand, and then leave it to the motor system to translate that plan for us to activations of the muscles and movements of the joints. In our 3rd lecture we will see that this planning is performed by the posterior parietal cortex. The translation of this plan into motor commands relies on the motor cortex.

Refer to Slide 3. Let us take the example of signing our name. The first task is to motivate us to do this action. This executive function may take place in the prefrontal cortex and involves the basal ganglia. Next, we need to pick up a pen. Perhaps we see the pen on the table. Posterior parietal cortex receives information from the visual cortex and localizes the pen with respect to our body, taking into account eye and head positions. This information is sent to the premotor cortex, where cells represent the position of the pen with respect to our hand. Here, a plan of action to move the hand from its current position to the position of the pen is formed. The primary motor cortex receives this plan from the premotor cortex and begins translating the kinematic plan into activations that are necessary for the muscles. The cerebellum makes a contribution to this translation by compensating for the motions that involve rotations in multiple joints.

Refer to Slide 4. The brain stem motor centers ensure that we don't fall over while we are reaching with our hand (maintaining postural stability). Once the pen is grasped, the somatosensory and visual cortices confirm success of this action, and the plan for signing is translated into muscle activations on the fingers. Descending motor tracts take the commands to the spinal cord and the motor neurons. These tracts originate from the motor regions of the cortex and the brainstem. In the brainstem, tracts originate from the red nucleus, the pontine and medullary reticular formations, and the vestibular nuclei.

The corticospinal tract

Refer to Slide 5. The axons running in the medullary pyramid make up the pyramidal tract. The vast majority of these fibers come from the cerebral cortex and run to the spinal cord. These fibers are known as the corticospinal tract. Other fibers leave the tract in the pyramids and innervate the cranial nerve motor nuclei, which reach the motor neurons that serve the facial muscles. This portion of the fibers composes the corticobulbar tract.

About 30% of the corticospinal and corticobulbar fibers originate from the primary motor cortex. Another 30% originate from the premotor cortex. The rest originate from the primary somatosensory (Brodmann's areas

1,2,3) and parietal cortex (Brodmann's areas 5 and 7). In humans, there are about 1 million fibers in the corticospinal tract, 800k in the chimpanzee, 400k in the macaque, and 180k in the cat.

Refer to Slide 6. Approximately 90% of the fibers cross in the lower medulla (pyramidal decussation) to form the lateral corticospinal tract. This tract projects to sensory neurons in the dorsal horn, interneurons in the intermediate zone, and motor neurons innervating distal limb muscles. Therefore, the cortical motor areas have dominant control over the contralateral limbs.

Most of the fibers that don't cross form the ventral contricospinal tract, which projects onto the ventromedial motor pools innervating axial muscles. This tract originates from the neck and trunk region of the premotor and motor cortex. Unlike the lateral corticospinal tract, this tract has bilateral projections in the cord.

A tiny minority of fibers do not cross and travel laterally in the spinal cord, forming the lateral corticospinal tract. They allow the motor cortex to have a small amount of control over the ipsilateral limb.

The fibers that map to the dorsal horn originate in the somatosensory cortex (areas 1, 2 and 3). The fibers that terminate in the ventral horn originate in the motor cortex and act on alpha and gamma motor neurons of distal muscles (e.g., hand muscle) in primates. They have strong excitatory action. For example, a prominent function of the corticospinal tract in humans is to control individual fingers.

Corticospinal fibers have a small diameter (1-4 microns), meaning that they are slow conducting. They are probably involved in fine control. Their involvement in movement will require less dependence on afferent feedback and more dependence on predictive mechanisms because of their relative slowness in response.

Refer to Slide 7. The strongest connection from the cortex to the muscles is from the primary motor cortex. Stimulation of the primary motor cortex produces an excitatory response primarily in the hand muscles (distal), and less so in the arm muscles (proximal).

Refer to Slide 8. In contrast, stronger currents are needed to stimulate more anterior motor areas of the cortex. In this slide you see the effect of stimulation of the Supplementary Motor Area (SMA), a region anterior to the primary motor cortex. Stimulation here also produces excitatory response in the arm and hand muscles. The responses from SMA are typically 5ms later than seen from the primary motor cortex, and about 15 times less strong.

Study of motor control in split-brain patients

Refer to Slide 9. Study of split-brain patients provides fascinating examples of how each cerebral hemisphere controls movements of the limb. Normally, the cerebral hemispheres communicate and coordinate their actions via the corpus callosum. However, in rare instances patients have this massive structure sectioned to relieve epilepsy.

