

CENTRAL NERVOUS SYSTEM

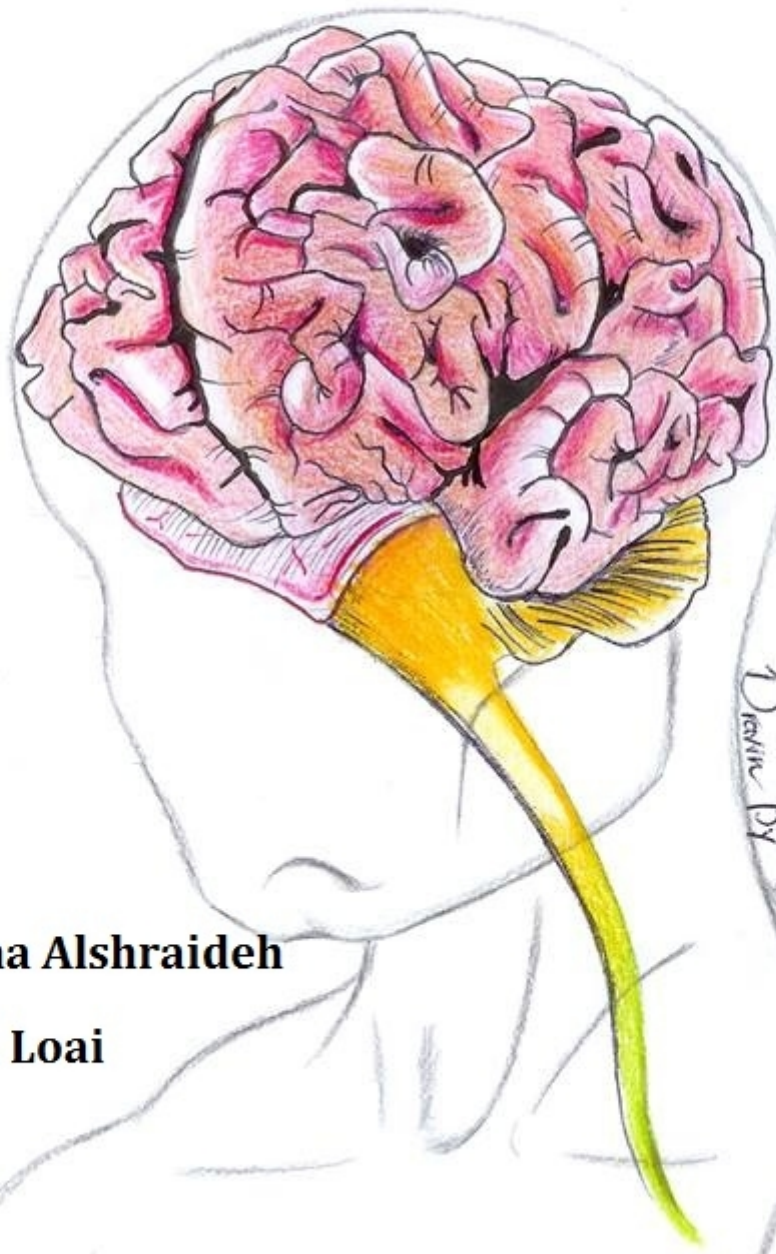
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Lec #: **8**



Drawn By Tariq Bushraaq...

