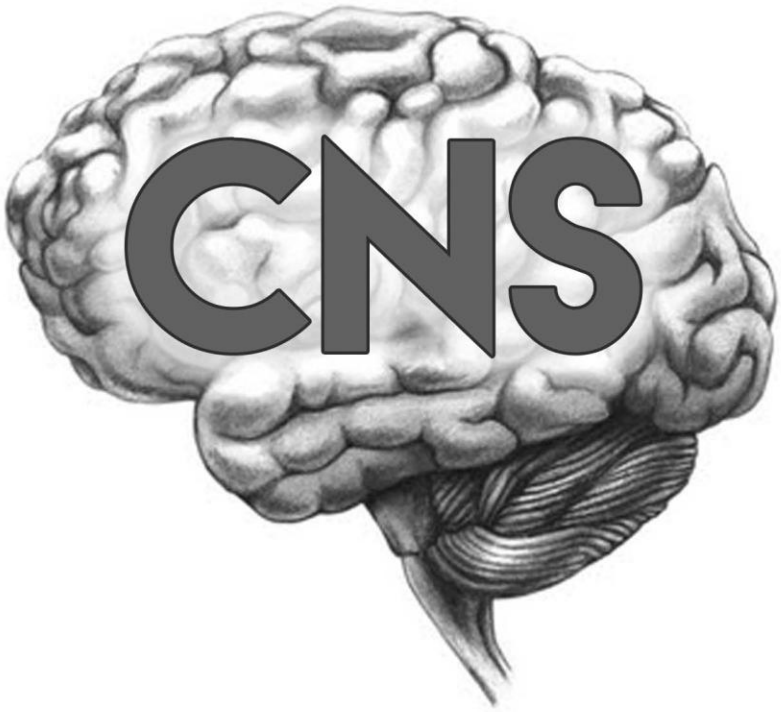


# ANATOMY

☑ Sheet ○ Slide ○ Handout

Number

8

Subject

Cerebellum

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Date: **00/00/2016**

Price:

بسم الله الرحمن الرحيم

This sheet was written according to section 3 recordings , lectures 7 and 8.
The first 6 pages will be complement to lec.7

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- RF: reticular formation

The doctor repeated that area 4 and 6 (both) contribute to the pyramidal tract and extra pyramidal.Pyramidal tract originates mainly from area 4, while extra pyramidal tract originates mainly from area 6.

Area 6 (with its premotor and supplementary parts) gives output to **area 4** ,**brainstem and spinal cord**: area 4 takes information from area 6, and applies the movement. Its effect on brainstem is inhibitory (inhibition to pontinereticulospinal tract). Regarding the spinal cord it gives the **ventral corticospinal tract** (for excitation of proximal and axial muscles) >>>that's why we said that the main function of premotor cortex is **postural preparation for coming movement**>>> because posture needs axial and proximal muscles (ex; if you want to write something on the board, you need to take a certain posture for you to start writing).

A LESION or DEFECT in the **premotor cortex** (the outer part or lateral part of area6) will result in a response called **grasp response**: if you put your finger between the thumb and index and you rub the palm of your patient, the patient will grasp your finger and can't release it. We don't know the mechanism for this response yet, but it happen **only** in premotor lesions. We have to know that it's a normal response in newborn. if it happens later in life >> think about **premotor lesions** .

- The function of **supplementary motor area** is programming, and that includes:
  1)what muscles should be used.

2) In what sequence (inhibit the antagonist, stimulate the agonist ,…)

3) What's the strength of the contraction .

→Supplementary motor area(SMA) to do its programming function , there is an interplay with its partners (basal ganglia and lateral cerebellum) by a **reciprocal** relation: SMA sends output to the lat. cerebellum and basal ganglia directly , but receives input from them indirectly through **VL** nuclei in the thalamus (the secretary ). SMA also receives input from **posterior parietal cortex** (the information storage).
SMA sends output to area 4 ,brainstem and spinal cord, just like premotor area (rem. both of them belong to area 6).

- We have some evidences that SMA participates in the programming:
- When we put electrode in this area we will get a signal just before the onset of the movement.
- When we measure the blood supply, it increases in the SMA before the onset of the movement.
- If we stimulate SMA, we will notice the execution of **complex** bilateral movement involving both hands, and simply complex means we need programming (if SMA injured the patient will face difficulty in handling objects with his **both** hands).

