CENTRAL NERVOUS SYSTEM

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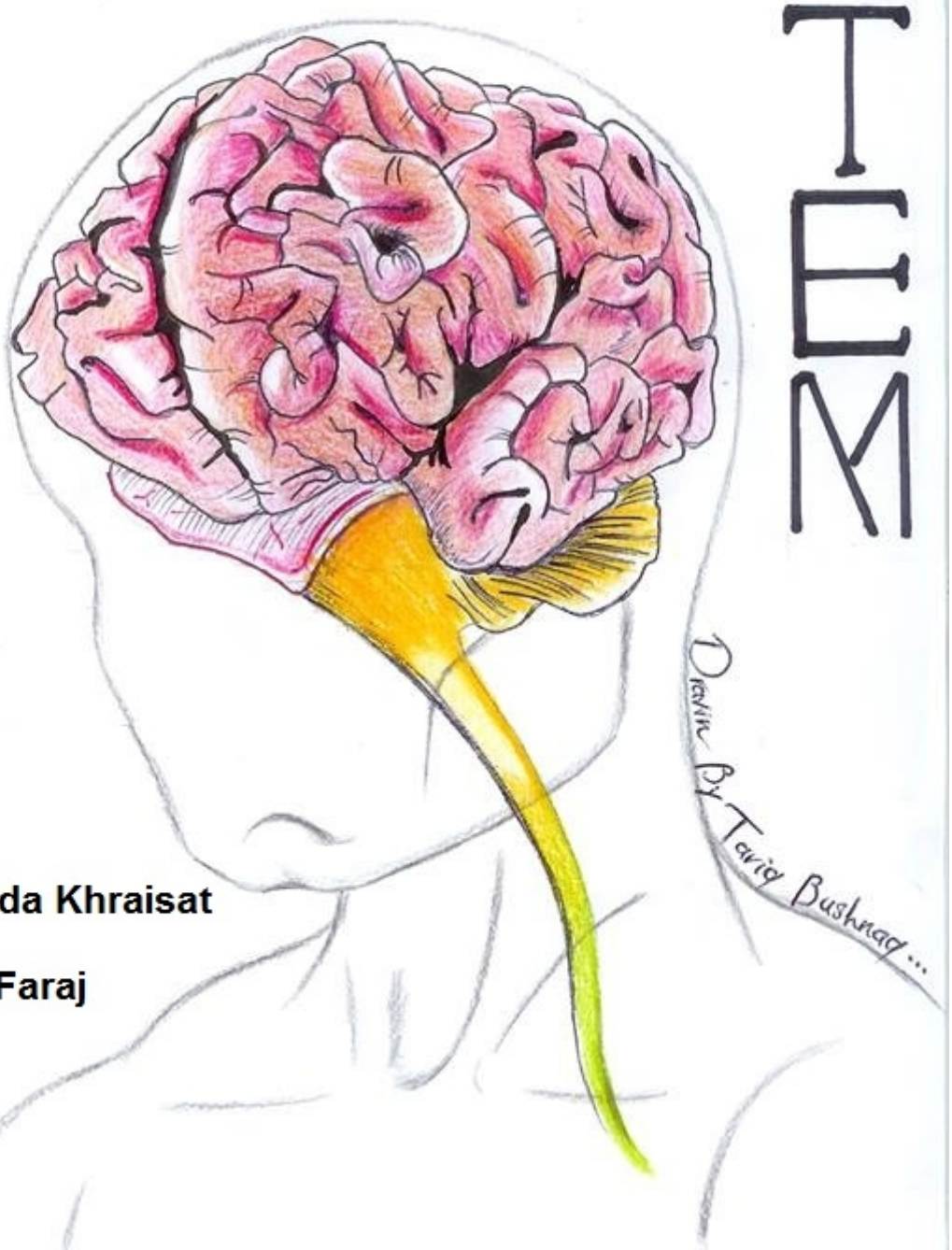

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Lec # : **6**

Motor neuron lesions and introduction to cerebellum

Upper motor neuron lesion:

In the last lecture we talked about the signs and symptoms of upper motor neuron lesion (symptoms of stroke).

You should also remember that Upper motor neuron lesion is: any damage induced to the neurons in the cortex or to their axons downward anywhere BEFORE reaching and synapsing in alpha and gamma in the spinal cord.