Corticofugal Systems and the Control of Movement

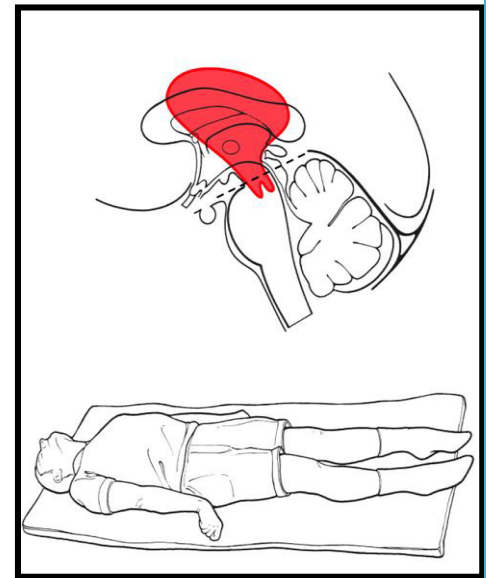
The following sections focus on the three pathways that originate in the brainstem: the vestibulospinal, rubrospinal, and reticulospinal tracts. Which are over controlled by higher Neural-centers, most importantly cerebral cortex and cerebellum.

In a case of a patient had a lesion related to the brainstem there will be two main common locations, either *above* or *within* the brainstem, leading to loss of control of the cortex over the component of the brainstem, giving the chance for the brainstem to have its own dominant control on its three pathways mentioned above, and mainly seen as a hyperactivity of the reticulospinal and vestibulospinal tracts.

Decerebrate Rigidity

-The lesion *within* the brainstem leading to isolation of the cortex and red nucleus *which is the origin for rubrospinal tract* from the nerves tracts and body extremities, respectively.

-Reticulospinal tract is left active with no inhibition from the cortex or any other regulatory system. The reticulospinal tract is mostly responsible for the activity of gamma motor neurons on the muscle spindle receptor causing hyper excitation to the **neck, axial, lower and upper limb extensor muscles**.

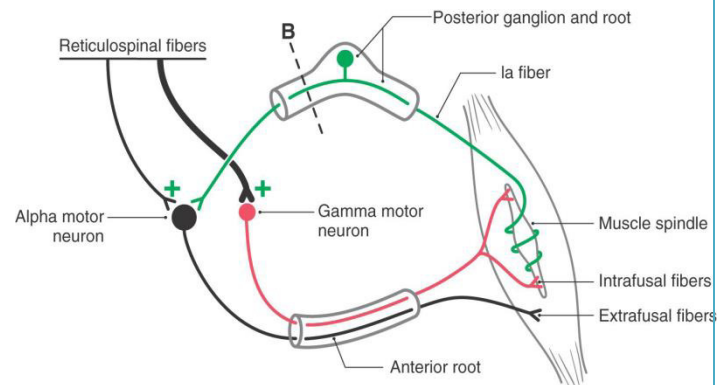


Note from the book: In the gamma loop, the supraspinal input activates the gamma motor neurons so that the intrafusal muscle fibers contract. Because the contraction of an intrafusal fiber has the effect of stretching the equatorial region between its two polar regions, it results in increased Ia fiber activity. In the spinal cord, this increase in Ia fiber discharge activates alpha motor neurons, which then activate extrafusal muscle fibers, resulting in muscle contraction.

****Board Question:** A patient had an accident trauma on his head in the midbrain region and got decerebrate rigidity and ulceration to the posterior root of cervical spinal nerves, which of the following symptoms he doesn't have?

Answer: extensor rigidity in his upper limb

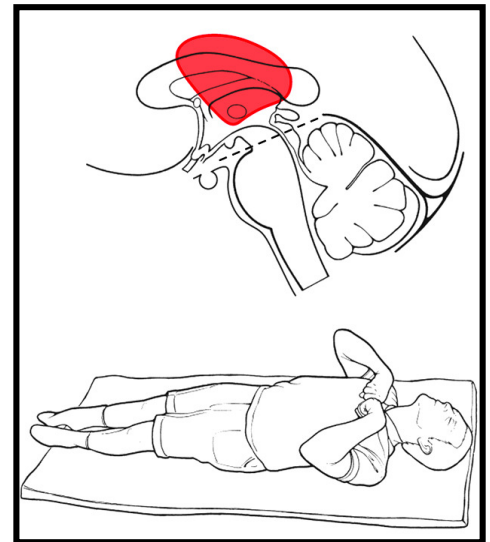
Explanation: gamma motor neurons are active and stimulate muscle spindle leading to increase in its stretch but the problem is it can't stimulate alpha motor neuron to increase muscle contraction and rigidity due to the ulceration and damage to the dorsal root.