When you fixate a point with your eyes, you are putting the image of that point on your fovea. The part of the visual scene to the right of fixation (here, a cup) falls on the left hemi-retina, while the part of the visual scene to the left of fixation (here, nothing) falls on the right hemi-retina. The left hemi-retina sends its information to the left visual cortex. The right hemi-retina sends it information to the right visual cortex. In a healthy individual, the visual information in the two hemispheres is shared (via the corpus callosum) so that both visual cortices know about the information to either side of fixation. However, in a split brain patient, the corpus callosum is cut. Therefore, when a split brain patient fixates a point, the image of the cup is only available to the left hemisphere.

Refer to Slide 10. If you verbally ask the patient to use their <u>left hand</u> to pick up the item that they see (the cup), they will have to rely on the left hemisphere to understand what was said (as language centers are in the left side). The left hemisphere in a normal individual would then send this information to the right hemisphere so that the right motor centers would move the left hand. However, in a split brain patient, this is not possible and the left hemisphere will attempt to move the left arm. There are a small number of ipsilateral projections to the arm, and therefore the volunteer is able to reach to the object. However, there are no ipsilateral projections of the corticospinal tract to the fingers or the hand. Therefore, the volunteer will have a hard time making the right posture for grasping with the left hand.

Refer to Slide 11. Because each hemisphere has dominant control over the contralateral arm, the actions performed with one arm may not conform with the actions done with the other arm. Because the language faculty is in the left hemisphere, the split-brain patient might claim that the left arm is an “alien arm” and “doing its own thing”.

Refer to Slide 12. Language is a faculty of the left hemisphere. When a spoon is shown to the right hemisphere, the patient will claim that they see nothing because indeed, the left hemisphere has not seen the spoon. However, when asked to use the left hand to choose among a number of objects, the patient uses the right hemisphere to pick up the spoon.

Refer to Slide 13. The corticospinal tract (CS) is the only descending motor pathway that is accessible for evaluation in humans. This is done with transcranial magnetic stimulation, activating neurons in the motor cortex,

and recording the resulting action potentials of the hand motorneurons along the nerve that supplies the arm/hand muscles. Between the ages of 2-4 the speed of conduction in the CS tract drastically improves (5 times faster). This is because at birth pyramidal tract is incompletely myelinated. Note that the conduction time remains constant from age 4 to adulthood even thought distance between motor cortex and muscles greatly increases.

Descending pathways from the brain stem

The brainstem gives rise to the rubrospinal tract (from the red nucleus), vestibulospinal tract, and pontine and medullary reticulo-spinal tracts.

Refer to Slide 14. Two pathways project onto the spinal cord from the brain stem:

(A) A ventromedial pathway that includes the reticulospinal and vestibulo-spinal tracts. These tracts terminate in the ventromedial part of the spinal cord and influence proximal muscles. They are important for maintaining balance and posture. Pontine reticulospinal tract excites extensor muscles of the lower limb (these muscles help us stand up). Medullary reticulospinal tract inhibits motor neurons at all levels.

(B) The dorsolateral pathway terminates in the dorsolateral part of the spinal cord and influences distal muscles of the limbs. It is important for moving our limbs and fingers. It is primarily composed of the rubrospinal fibers that originate in the red nucleus in the midbrain.

The red nucleus and the rubrospinal tract

Refer to Slide 15. The red nucleus is a pinkish, yellow colored structure in the midbrain. It is cytologically divided into two regions, a magnocellular part (large cells, caudal end), and a parvocellular part (small cells, rostral end). It has larger fibers than the corticospinal tract. The tract decussates near its origin and as it descends in the brain stem, it gives off collaterals to interpositus of cerebellum and vestibular nucleus.

Inputs: The red nucleus receives fibers from the cerebral cortex and the cerebellum. Projections are somatotopically organized. The cortico-rubrospinal system is composed of two parts: One originates from the leg and arm region of area 4 (primary motor cortex) and projects to the magnocellular part of the red nucleus (located in caudal regions of red nucleus), which gives rise to the rubrospinal tract. The second, larger component projects from areas 4 and 6 to the parvocellular part of the red nucleus (rostral regions of RN), which in turn connects to the cerebellum through the inferior olivary nucleus (a center in mid medulla).

Refer to Slide 16. Outputs: Rubrospinal fibers cross completely in ventral tegmental decussation and descend to the spinal cord. In primates, its most significant projections are to the upper limbs, including the shoulder/elbow muscles and the wrist and digit muscles of the hand. Stimulation of the red nucleus tends to excite extensor muscles of the arm and inhibit flexors. Based on this, one would guess that it has a role to play in control of reaching movements.

