

Lecture 8: motor control by upper motor neurons

Overview:

(This is not said in the lecture, it's written here to help you visualize the whole topic of motor control in the body. If you like to read only the lecture, please skip to the next page.)

Controlling of any movement in our body has to do with controlling of both skeletal muscle contraction **on one side** and deciding that the body must move immediately in this direction not in that one, and smoothly not forcefully and other issues like this **on the other side**.

Skeletal muscle contraction is initiated by lower motor neurons in the spinal cord and brainstem, these neurons as we already know send axons directly to skeletal muscles hence the other name: final common pathway. Activation of them is determined by local circuits located within the spinal cord and brainstem, these local circuit neurons receive direct input from sensory neurons and mediate sensory-motor reflexes and coordinate the activity of lower motor neurons. Descending pathways modulate the activity of lower motor neurons by influencing this local circuitry.

Descending pathways are the axons of upper motor neurons which are located in cerebral cortex (mainly the frontal lobe) and in brainstem centers. The upper motor neuron pathways that arise in the brainstem centers are essential for basic movements and postural control. And the ones that arise in the cortex are essential for the planning and initiation of voluntary movements and for directing complex skilled movements.

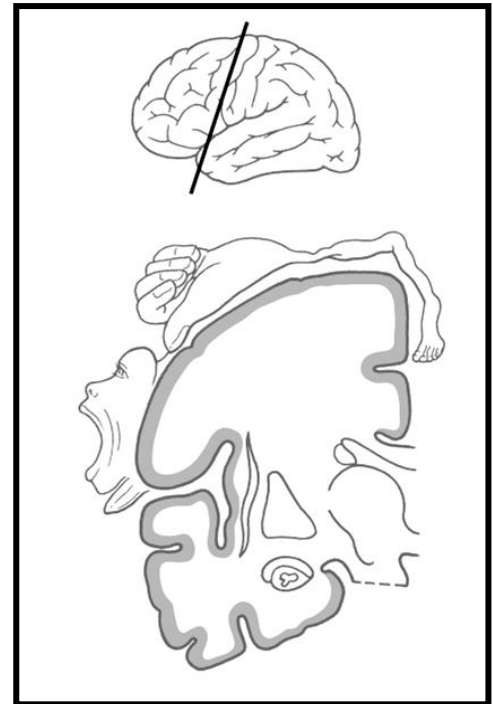
The activity of the upper motor neurons is regulated by basal ganglia and cerebellum in association with other brain regions such as the thalamus.

The information is not in same order mentioned in the lecture

Somatotopic organisation of the motor cortex:

Motor system starts in the cortex. The correspondence of body parts to areas of motor cortex is what's known as somatotopic organization. From medially to laterally, the cortex represents foot, leg, trunk, arm, hand, face, mouth, internal mouth, and esophagus being most lateral.

We can see that the lower extremities are located medially on brain cortex and this is the only place where the body is represented upside down; this means that once the axons descend from here they switch, i.e. the lower extremities go laterally and body trunk along with upper extremities goes medially. This new arrangement goes all through the corticospinal tract in the midbrain and in all the brain stem, even after decussating in the spinal cord, in the cervical segments, and down through the spinal cord.



It's important to know somatotopic organization in order to understand the lesions of the whole tract, in any part of it, in the cortex, as well as expanding lesions whether extra or intra medullary.

The components of the corticospinal pathway:

60% of upper motor neurons of corticospinal pathway reside in the cortex anterior to the central sulcus, and 40% of them reside posterior to it.

- The area anterior to the central sulcus is divided into different motor maps that represent the body: one map is the homunculus in the primary motor cortex (area 4), two or three maps are in area 6. Area 6 is divided for only 2 functional areas for simplicity: the premotor cortex, and the supplementary motor cortex. All the motor cortex areas interfere and synapse and connect with each other and control each other, and all of them contribute to the corticospinal tract.
- Those 60% of the corticospinal tract which are anterior to the sulcus are divided into:
29% comes from primary cortex only (area 4) and 31% comes from both supplementary and premotor cortex (area 6).
- The other components of corticospinal tract that come from the cortex posterior to the central sulcus and descend directly to it to control the motor functions are:
 - Primary somatosensory cortex (25-30%) sends fibers that go down with the corticospinal tract.
 - Posterior parietal cortex area (areas 5 and 7; sensory cortex) but this is almost neglected.