Decorticate Rigidity

-The lesion *above* the brainstem, other way saying the midbrain is still intact leading to isolation of the cortex from the nerve tracts only while the Rubrospinal tract is spared in this case.

- Reticulospinal tract activates most of gamma motor neurons leading to extension of neck, axial and lower limb muscles, but not the upper limb due to the stronger stimulation of rubrospinal tract on upper limb flexors, causing it to flex.

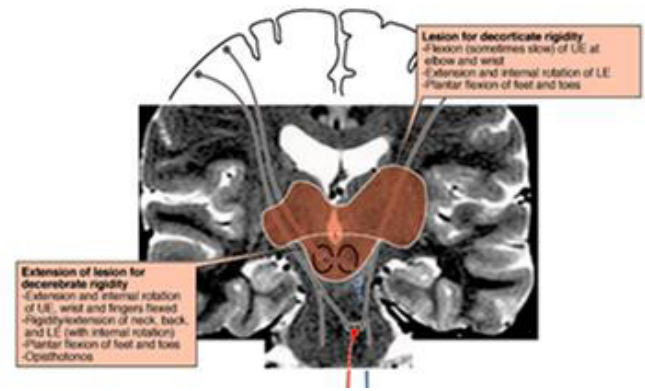


****Question:** What increases the presentation of decerebration or increase muscle spasticity for these patients, who have midbrain lesion?

Answer: the activation of the reticular formation by the excitement of ALS or pain tract, this will increase decerebration rigidity and muscle spasticity.

****MRI detection****: Best way to differentiate between decorticate or decerebrate, is by looking at the level of the lesion.

- Lesion above the level of cerebellum → • decorticate
- Lesion below the level of cerebellum → • decerebrate

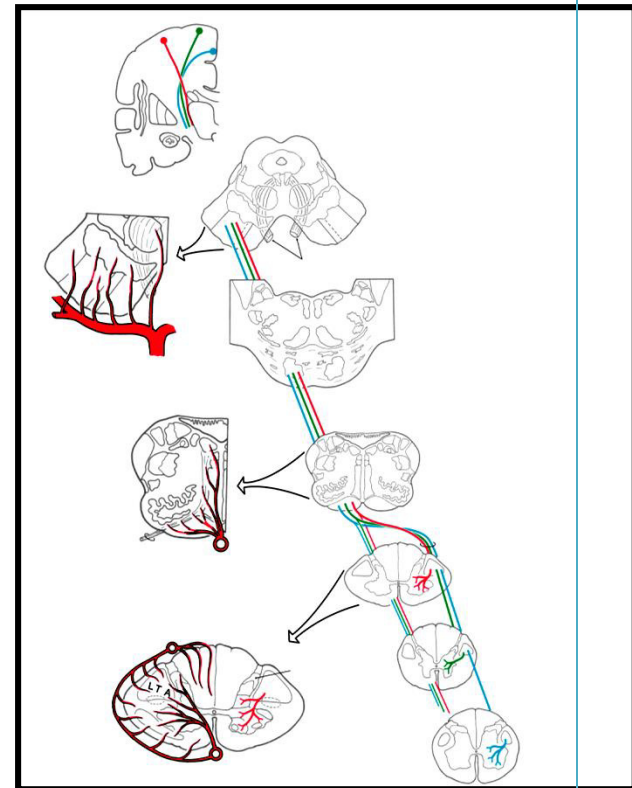


Corticospinal pathway

It's a voluntary tract that is involved with muscles, corticospinal pathway neurons start from the cortex and they give rise to axons that descend through the posterior limb of the internal capsule toward the brainstem, reaching the medulla where they cross and in this region it's called **motor decussation** area, they continue their way inside the spinal cord in the lateral column reaching its terminal destination which is alpha, gamma and interneuron cells.