In man, the magnocellular component of the red nucleus is composed of only about 200 cells, so the rubrospinal tract is likely to be very small (this area is much larger in the cat). The function of the red nucleus is unknown. However, in man it is likely that the role of red nucleus is much more important in its interaction with the cerebellum than direct spinal influence. Lesion in the red nucleus results in tremor of the arm and ataxia. This is likely due to destruction of the cerebellar fibers that pass through the red nucleus.

Reticulospinal tracts

Refer to Slide 17. The reticulospinal system is composed of large fiber axons, serving as a fast transmission line to the spinal cord. It serves postural control and balance, acting on the anti-gravity muscles of the arms and legs.

Pontine reticulospinal tract is uncrossed, descends the length of the spinal cord and has direct monosynaptic excitatory inputs on motor neurons innervating anti-gravity muscles of the legs (lower limb extensors) and upper limb flexors.

The medullary reticulospinal tract is bilateral (both crossed and uncrossed). Descending motor tracts that originate in the medulla have an inhibitory action on the muscles. When the medulla is electrically stimulated, muscle tone vanishes, almost as if one has thrown a switch for preventing movements at all levels.

Refer to Slide 18. The function of the reticulospinal tract is to maintain our posture. Before we lift a weight with our arm, muscles of the leg are excited to support our body posture. This postural control is via the fast acting excitatory action of the pontine reticulospinal tract.

In this figure, the subject will lift the lever by activating his biceps. The weight of the object, which extends far beyond the body's center of gravity, tends to pull the person forward and off balance. This weight, if uncompensated, will flex the knees and ankles. To counter these forces, the leg muscles co-contract around the knee and ankle joints to stiffen the leg. Note that activity in the leg muscles precedes the activity in the biceps. It is as if the brain recognizes that an arm movement that is about to be produced will have consequences on the leg. The

interesting point to note is that the consequences of a given arm movement are different depending on the configuration of the body. For example, imagine yourself lifting a load when you are standing vs. when you are sitting down on a tall stool with your legs dangling. In the standing posture, it would make sense for your brain to activate your leg muscles slightly before you begin your lift. However, when you are sitting, it should be your back muscles that are activated first and the leg muscles should be quiet as they have no postural role to play. Therefore, the activity of the leg muscles cannot be a stereotypical program that is played out every time there is an elbow flexion. Rather, the brain needs to take into account the context in which the arm movement is about to be performed in computing the consequences on other body part.

Refer to Slide 19. Volunteers were asked to pull on a fixed handle when they heard a tone. Because the handle was attached to a solid wall, the pull caused the subjects to sway forward. This sway would produce a flexion in the ankle and in the knee. Just before the brain activated the biceps to pull on the handle, the brain activated the quadriceps (a knee extensor), hamstrings (a knee flexor), and the gastrocnemius (an ankle extensor). The co-activation of the muscles around the knee will stiffen the knee joint.

To test whether this anticipatory postural adjustment was context dependent, consider a condition where the volunteer was supported by a padded cross brace at shoulder height. In this condition, the act of pulling on the handle will not result in a sway because of the shoulder brace. This changes the context of the pull. Now when the biceps is activated, you see little or no activity at the gastrocnemius and the hamstrings. Therefore, the leg muscles are activated only when the arm movement is anticipated to have a consequence on the position of leg joints. This anticipatory behavior is believed to rely on the pontine reticulospinal tract.

Narcolepsy: Much of what we know about the motor function of the medulla comes from the unlikely source of the study of sleep disorders. If you have ever gone without sleep for 48 hours, you have experienced the sleepiness that a narcoleptic lives with every day. In spite of being so sleepy, they tend to sleep poorly at night. As a result, narcoleptics fall asleep at dangerous or inappropriate times. A major advance in narcolepsy research occurred in the early 1970s, when investigators observed that some dogs display symptoms very similar to those of human narcoleptics. Recordings from the medulla in these animals showed that the main function of the muscle-tone control system in the medulla is in suppressing muscle activity during a portion of sleep called REM sleep. It is during this period that vivid dreams take place and normal tone is completely absent from limb and back muscles. The medullary motor region is inactive when animals are moving, moderately active when animals sit or lie down, further activated during non-REM sleep and maximally active in REM sleep. When you try to relax or "turn off" your muscles, you are actually trying to "turn on" this brain region. In narcoleptics, it appears that higher brain regions fail to properly inhibit the medulla.