- Cingulate gyrus which is on the middle part on the sagittal section of the brain (medial side) also contributes to the corticospinal tract. It is usually connected to the limbic system.
- So the corticospinal tract is multi-area tract that has many components that contribute to it either directly or indirectly; the components that interfere indirectly with it are:
 - The parietal cortex (areas 5 and 7) synapse reversibly with the motor cortex for modulating the cortex function.
 - The prefrontal cortex (areas 9 and 46) also communicates with supplementary, premotor, and primary motor cortex to modulate their functions without descending down in the tract (mainly with the supplementary and premotor).
 - Areas 44 and 45 which have to do with language too.
 - The limbic system and its connection to the premotor.

So these brain areas, although don't descend directly in the tract, can modulate the motor cortex function. Don't worry if you didn't get them now, they will be discussed again later.

Primary motor cortex (M1):

It controls one-joint movement. According to Guyton, "point stimulation in the hand and speech areas in the primary cortex on rare occasion causes contraction of a single muscle; most often, stimulation contracts a group of muscles instead i.e. excitation of a single primary motor cortex neuron usually excites a specific movement rather than one specific muscle."

Supplementary motor cortex:

This is the upper and medial side of area 6. Its function is to control more than one joint or sequential movement (successive one-joint movements) in addition to their control by the primary motor; the supplementary motor area causes much more complex patterns of movement than the one-joint movements generated in the primary cortex.

The main function of the supplementary motor cortex is thinking and planning for the movement. It produces an image of the movement; it imagines the total muscle movement that has to be performed then it excites the primary cortex to produce each successive pattern of muscle activity required to achieve the image. So it's responsible for processing what should be done to move and send this image to the primary cortex.

Don't forget that It also sends direct corticospinal fibers to execute directly some complex multi-joint movement, that is, multi-joint movement could take place under the control of supplementary directly without the activation of the primary cortex (M1).

So: If we ask the patient to raise his finger, this is one-joint movement and here the primary motor cortex will work.

If we ask the patient to raise more than one finger, this is more than one joint movement and here both the supplementary first and the primary second will be activated.

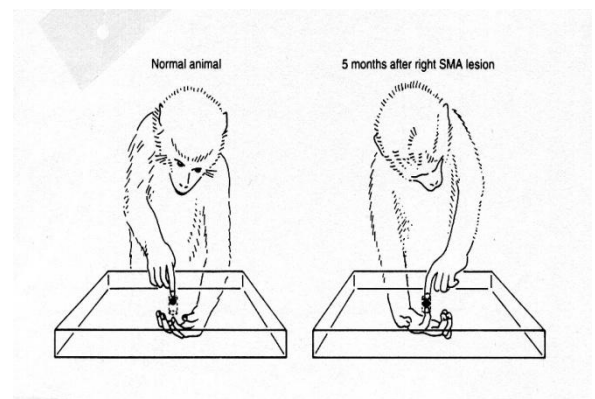
If we asked the patient to just imagine raising more than one finger or imagine doing a sequential movement, only the supplementary motor cortex will be working.

Another target for the supplementary motor cortex other than “talking” with the primary cortex is to connect and synapse with the contralateral motor area in the brain and control it so that the contractions that are stimulated by the supplementary motor cortex are bilateral rather than unilateral. If the supplementary motor area is lost, this won't affect sequential and complicated movement too much and skilled movement will be a little affected because the premotor area will almost compensate (as you'll see shortly), but there will be marked loss in communication with the contralateral side because the premotor area doesn't synapse with it so coordination between the two limbs will be lost.

For example if left supplementary cortex is lost, the patient will lose only a little of the complicated movements. But when he tries to move both hands in the same way, he can't coordinate that; this is the main deficit and the most obvious symptom in supplementary motor cortex loss.

Experiment was held to study this is on a monkey:

They made a hole in the table and put a piece of raisin (زبيب) inside it. Normally, the monkey uses both his hands coordinately to get the piece out (by one hand pressing and the other catching the piece when it drops). But after removing the supplementary cortex, that monkey, in spite of normal muscle contractions and movements, couldn't coordinate between the two hands (the monkey was pressing on the piece with the two hands so it couldn't manage to get it out).