- **MOTOR NEURON DISEASE**:

Motor neuron disease is a collection of diseases affecting neurons. This disease is an autoimmune disease that can affect the spinal cord and therefore causing upper motoneron lesion (UMNL) and lower motor neuron lesion (LMNL), to understand this read the following example:

In the same patient:
1- Affecting the lower part of spinal cord (C6-C8,T1) leads to degeneration in alpha + **gamma** andthe result is **flaccid paralysis** in the muscles of the upper limb with muscle atrophy (alpha and gamma maintain the tone of the muscle through stretch reflex).
2- This disease also can affect the fibers of pyramidal and extrapyramidal tracts -> this will affect the lower limb and we may have **spastic paralysis** (inhibiting the inhibition of pontinereticulospinal tract)

- SO: flaccid paralysis in the upper limb, and spastic in the lower limb. Is it contradictory to what we already have learned about STROKES !!(strokes that result mostly in spastic paralysis in one half of the body; upper and lower limb ).

- AT THE END OF THE MOTOR SYSTEM ,YOU SHOULD BE FAMILIAR WITH THESE TERMS:
  - Monoplegia : paralysis involving one hand or one leg .
  - Paraplegia :paralysis involving both hands or both legs .
  - Quadriplegia: paralysis involving all four limbs.
  - Hemiplegia:paralysis of one hand and one leg on the same side (one half of the body)>>>>commonly seen in STROKES .

## CEREBELLUM

Cerebellum+pons+medulla =hind brain (located in the posterior cranial fossa /infratentorial compartment)

Cerebellum is covered by **tentorium cerebelli** .

- The cerebellum can be divided horizontally by two fissures:

➔ **Primary** fissure(the pointed structure below ) and **posterolateral** fissure (we can't see it).

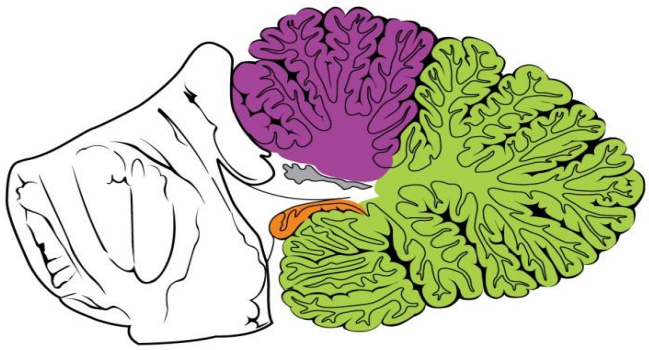
These two fissures divide the cerebellum into three lobes:

**A- anterior lobe**: ant. to the primary fissure .

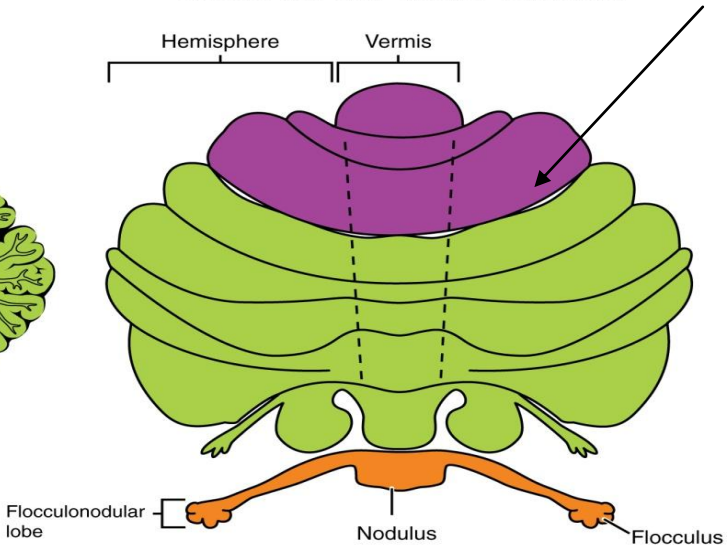
**B-post. Lobe** : post. to the primary fissure (the largest part).

**C- flocculonodular lobe:** small parts behind the posterolateral fissure (flocculus + nodule)

Midsaggital section of cerebellum



Superior view of an "unrolled" cerebellum




The cerebellum can be divided sagittalyto:

1- Vermis

2- Intermediate zone (or **paravermis**).

3- Lateral part. (the largest part of the hemisphere).

Developmentally:

Archicerebellum/Flocculonodular/Vestibulocerebellum( mentioned later in this sheet )

Next part developmentally is **Paleocerebellum** (which is old but not as deep-rooted as archi). Anatomically consists of **vermis and paravermal** region (the anterior lobe); functionally called **spinocerebellum** because it connects with the receptors in the muscles and joints (Golgi tendon organ and muscle spindle) through the spinal cord .