Also, we mentioned that Stroke could occur either in:

1- The cerebral cortex: the patient usually will haveLIMITED paresis or paralysis (Monoplegia:in one limb).

2- Internal capsule: (The most common site for stroke),the condition here will be more extensive, and the patient will have: HEMIPLEGIA,HEMIFACE and temporarily HEMIANESTHESIA(just in the first 3 hours after stroke).

*****In both cases the effect of damage will be CONTRALATERAL*****

3-in the brain stem: (either in the mid brain, pons or medulla), this case is called commonly ""Alternating Hemiplegia"": paralysis in one side of the body and lesion in a cranial nerve in the other side.

*other signs could be seenin the upper motor neuron lesions (in addition to paresis or paralysis):

1- Spasticity. (At the injured side)

2- Hyperreflexia. (At the injured side)

3-Clonus.

*These 3 signs are associated with the release of pontine reticulospinal tract (the most important one) from inhibition from the cortex**

4- Positive Babinski's sign.

5- Absence of abdominal reflex.

6- Absence of cremasteric reflex.

The last 3 signs are associated with the disturbance of flexor reflex as a result of damage in the pyramidal tract

*the earliest sign that appear in the upper motor neuron lesion is the Babinski's sign (appear immediately after the stroke), hyperreflexia needs 1-3 days approximately after the stroke to appear.

*HYPERREFLEXIA isn't of that significance unless it is associated with +ve Babinski's sign.

*if the upper motor neuron lesion occurs **ABOVE** the motor decussation(above the lower part of medulla), the effect will be **CONTRALATERAL**.

*if it occurs **BELOW** the motor decussation,below the medulla,within thewhite matter of the spinal cord (before synapsing at alpha and gamma), the effect will be below the level of the lesion and **IPSILATERAL**; for example if we cut the spinal cord at the level of T10 the paralysis will be ipsilateral below the level of T10 (in the lower limb, ipsilateral).

**Some typesof jerk reflexes(deep tendon reflexes) that can be done totest patients:

Name of jerk reflex	Root value of afferent and efferent nerves	Contracted muscle	Resulted movement
Biceps jerk	C5,C6	biceps brachii	Flexion of elbow
Ankle jerk	S1	gastrocnemius	Planter flexion
Knee jerk	L2,L3,L4	quadriceps femoris	Extension of knee joint
Triceps jerk	C7,C8	Triceps	Extension of elbow joint

Spasicity and rigidity:

These two terms are NOT equivalent!

** **Spasticity**: is a sign of upper motor neuron lesion, it affects Mostly flexors of the upper limb and extensors of the lower limbs so called "antigravity muscles". It's associated commonly with hyperreflexia.

****Rigidity**: this term is used commonly to describe the effect of diseases of basal ganglia/basal nuclei; for example: in Parkinson's disease. Rigidity affects BOTH extensors and flexors (increased tone in extensors and flexors) results in difficult and very limited movement. Finally, rigidity is NEVER associated with hyperreflexia.

Lower Motor Neuron Lesion:

It's any damage introduced to alpha and gamma or their axons (which run in the spinal nerves).

For example, if the median or ulnar nerves are being cut, the **muscles** which are supplied by these two nerves will suffer from the effect of the lower motor neuron lesion.

-Signs of lower motor neuron lesion:

1-Very**limited** paresis or paralysis **in a muscle or muscles** supplied by the affected segment(not the whole limb). For example, damage in C8, T1 segment results in paralysis JUST in the small muscles of the hand.

2-flacid paralysis in the affected muscles: paralysis here is associated with decrease or absence of tone (Hypotonia or atonia).

This is because the stretch reflex has beeninterrupted by the lesion (remember: any interruption of the stretch reflexanywhere: [receptor, dorsal root, ventral root, afferent, efferentnerves, alpha or gamma] there will be no reflex (areflexia or hyporeflexia)) as a result hypotonia, atonia.

3-Spontaneousinvoluntary contractions (muscle twitch) in the affected muscles; this sign is the most important and earliest sign that can be seen in the **acute stage of the lesion**(justin the earliest hours after the lesion)

If you can see theses spontaneous contractions by your naked eyes then this sign is called:**fasciculation**. (And it's the earliest sign seen in the acute stage of lower motor neuron lesion)

If you can't see these contraction by your eyes but you see them with the electromyography then this sign is called:**Fibrillation**

-The explanation of this sign:

At the site of lesion occur what so called "Injury potential":

When there is a lesion in the nerve, this will result in opening of voltage gated Na channels then depolarization and action potential will occur and it spreads all over the branches of the nerve (every branch goes to a motor unit), so many motor units will be activated at the same time and this results in obvious contractions seen by the naked eye (**Fasciculation**:A coarser form of muscular contraction,consisting on involuntary contraction of a group of muscle fibers).