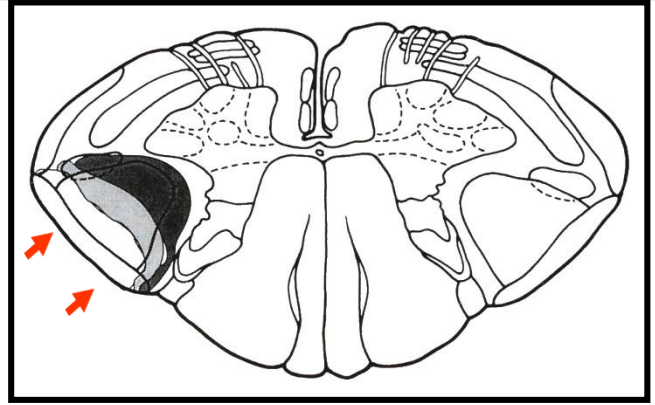
Somatotopic organization:

In the cortex, it's similar to the sensory somatotopic organization, which is the lower limb is most medial then there is the trunk to its lateral and finally the upper limb is most lateral. Once the neuron axons descend in the crus cerebri of midbrain, they invert making the upper limb most medial and the lower limb at the most lateral. ****NOTE:** after motor decussation the somatotopic remains the same, lower limb tract lateral, upper limb tract medial, and trunk tract at the middle.



The benefit of knowing this is to distinguish what is affected in case of an incomplete lesion of the tract, or an Expanding Extramedullary cancer on the main tracts.

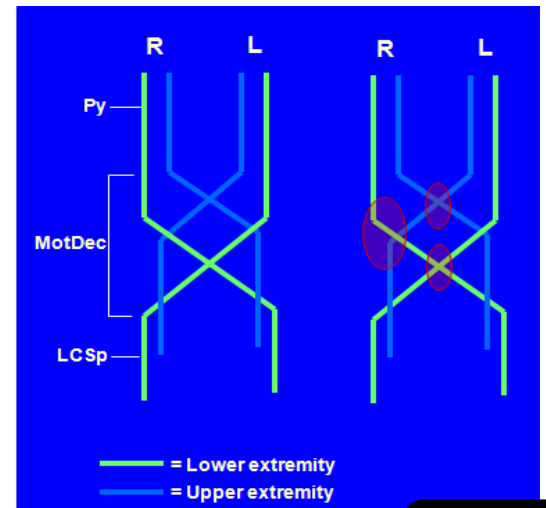
****Question:** by looking at the picture of an expanding tumor in the spinal cord coming from lateral to medial, what are the first affected muscles, and the symptoms seen in this patient?



Answer:

- Mostly common affected muscles are distal flexors, symptoms of weakness or paralysis start distally in the lower limb and ascend toward the trunk and upper limb.
- Dorsal spinocerebellar tract is the most lateral tract in the spinal cord and it is connected to the cerebellum, so it is the first nerve to be affected and damaged in this case and that will lead to → Ataxia.

*Note from the book: Consequently, lesions of corticospinal fibers rostral to the motor decussation result in **contralateral motor deficits**, whereas lesions of the corticospinal tract in the spinal cord result in **ipsilateral deficits**. An understanding of this concept of laterality is essential in the diagnosis of the neurologically impaired patient.*



****The crossing takes place at a region of 2 mm in the medulla, where the medial tract crosses first rostrally then the lateral tract follows caudally, and based on that there is different types of possible lesions:**

Example figure

<u>Region of the lesion</u>	<u>Place of weakness and paralysis</u>	
	<u>Upper limb</u>	<u>Lower limb</u>
All crossings	✓	✓
Rostral	✓ (both of the limbs Rt and Lt)	✗
Caudal	✗	✓ (both of the limbs Rt and Lt)
Most lateral	In this case there will be paralysis in one upper limb ipsilateral because the lesion was after the crossing and on one lower limb contralateral because the lesion was before the crossing. **In the example figure: paralysis in left leg and right arm.	