In human beings and monkeys, the **lateral part of the hemisphere** enlarges and becomes **neocerebellum** (neo: new). This part deals and takes the orders from the leader (ie;cerebral cortex) that's why functionally called **cerebrocerebellum**.
This part **Controls the command for the movement before the execution of the commands** . It means that the cerebrocerebellum will review commands and plans that were organized by the premotor area and other components of the motor triangle, and corrects anything wrong before the movement starts.

The hemispheres: **(min 51:04 of record 7)**
1) The outer part is the cortex
2) More to the inside >> white matter
3) Inside the white matter (three pairs of deep nuclei).

The main cell in the cerebellar cortex is **purkinje cell**. Any input that comes to the cortex will go to the purkinje cell, and the output from these purkinje cells will go to the deep nuclei. So the FINAL OUTPUT is coming from the deep nuclei and therefore it's considered the most important structure. Each lobe has its own nuclei.

Three pairs of nuclei that give the final output from the cerebellum:

1) Lateral zone of hemisphere (neocortex): dentate nucleus
2) Paravermalregion :interpositus
   (consists from emboliform and globose - forget about it).
3) Vermis: fastegial .

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✓ There are TWO types of input fibers to the cerebellum : (min 10:40 of record 8 )

1) ***climbing*** fibers : that goes directly to the purkinje .
(e.g. only***olivocerebellary***tract ; as it comes from the inf. Olive of the medulla)

2) ***Mossy***fibers : that goes at the end of its journey to the purkinje, BUT after passing by many stations through granule cells . (e.g. ***corticopontocerebellar / vestibulocerebellum /spinocerebelum.)***
(MOST OF THE INPUT to the cerebellum are mossy ; few are climbing ).

- ✓ so any input must end in **the purkinje** either ;
  - **directly** (climbing)
  - **orindirectly** (mossy)by synapsing with granule cells >to purkinje**.**
- ✓ both inputs are ***excitatory*** ; in their way to the cerebellum (before reaching the purkinje) it will excite the nuclei , so the nuclei receive early excitation from mossy and climbing .
- ✓ when these inputs stimulate the purkinje {directly & indirectly}→

As granule cells excite basket and stellate cells, these cells will inhibit purkinje around them to concentrate the input for the required cell and increase its activity. While Golgi will inhibit the granular cells .Then the neck of the bottle (purkinje)will inhibit the nucleus >>but at the end the nuclei will send ***output*** at anytime .

This output has 3 effects on the muscles:Remember; the effect on αand γ**indirectly**:

1) Very early in the movement: inhibit **the antagonist**.
2) Then**: stimulate the agonist**.
3) When the movement is about to finish**: stimulate the antagonist**.)

Ex; putting your finger at the tip of your nose:
1) Before you start: inhibitsthe triceps.
2) Then**stimulate** the biceps and brachialis (flexors).
3) When it comes to end: what will stop your finger exactly at the tip of the nose and prevent it from reaching your eye or ear is**stimulating the antagonist.**

**Again :basket and stellateare stimulated in order to inhibit purkinje cells lateral to the stimulated purkinje cellsto increase the activity of a certain cell by concentrating the stimulus or input

☞ Now, how does the cerebellum act?

Through feedback and reciprocal way, and that will be explained by two examples:

**1-** The cerebellum has 3 "windows": Superior, middle, and inferior cerebellar peduncle.

One of the input tracts to the cerebellum comes from the reticular formation(reticulocelebellar tract), it enters through the inferiorcerebellar peduncle then goes to the purkinje. Through its way to the purkinje , it will **stimulate** the nuclei . Then the purkinje will send **inhibitory** output to the nuclei.

So, these nuclei will have **early excitation** (by the input tract) and **late inhibition** (by the purkinje output ). There is a balance between them.

At the end, these nuclei will send an output to the same place the input came from (in this case to the reticular formation) .

If nuclei were lost we will have diseases of the cerebellum, that's why they are the most important structures in the cerebellum.

The cerebellum does not deal or react directly with alpha and gamma ; in this case it react through the pontine and medullary reticulospinal tract (Reitcular formation -> medullary and pontinereticulospinal tract -> alpha and gamma. So there is NO cerebellospinal tract).