After some time**, degeneration** will occur in the **beginning** of the nerve and action potential (injury potential) will be limited to the terminal parts of the nerve. As a result, contraction will be limited to only one or very small number of segments (very limited contractions) and it won't be seen by the naked eye. However, it could be detectable by electromyography (**fibrillation:**rapid twitching of individual muscle fibers with little or no movement of the muscle as a whole).

4-after several weeks, the affected muscle or muscles will suffer from **Wasting** or **atrophy** because of not using them.

The most important example for a lower motor neuron lesion is Poliomyelitis which is caused by infection with the poliovirus, this virus infect the cell bodies of neurons. The earliest sign of this lesion seen in the acute stage with fever is fasciculation or fibrillation in the affected muscle/muscles. **

Motor neuron diseases:

Diseases that: involve, affect both upper and lower motor neurons (degeneration in both of them).

For example, if you see a patient with paresis or paralysis of both upper and lower limbsat one side as if he has hemiplegia, you expect that this patient has upper motor neuron lesion, then you further examine him and you find that:

In the upper limb: he has paralysis, paresis, atrophy, hypotonia andhyporeflexia (signs of **LOWER**motor neuron lesion).

In the lower limb at the same side: he has paralysis,paresis, spasticity,hyperreflexia and clonus (signs of **UPPER** motor neuron lesion).

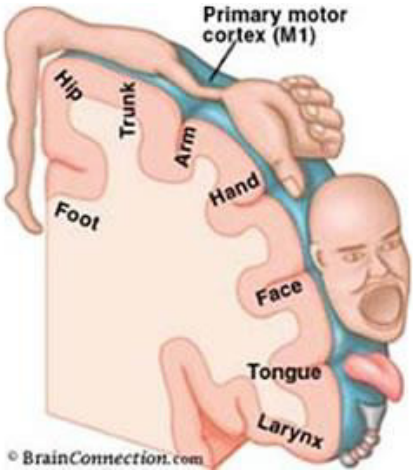
How this happened?(You expect to find signs of **UPPER** motor neuron lesion in both upper and lower limbs,because hemiplegia is a sign of upper motor neuron lesion, but actually you find symptoms of lower motor neuron lesion in the upper limb and signs of upper motor neuron lesion in the lower limb)!

Don't be confused, this is a typical case of a motor neuron disease in which both upper and lower motor neurons got damaged, for example , lesion in alpha and gamma in the cervical region (C5,6,7,8) results in lower motor neuron lesion(flaccid paralysis and atrophy) in the upper limb, at the same time the adjacent descending pathways (pyramidal and extrapyramidal) got damaged, this will result in an upper motor neuron lesion **below** the level of damage (below c8,in the lower limb) (spastic paralysis in the lower limb). Look at the figure page 45 from the handout☺

Functionalareas of the cortex:

***Area 4:** it has several names, according to Brodmann:area 4, functionally: primary motor area, anatomically: precentralgyrus and it is found in the frontal lobe. The body is presented in this area Upside down,precisely, but disproportionately.

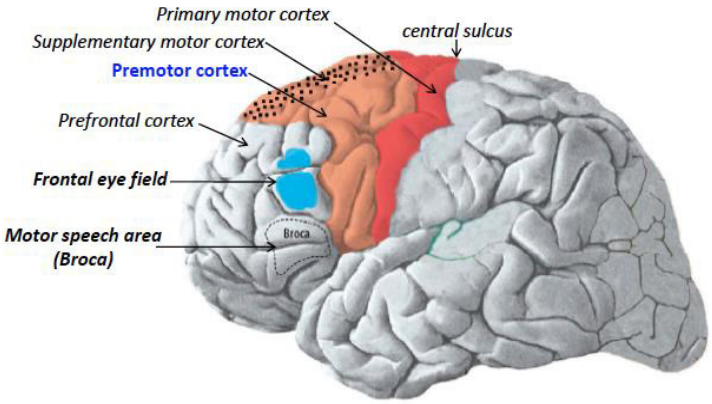
The face is presented in the lower part of this area, the upper limb (especially the hand) is in the middle, and the lower limb is in the medial side (in the Paracentral lobule).