In other words:

When you want to do something with the two hands, the left hand is stimulated by the right cortex and the right hand is stimulated by the left cortex. But the two hands need to coordinate with each other so the two cortices must talk to each other; the area that's responsible for this is the supplementary motor area.

Premotor cortex:

It's in the other part of area 6; it's divided into dorsal and ventral areas:

The dorsal part is located next to the supplementary motor cortex. It is usually for planning of the movement and imagining a little bit of it. The planning by the premotor cortex is mainly for multi-sequential movements or steps rather than for complex movement, it also sometimes plans for sensory directed movement. It has many maps that hold thinking and processing of how to do the movement. After getting inputs from multisensations, it has to do with targeting and initiation of movement especially in space. Like the supplementary motor cortex, the premotor cortex imagines the total muscle movement that has to be performed then it excites the primary cortex to produce each successive pattern of muscle activity required to achieve the image. So it can compensate for the loss of the supplementary motor cortex, but if both are lost, general apraxia will occur (here the patient can't do complex movement even with one hand). Apraxia is inability to hold complex movement functions although there's no any paralysis in any muscle; simple movement is still intact.

The ventral part mainly works with sensory guided movement, especially vision-motor coordination or hand-eye coordination of movement. It has to do with the processing of how to work in my vision field. It's recently discovered that the premotor cortex has a special class of neurons called the **mirror motor neurons**. These neurons fire to excite a pattern of muscles that imitate the pattern we see; they fire in a pattern to excite the same pattern I see. This means that when we observe a movement performed by other person, these neurons become activated and start stimulating the circuit areas that can perform that same specific movement that we see. It's responsible for visual motor learning of skills in the young, copying of the emotions (visual emotional learning) and learning of behaviors. Studies showed that mirror neurons stand behind some developmental disorders like autism and some psychiatric disorders like schizophrenia.

Lesion of this area will not only give symptoms of loss of visual guided movement, it will affect also some learning, communication, and some disabilities in personality.

So briefly: These mirror neurons are activated when you see any function, they are simply found to "mirror" that function. When someone next to you writes, yawns, or raises his/her hand, your mirror neurons stimulate the area of motor neurons that can perform that same task you see. Of course there are other regulators that inhibit mimicking every act we see.

Decorticate and decerebrate rigidities:

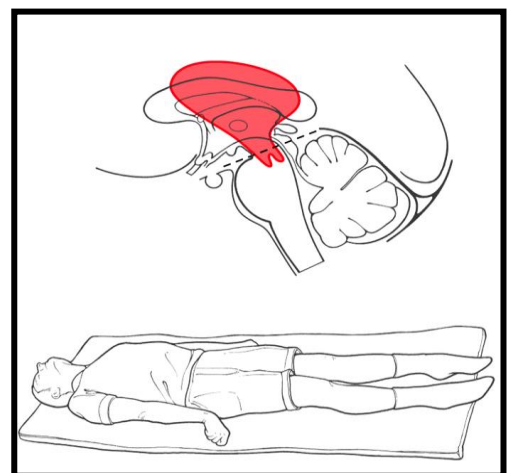
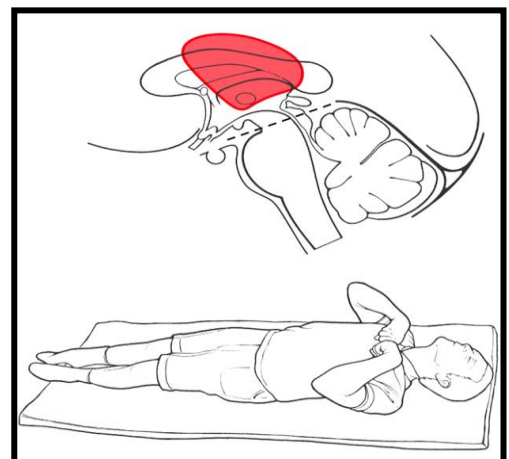
As you know, lower circuits in the spinal cord mediate the spinal reflexes, and cortex controls these circuits via the extrapyramidal tract/ indirect pathway (through brainstem centers) and the pyramidal tract/direct pathway.