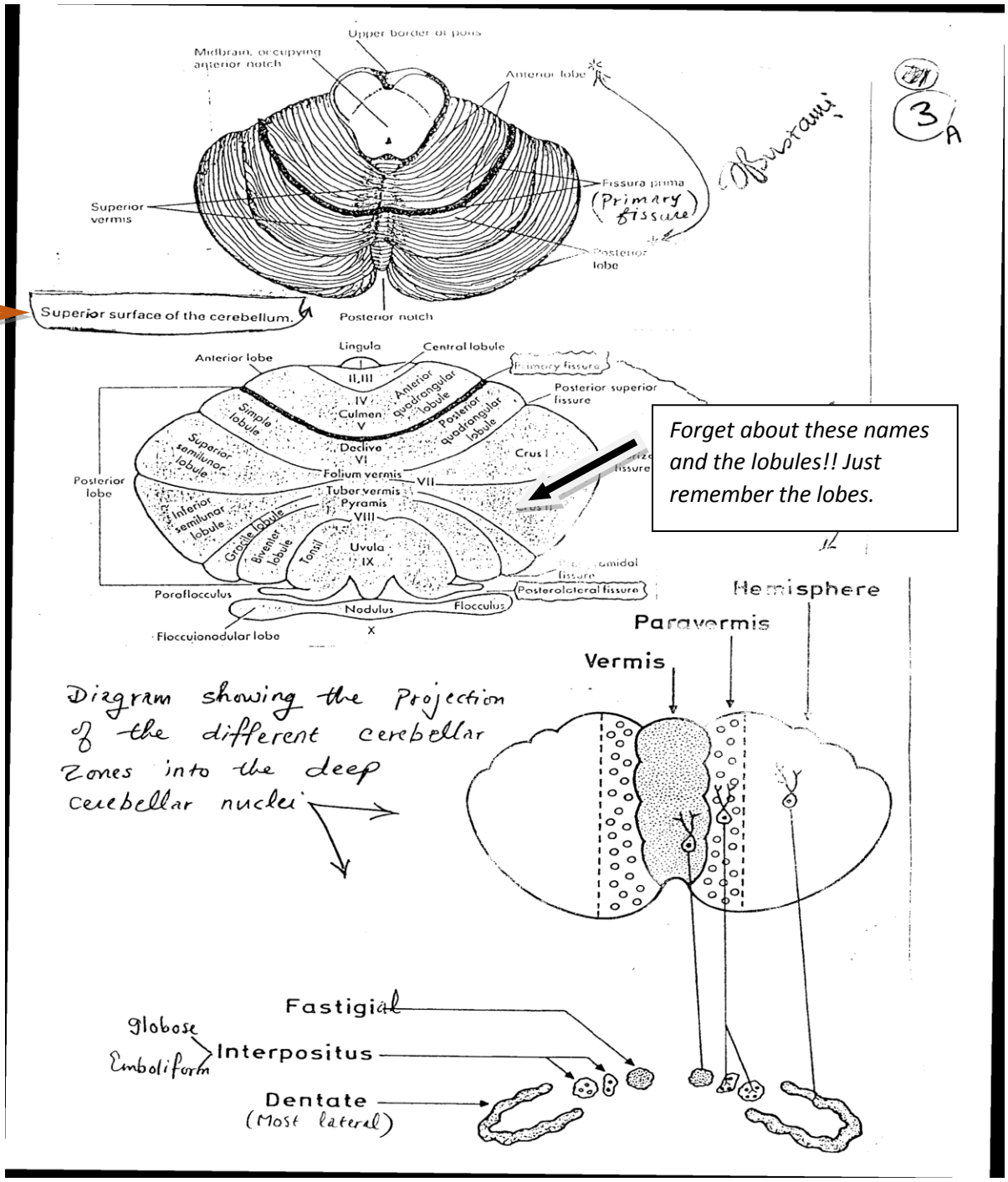
Notice that the**reticulospinal** tract is part of the **medial** motor system .

*Important: The main function of cerebellum is **coordination** between the agonist and antagonist. It stimulates the agonist and inhibits the antagonist. (rem. **C**oordination and **C**erebellum start with ©).*

**2-**fibers from the cerebral cortex to the pons then to the cerebellum (**corticopontocerebellar tract** )through middle cerebellar peduncle :

- First of all ,it gives stimulation to the dentate nucleus then to the purkinje>>to the dentate nucleus >>then the final output will go to the same place where the input comes from (which is in this tract ;the cerebral cortex)

- For example ; if it comes from the association cortex , the output tract will return back to the premotor and motor cortex . and as you know these two areas will send the pyramidal and extra pyramidal tract to deal with alpha and gamma >>so even in this path ,the cerebellum doesn't deal with alpha and gamma directly (it's through motor pathways ).