-this area is primarily control distal muscles and works mainly *bylateral corticospinal tract*. Lesion in area 4 **alone** results in contralateral flaccid paralysis, But if area 6 is also involved this will result in contralateral spastic paralysis (because the pontine reticulospinal tract is released from inhibition from the cortex when area 6 is damaged).

***Area 6:** also it's located in the frontal lobe, it's composed of a lower part: premotor area and an upper part extending in the medial surface: supplementary motor area. Area 6 -as a whole- controls axial and proximal muscles *bythe ventral corticospinal tract and extrapyramidal tracts* (corticoreticulospinal tract,corticorubrospinal tract,corticotectospinal tract).

The activity of area 6 (premotor cortex and supplementary cortex) increases before the beginning of movement, so if we put an electrode at the surface of area 4 and area 6 the activity appears before the movement, this indicates that they are involved in**programming of movement** (especially area 6).



-premotor cortex is responsible for *postural preparation for coming movements*,so it has both inputs and outputs.

-inputs for premotor cortex:

1-from cerebellum passing by VL nucleus in the thalamus (remember that nothing comes from the cerebellum to the cerebral cortex without passing in the thalamus).

2- from posterior parietal cortex.

3-from supplementary motor area (upper part of area 6).

-Outputs from premotor cortex:

1-output to the brain stem inhibiting pontine reticulospinal tract.

2 –output descends in the spinal cord as a part of corticospinal tract.

3 –output to area 4 (very important)as area 6 is programming movements and area 4 is executing.

If the **premotor cortex** is involved in a lesion,**grasp response** reflex will appear in the patient. This reflex is initiated by putting your index between the thumb and the index of the patientand moving your index at the palm of the patient's hand, the patient willinvoluntary holdyour index. This reflex is normally (physiologically) found in the newborns.

Grasp reflex in anew born, and it could be seen pathologically in case of lesion in thepremotor cortex

Supplementary motor area:

As you know that simple movement as flexion and extension doesn't need programming, it can be carried out by motor areas without any need for area 6. However, complex movements (bimanual tasks for example) need programming i.e. need supplementary motor area.

Programming specifies the sequence of events, and which muscles I will use in order to achieve a coordinated complex movement.

For example if you want to carry something with your 2 hands, you will use flexors muscles in order to flex your fingers and wrist, also you need to stabilize your shoulder , the plan also determines the extent of movement, and all these are essential for complex movements.

So both premotor cortex and supplementary motor areasare responsible for complex movements (premotor cortex for posture preparations, and supplementary motor for bimanual complex movements).

As we know that also cerebellum and basal ganglia have a role in programming, so there is must be a connection between them and the Supplementary motor area.

-Inputs toSupplementary motor area:

1- from posterior parietal cortex (complex of sensation).

2-from cerebellum (passing by VL nucleus in the thalamus) and basal ganglia. (Remember that nothing comes from the cerebellum to the cerebral cortex without passing in the thalamus).

-OutputsfromSupplementary motor area:

1- to the cerebellum and basal ganglia.

2- to the spinal cord as a part of corticospinal tract.

3- to area 4 motor cortex (the most important one).

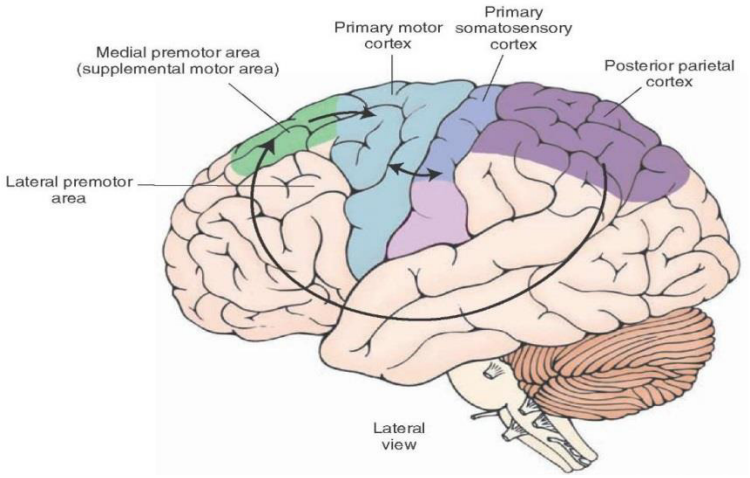
4-to the brain stem (inhibitory to pontine reticulospinal tract)

***remember that area 6 (with its two parts) inhibits pontine reticulospinal tract , so any damage to this area will result in release of inhibition and a hyperactive pontine reticulospinal which will eventually lead to spasticity as in UMN lesion**

-in bimanual complex movements, the blood supply will increase in supplementary motor area.