If the lesion is rostral to the midbrain and the control by the cortex (pyramidal) is lost but the control by the brainstem (extrapyramidal) isn't affected, the excitatory activity of flexor muscles by the cortex will be lost and all the body will have extension which is triggered by the brainstem (extrapyramidal), except for the areas where the intact red nucleus inhibit their extension by the brainstem (mainly the upper limbs) and flexion of them will occur. This is the **decorticate rigidity**.

In decorticate posture the person is stiff with bent arms (extension of lower limbs and flexion of upper limbs), as shown in the upper picture.

But if the midbrain with the red nucleus inside it is lost (by haemorrhage, tumor, or other lesions), there will be no flexion because there is no any inhibition of upper limb extension. This is the **decerebrate rigidity**.

In decerebrate posture, the person has his arms and legs held straight (extended), as shown in lower picture.



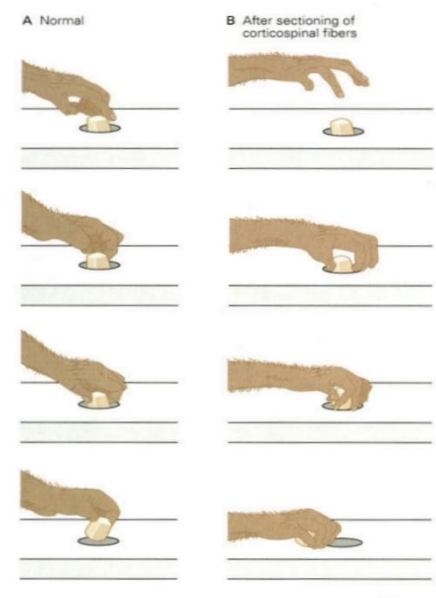
Pyramidotomy (pyramidectomy):

As we already know, all the components of the corticospinal tract send fibers to the midbrain as well as to the spinal cord and affect both the pyramidal and extrapyramidal tracts. Some of the extrapyramidal tracts descend and function ipsilaterally, and others decussate and function contralaterally.

A lesion in the corticospinal tract alone is almost very rare in humans; normally other things are lost in addition to the loss of the corticospinal tract. But by experiments, it's found that the lesion in corticospinal tract alone (especially in the medulla when we section the pyramid) doesn't give rise to hemiplegia nor to hemiparalysis because there are still intact indirect extrapyramidal pathways of motor control especially the corticopontine pathway and other pathways between the cortex and the brainstem. So instead, it leads to hemiparesis (weakness of movement) and loss of very fine control of movement. But if the lesion happened to the spinal cord, to the midbrain (peduncles), or to the internal capsule, then all the pathways will be cut and all the movements will be lost i.e. hemiplegia.

There are many experiments on monkeys to study the effect of pyramid sectioning at the level of the medulla. Four days after the surgery, they were jumping and moving normally but without being able to bring items using their fingers (very fine movements). After some time the monkeys try to grasp any item by rolling it out by all the hand, and sometimes they successfully do it (see the next figure).

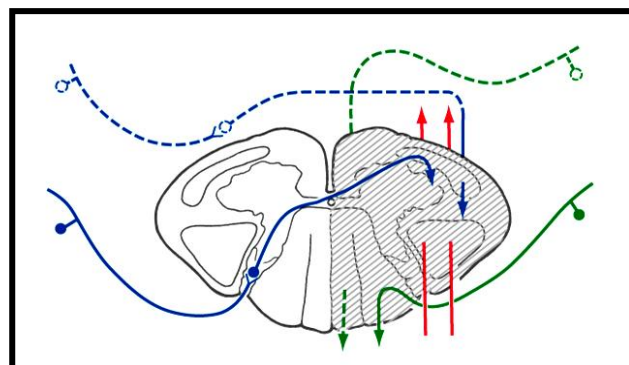
Induced removal of the corticospinal tract, or pyramidotomy, is clinically important in relieving hemiballism and chorea. These are disorders in the basal ganglia where involuntary movements occur in one limb. In the past they were treated by pyramidotomy, but although chorea or hemiballism is cured, fine movements were lost after the recovery. Nowadays the treatment is by drugs and pyramidotomy is still a way of therapy when the drugs don't work.