****MRI detection****: lesions in the crossing area of the medulla are hardly distinguished by using MRI, its mostly recognized and observed by clinical manifestations.

Corticospinal tract originate from the motor cortex, which is divided to many areas and regions that are involved in the creation of corticospinal nerve, for instance, precentral gyrus (primary motor cortex, area 4) which is responsible for the formation of one third of corticospinal cord, and many other areas like premotor cortex and supplement motor cortex (area 6) are also involved with different proportions. On the other hand about 40% of the corticospinal cord comes from postcentral gyrus from area (3,1,2) and posterior parietal cortex (5+7).

Function of the motor areas:

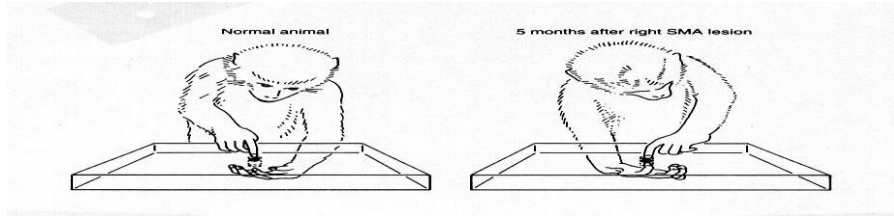
Generally saying they are complementary to each other.

→**Primary motor area**: simple direct action.

→**Premotor area**: complex thinking, preparation, and processing of motor movement. Coordination of movement with other sensations, especially the ventral side has a big role in visual coordination and processing due to its communication with the eyes.

→**Supplement motor cortex**: thinking of a complex movement before doing it, and coordination between muscles especially between the two hands. Because it's the most region that communicates between the two hemisphere in the regions of motor cortex.

****Experiment Note**: a monkey with a supplement motor lesion, couldn't figure and coordinate between his hand when he was introduced to a raisin was placed inside a hole in a table, which required the movement of both hands to get the raisin out of the hole, one hand pushes the raisin from one side and the other hand catches from the other side.



A Reminder Note: *The imagination of doing a certain movement without actual action, that will lead to the activation of the premotor area and supplement area, but in case of imagining doing a certain movement, and the actual process of doing it has occurred, the two areas of Premotor and supplement will be activated as well as the primary motor area.*

→ Area (3,1,2) Somatosensory, area (5,7), and cingulate motor cortex **directly contribute** to the corticospinal tract.

→ Other areas have a motor function in the brain, mainly contribute and plan the process before they get out and they **don't contribute directly** to the corticospinal tract like prefrontal and limbic cortex (area 9, 46).

Cortex takes over control of the voluntary muscles not only through the corticospinal tract, but also through innervating the reticular formation inside the Pons and medulla, vestibular system, and the red nucleus by axons descending from the cortex through the posterior limb of the internal capsule and crus cerebri terminating on the brainstem centers mentioned above.

Injuries and lesions:

- If the lesion was below the brainstem centers, there will be no Decerebrate or Decorticate rigidity.

-If the lesion was above the brainstem, there will be Decerebrate or Decorticate rigidity, due to loss of control from the cortex to the brainstem centers.

-If the lesion was on the corticospinal tract only (Pyramidotomy), there will be NO spastic rigidity neither paralysis, because the other upper tracts are still intact with the cortex, so the patient can still move, stand and walk, but what was lost is the fine movement.

****Experiment Note:** a monkey had a corticospinal tract cutting in the medulla, after four days he could climb the cage, but no fine movement was applied or seen.