-lesion limited to the supplementary motor area will result in loss of coordination for bimanual tasks (no coordination between muscles of the right and of the left). In addition to inability to orient the hand towards a certain object.

***Posterior parietal cortex:(superior parietal lobule, area 5, 7):** this area lies in the parietal lobe posterior to area 3, 1, 2. It receives inputs from wide areas in the cortex; it receives sensory inputs from somatosensory cortex (area 3, 1, 2), visual, and auditory cortex. Also it receives Motor inputs, so within this area sensory and motor information come together. Once we have sensory and motor information in one functional area, they enable this area to generate **The Conscious map of the body**(the position of the body in the space, what I see, what I hear…). The most important function of this area is orienting the body towards the surrounding subjects. Lesion in this area will result in many symptoms but the most important one is **hemispatial neglect** "or **hemineglect**"(the patient ignores and even denies the presence of one half of the body and he can't orient his body towards the surrounding subjects so he easily and frequently shocks them.



***area 3, 1, 2:** (primary somatosensory cortex), post central gyrus. It receives somatic sensations, pain,temperature, touch, as well as proprioception (sense of position), from the **OPPOSITE** half of the body.

This area is divided into3, 1, 2 from anterior to posterior

Area 3 is divided into 3a, 3b.

Area 3a+2 receive proprioception. While area 3b+1 receive exteroception (pain, temperature, and touch).

Area 3,1,2 is very important since it gives us an accurate sensation ,so if it -or the sensory fibers that it receives- got damaged, the patient will lose the sensation Temporarily(for hours),then the sensation is sustained again at the level of thalamus, but it is not accurate (sense crudely).

Any sensory fiber ascending toarea 3,1,2 must run in one or more parts of the internal capsule (the most important one is the posterior limb of the internal capsule).

The last station for sensory pathways before reaching area 3,1,2 in the cortex is the thalamus, it must synapse at nucleus in the thalamus, the most important nucleus in the thalamus for sensation is the Ventrobasalcomplex with its two parts (VPM:sensation from the face ,and VPL: sensation from the body)

Propriospinal tracts:

Collections of intraspinaltracts that interconnect various levels of the spinal columns.

For example, if you look at the spinal column's muscles (in the cervical, thoracic, lumbar, sacral) they must act together in order to extend or flex the trunk.Another example is connecting the small muscles of the hand (connect c8, t1/connect two segments or 3).

All of these tracts are neurons within the gray matter of the spinal cord and their axons pass in fasciculus proprius(either anterior or lateral).

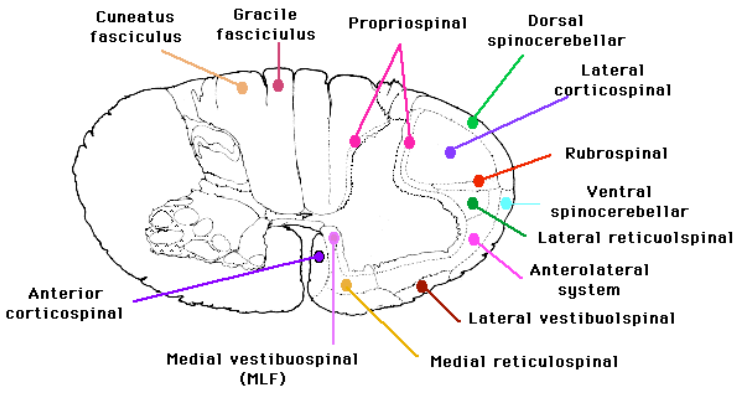
These tracts have 3 different types:

1-**Long**propriospinal tracts: their axons pass by the **ANTERIOR** fasciculus proprius and **connect wide areas** (for ex: cervical withthoracic and lumbar) of the spinalcord muscles.

2-**Intermediate**propriospinal tracts: their axons pass by **the LATERAL** fasciculus proprius and **connect proximal muscles with each other's**.