Lesions:

- **Hemi-lesion of the spinal cord at the level of C5 (The Brown-Sequard Syndrome)**

This is a rare lesion, It happens commonly by stabbing by a knife. This is a case of hemi lesion of spinal cord at the level of C5 on the patient's left side (remember that the right side on the section is the patient's left side), the symptoms are:



1- Somatosensory modalities and location:

- PCML, or dorsal column system, will be damaged on the ipsilateral side at the level of C6 and below, which means that two-point discrimination, vibration, and proprioception will be lost in the patient's left side.
- ALS will be damaged on the contralateral side at the level of C7 and below (almost 2 segments below C5), which means that pain, temperature, crude touch, and all the general sensations will be lost in the patient's right side. (ALS is represented in the picture by the decussating blue arrow.)
- Loss of all the modalities of somatosensory at the level of C5 on the ipsilateral side (left side).

2- Motor manifestations:

- Loss of motor at the level of C6 and below at the ipsilateral side and this will be manifested as **upper motor neuron paralysis/hemiplegia** on the left side. (Note that if the lesion was in C7, paraplegia will occur not hemiplegia)
- Loss of motor at the level of C5 at the ipsilateral side and this will be manifested as **lower motor neuron hemiplegia** on the left side (hypotonic paralysis).

Remember that there are two types of hemiplegia:

- 1- Upper motor neuron hemiplegia.
- 2- Lower motor neuron hemiplegia.

TABLE 16.1

Signs and Symptoms of Upper and Lower Motor Neuron Lesions

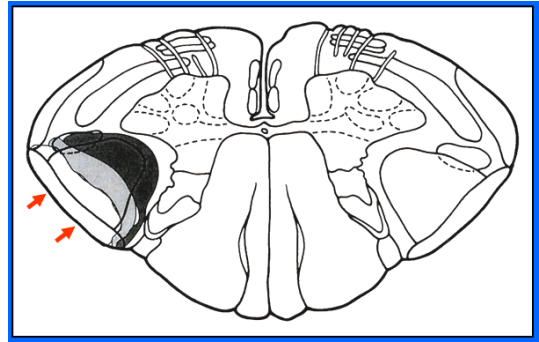
<i>Upper Motor Neuron Syndrome</i>	<i>Lower Motor Neuron Syndrome</i>
Weakness	Weakness or paralysis
Spasticity	Decreased superficial reflexes
Increased tone	Hypoactive deep reflexes
Hyperactive deep reflexes	Decreased tone
Clonus	Fasciculations and fibrillations
Babinski's sign	Severe muscle atrophy
Loss of fine voluntary movements	

- **Expanding extramedullary lesion :**

When there is a lesion in the lateral side (of the upper cervical segment C7, for example) that's expanding in the direction of the arrows, the symptoms will be:

1- There will be loss of posterior spinocerebellar tract so loss in the proprioception (position of body parts) will happen in the same side (ipsilateral).

2- There will be loss of corticospinal tract so loss of skilled movement starting from distal parts and loss of coordination (ataxia) will occur. Ataxia will start first in the lower extremities then weakness then paralysis (plegia) will follow. This expanding lesion will lead to progressive and continuous loss so it will start from the foot then ascends up (the lesion will expand from lateral to medial, that is, from lower extremities upwards; feet then leg then trunk and so on.)



Applications:

The interesting part of knowing the functions of the many motor cortical areas is the invention of brain-machine interface to restore mobility. In a study on a monkey, the monkey can control the robotic arm only by thinking held by the premotor and supplementary cortices (mainly the premotor) and without even moving any muscle. The research on brain-machine interfaces (BMI) is a modern field that is developing and is a big advance in neuroscience to help paralysed people to walk again only by imagining and thinking! The first demonstration on this was in the opening ceremony of the last world cup in Brazil; the first kick was shot by a paralysed guy wearing the mind-controlled robotic suit (exoskeleton). Credits go to the Brazilian neuroscientist, Miguel Nicolelis.

The simple concept of this great application is the control acquisition by the brain on the body and that all the body gains activity from the cortex, so the cortex can also control a robot to help people work and function again!



All the best!