A Reminder Note: *There is two parts of the motor system:*

→ *Lower motor neurons, are the motor neurons in the gray matter of the spinal cord, responsible for the innervation of the muscles and can be called common pathway.*

→ *Upper motor neurons, are the motor neurons that descend from the cortex down toward the spinal cord.*

-If the lesion was in the lower motor neuron there will be no way to excite the muscle, leading to **flaccid paralysis**, No reflexes, tone, and even baseline firing, leading to more weakness and paralysis, and giving rise to fasciculation or fibrillation.

-If the lesion was in the upper motor neuron and the lower motor neuron was still intact, the muscles increases its sensitivity, the tone is increased as well as the reflexes, may lead to spasticity. Clonus, Babinski sign is positive.

Clonus: is a test applied on the lower limb, by initiating the dorsiflexion of the foot it will lead to the vibration of the foot for couple of seconds before it comes to rest.

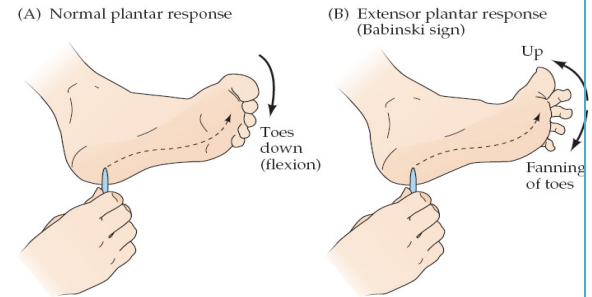
Babinski: is a test applied on the sole of the feet by stroking it, in normal people there will be curling of the toes and feet and it's called **negative Babinski** sign, but if the

patient had his foot sole stroked, there will be fanning of his toes and it's called **positive Babinski sign**.

These two signs are due to special connections in the gray matter of the spinal cord, involving the inhibitory interneurons called Rinshaw cells

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	



****Question:** a patient had a lower motor neuron lesion, would he have a negative or positive Babinski sign?

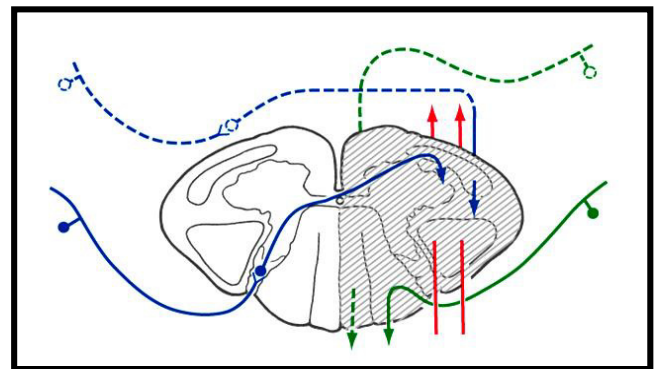
Answer: neither of them, because he has flaccid paralysis.

****Question:** a patient had a lesion on the left side of his spinal cord at the level of C7 as seen in the figure below, what are the symptoms seen and detected on the patient.

(remember: the right side on the section is the patient's left side)

Answer:

Sensory system point of view:



- 1- PCML, at C7 and below ipsilateral side lost two point discrimination, Proprioception, and vibration. And in dermatomes sensation the thumb is intact and the index is lost.



2- ALS, at the level of C7 there will be loss of thermal sensation and pain on the ipsilateral, but C8 and below will be contralateral for thermal sensation and pain.

Motor system point of view:

1-corticospinal tract, below C7 there will be no corticospinal tract that means upper motor paralysis.

2- But on the C7 level itself will be lower motor paralysis (flaccid).

References:

- Fundamental Neuroscience for Basic and Clinical Applications
- spinal cord reflexes slides #8
- lecture recordings
- medical-dictionary.thefreedictionary.com (some meanings of word)

This sheet is dedicated to : Ala'a Shaban , Mohamed Abualia ,Rakan Radi,Hamzah Mahafzah,Hasan Hammo . peace