Vestibulospinal tract

Refer to Slide 20. The vestibular system serves to regulate posture and to coordinate eye and head movements. For example, to keep the head upright and define the vertical, the vestibular system acts on the neck muscles using input from both the visual system and from the semicircular canals. It uses information from both of these sources, as well as proprioception, to excite the antigravity muscles to hold the body upright. If you were to spin yourself around until you felt dizzy and then try to stand still, you will stagger. This is because the vestibulospinal system is being over-stimulated. Finally, it plays a dominant role in stabilizing the eyes in space when the head is moving. It gives you the ability to keep looking at an object (maintain gaze) even though your head may be rotating.

Vestibular nuclei reside in the mid and lower pons and the medulla, and are composed of the superior, lateral, inferior, and medial nuclei. They send most of their output to the spinal cord and to the extraoccular muscles. There are two vestibulospinal tracts, lateral and medial. Names derive from nuclei of origin. LVST is the much bigger tract in terms of number of axons. It descends down the length of the cord and excites antigravity muscles. Its function is to hold the body upright and prevent collapse. Its biggest input is from the cerebellum. For example, if the floor under us suddenly shift backwards, tilting the body forward, the action of the LVST is to contract the gastrocnemius (stabilize the ankle) and hamstrings (stabilize the knee). A patient with cerebellar damage might be unable to do this.

MVST has smaller number of axons and descends only to the cervical spinal cord. The function of MVST is to stabilize the head in space when the body is moving (for example, when we are walking around), and to stabilize the eyes and maintain gaze during head movements.

Refer to Slide 21. Summary slide of the various tracts.

Lesion at the level of the internal capsule

This lesion involves the corticospinal tract and the input of the cortical structures onto the brainstem motor centers.

Refer to Slide 22. Lesion of the internal capsule is the most common cause of damage to the descending cortical motor pathways. The symptoms are both due to a loss of corticospinal input and cortical input to the red nucleus and brain stem motor regions. This lesion is characterized by the following sequence of events: Immediately there is a period of paralysis. Limbs are flaccid and no reflexes can be obtained.

Refer to Slide 23. The first reflexes appear at 10-12 hours in the form of the Babinski sign: normally, stroking the foot results in flexion of all the toes. In lesioned corticospinal tract patients, there is an extension of the big toe in response to bottom of the foot stimulation (Babinski sign). This is believed to be a result of loss of excitatory input from the corticospinal tract on the flexion reflex afferent pathway.

Lesion of the corticospinal tract in the medulla

Refer to Slide 24. This lesion spares the brainstem motor centers and their descending tracts. Therefore, control of proximal arm muscles and control of posture is generally intact. However, the patient will have substantial deficits in control of distal muscles of the arm, hand and fingers. There is no spasticity. Spasticity refers to the level of resistance that is offered by the limb as it is passively moved. In a spastic patient, the spinal stretch reflexes are hyperactive. In a corticospinal tract lesion, the patient is not spastic. However, the Babinski sign is observed in these patients.

Lesion of vestibulo-spinal and reticulo-spinal pathways

This lesion results in severe postural deficits. The patient cannot right herself but has no problems making arm movements. Control of finger muscles is intact because the corticospinal tract is intact.

Lesion of the spinal cord

This lesion results in loss of tonic excitation from the corticospinal and brainstem tracts. Lesion is immediately followed by paralysis and areflexia (all spinal reflexes have extremely high threshold for activation). First signs of recovery are the appearance of cutaneous withdrawal reflexes. Some months later, enhanced flexor withdrawal reflexes are observed and may lead to permanent posture of flexion of the hip, knee and ankle.

At about 12 hours after the lesion, spasticity is observed in the extensors of the leg and flexors of the arm. Spasticity is detected by passively moving the limb about the joint and noting the resistance. Normally resistance is mild. In spasticity, there is increased resistance.

Recovery from lesion of the corticospinal tract may involve the rubrospinal tract

Refer to Slide 25. In this experiment, the monkey's corticospinal tract was lesioned in the left medulla, rostral to the decussation. The lesion affected control of movements with the right arm. However, after about 10 days, there was significant recovery.

Refer to Slide 26. It was hypothesized that a reason for this recovery may be a change in the role of the rubrospinal tract. Whereas in a control animal this tract is predominantly involved in excitation of extensor muscles of the arm, in the lesioned animal that had recovered the rubrospinal tract was much more balanced in its control of flexor and extensor muscles.