3- **Short** propriospinal tracts:their axons pass by **the LATERAL** fasciculus proprius and **connect distal muscles with each other's**.

You MUST differentiate between **propriospinal tracts**(neurons connecting different levels) and **Proprioception**(which is the sense of movement from muscles and joints)*



Lateral corticospinal tract:

This tract is found along the spinal cord and it affects alpha and gamma medially as well as laterally.

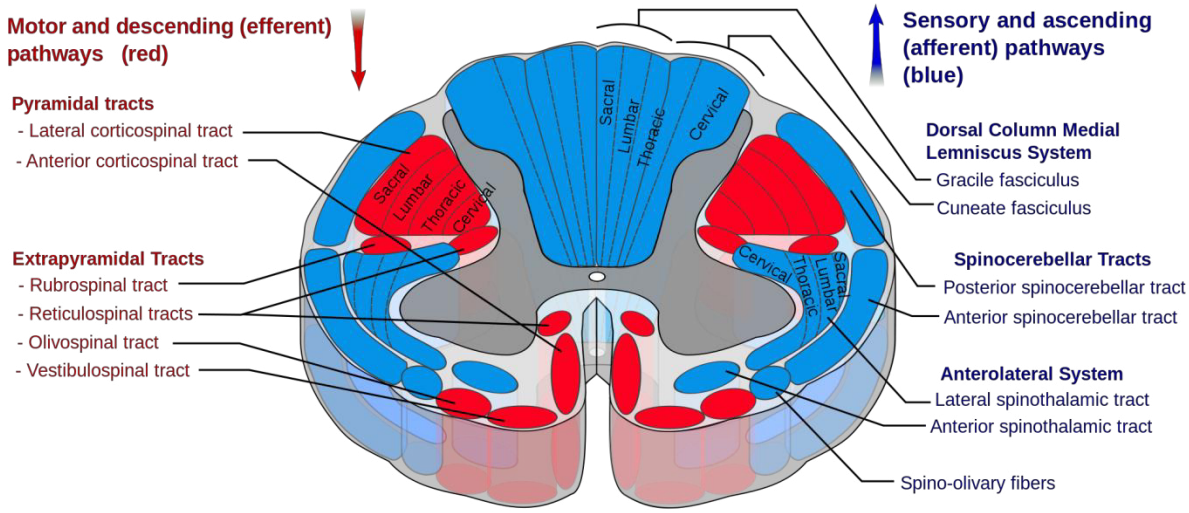
If alpha and gamma is affected medially: this effect is responsible for the axial and proximal muscles.

If alpha and gamma is affected laterally: this effect is responsible for the distal muscles.

So this tract has an effect in both proximal and distal muscles, but mainly it will affect the distal muscles (skill muscles).

-so a patient with a stroke will lose the skilled movements especially in the hand**FOREVER.**

-55% of the fibers of lateral corticospinal tract synapse at the lower cervical segments C5, C6,C7,C8,T1 which affect mainly the muscles of the upper limb and mainly distal muscles of the hand (C8,T1).

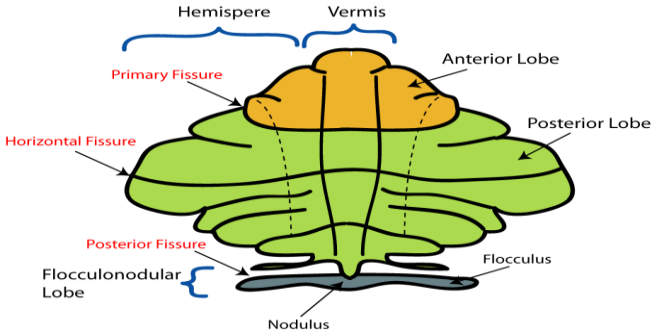


The Cerebellum:

-it controls (program) the movement of muscles, and send inputs for motor areas (area 4) for execution.

-It is dividedlongitudinally into: vermis,paravermis,and lateral part.

-It's divided anatomically (more important)horizontally by two transverse fissures into lobes (anterior, posterior and flocculonodularlobes) and lobules:



- If you look at the superior surface you will find the primary fissure; anterior to this fissure is the anterior lobe and posterior to this fissure is the posterior lobe(larger).
- The inferior surface of the cerebellum is ill-defined; the posterolateralfissure separates the posterior lobe from flocculonodular lobe.

-From phylogenetic perspective:

- The oldest part in the cerebellum is the **flocculonodular lobe.**This part of cerebellum (anatomically is called flocculonodular lobe), we call it **cerebellum of equilibrium**(itcommunicates with the vestibular system in the inner ear) we call it functionally**vestibulocerebellum**, and because it is the oldest in phylogenetic we call it also **Archi-cerebellum**.
- The anterior lobe is called spinocerebellum or paleocerebellum.
- The posterior lobe is called: cerebrocerebellum, neocerebellum, and it's the largest part of the lateral hemisphere.

- connecting the horizontal and longitudinal divisions:

- The anterior lobe is formed by vermis and most paravermis
- Posterior part consist of most of the lateral part hemisphere-

The vermis which is part of the anterior lobe controls axial(trunk), proximal muscles, head and neck muscles**BILATERALLY.(for example flexion of trunk needs contraction in both sides)**

The paravermis and the lateral part of the cerebellum affect muscles of the limbs **IPSILATERALLY**.(Each cerebellar hemisphere controlsipsilateralmuscles, while each cerebral hemisphere controlscontralateral muscles because of motor decussation).

**The cerebellum contains cerebellar cortex and the white matter inside contains groups of nuclei.

The major neuron in the cerebellar cortex is Purkinje cell.**

**Cerebellum receives inputs and gives rise to outputs.

The input should ultimately reaches the Purkinjecell, and then purkinje gives rise to output to a certain nucleus. And the final output comes from the nuclei.

#Purkinje of **vermis;**itsnucleus is called **fastigial nucleus**.

##Purkinje of **paravermis**has 2 nuclei**: globose** nucleus and the **emboliform** nucleus together are **called interposed nucleus**.

###Purkinje of the **lateral part** (the largest part of a hemisphere);its nucleus is**dentate nucleus.**

And there is an overlap between these nuclei (for examples, cells in vermis and paravermis may use the dentate nucleus), but generally each region has its specific nuclei.

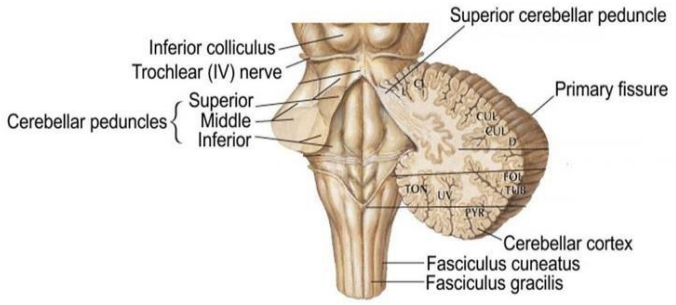
*How the cerebellum works?

-the cerebellum is connected to the brain stem by 3 pairs of cerebellar peduncles:

1- Superior cerebellar peduncle: connects the cerebellum with midbrain.

2- Middle cerebellar peduncle: connects the cerebellum with the pons.

3-inferiorcerebellar peduncle: connects the cerebellum with the medulla oblongata.



For example, certain part of the cerebellum receives the input from the reticular formation,this input enters the cerebellum by the inferior cerebellar peduncle, as it enters it gives collaterals, these collaterals excite the nucleus, ultimately this input should reach the purkinje cell,then purkinje cell gives output to a nucleus, after that the final output goes from this nucleus to the reticular formation again(notice that the input comes from the reticular formation and the output comes back to the RF).

-how we then reach and control muscles? (remember that the reticulospinal tract rises from the reticular formation) after the information reaches the reticular formation the reticulospinal tract will descend and synapse at alpha and gamma affecting the corresponding muscles),so the cerebellum **DOESN'T** react with alpha and gamma directly**.(THERE ISN'T a tract called cerebellospinal tract, always remember that the cerebellum affect alpha and gamma INDIRECTLY).**

-Another example, the cerebellum receives input from the cerebral cortex, passing in pons by a pathway called **Corticopontocerebellur tract**, this input stimulate a nucleus then ends in purkinje cells, which sends output to a nucleus then final output comes out from the dentate nucleus for example going back to the cortex.

So this is how the cerebellum work 1- Feedback (correction of movements after occurring)or 2-feed forward by sending stimulus to the cortex before the beginning of the movement (make sure that the movement is correct before it begins)

-posterior lobe of the lateral part of the cerebellum is responsible for learning and storage of sequential component of movement.

End of the sheet ☺

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