Now , let's start with lec.8☺
{here is the beginning of record }
Cerebellum is divided ;

- Horizontally into : ant.(spinocerebellum; includes vermis and paravermis) , post. (includes a large part of hemisphere)and flocculonodular lobes.
- Sagittaly into : vermis , intermediate and lateral zones(paravermis and hemisphere)

A. ***spinocerebellum*** : concerned with the FEEDBACK function .
→ {it receives information from the RF and sends it back to the reticular formation/ Also receives from the cerebral cortex and send it back to the cortex.>>>and through pathways from the RF and from the cortex , it reaches alpha and gamma (again , INDIRECTLY)}.

→the 1st tract that reaches the spinocerebellum is,

i. ***dorsal or posterior spinocerebellar*** tract /and this tract starts from ***receptors***(golgi tendon organ through (Ib)fibers/ and muscle spindle through(Ia/II)fibers) >>these fibers will travel with the ***dorsal root*** (rem. Their mother neuronal cells are in the ***dorsal root ganglion*** –without synapse in this ganglion)>>then the central process goes to ***2nd order neuron*** present in the ***nucleus dorsalis of clarke*** .(this nucleus is only present at the level of **C8-L2**).

And this tract starts from receptors in the muscles, joints even in the skin (BUT of the LOWER limb and lower trunk ).

Recall that lower limb afferents go to the sacral and lower lumber segments .right?

And the nucleus dorsalis of Clarke take afferents from (C8-L2)!
The question is : how this nucleus takes afferents from the lower limb below the level of L2 !!

Answer : these afferents will be carried by (***gracile tract*** of the dorsal column ).

- It will synapse within nucleus and gets out from that nucleus(this nucleus called ***transmission cells***).>>and from these cells ,the post. Spinocerebellar tract will arise>>this tract will ascend in the spinal cord in the cervical region then ascends in the medulla and entering the cerebellum through the inferior cerebellar peduncle .

This tract will send information about the actual movement (because it carries afferents from the muscle spindle and Golgi tendon when stimulated, and the spindle gets stimulated when there is stretch in the muscle i.e.Movement in the muscle).

→the 2nd tract is,

ii. ***cuneocerebellar tract*** :it carries similar information to that carried by the post. Spinocerebellar tract (BUT from the receptors of the ***UPPER limb***).

- The mother neuronal cells for this tract is ***accessory cuneate nucleus*** and is found in the medulla .>>>> so, cuneocerebellar tract carries information from the spindle and golgi tendon organ of the upper limb about the actual movement>>(dorsal cuneate tract) then to its accessory cuneate nucleus in the medulla >>then into the cerebellum through inferior cerebellum peduncle .

**note : actual movement: ongoing movement not planned.

→**The last tract is,

iii. ***anterior spinocerebellar***tract :it carries afferents from **golgi tendon** organ (Ib fibers)-golgi gets stimulated when there is increase in tension of the muscle by contraction of the muscle ->>>>then it synapses with lamina**( v/ vi /vii)**>>then it crosses the midline at the level of spinal cord >>then it ascends in the brainstem and crosses again in the midbrain >>then entering the cerebellum through the ***superior cerebellar peduncle*** (**ipsilaterally**).

☞ WHY ipsilateral??
because of the **double crossing** of this tract before it enters the cerebellum.

And this agrees with the fact that each **cerebellar** hemisphere control **ipsilateral**muscles. Opposite to that **cerebral** hemisphere which control contralateral muscles.

**this tract carries information mainly from **interneurons**about the activity of these interneurons ==or information about the ongoing movement .

{rem. That interneurons are of two types (inhibitory and excitatory )/in the flexion reflex we need to stimulate the flexors through excitatory interneurons , and inhibit the extensors through inhibitory interneurons }.

→ so , these three tracts carry information to the spinocerebellum about the actual movement.

The cerebellar cortex

Three layers :

**1)Outer :molecular layer :**in this layer we find:

a)* two interneurons :stellate and basket.

b)*dendrites of two cells :purkinje and golgi cells.

c)* small dendrites of the basket and stellate.
>>so the outer layer is full of dendrites.

d)*and axons of cells in the inner layer (which is granule cells).

**2)Middle:purkinje.{that sends its axons to the nuclei}**

**3)Inner :granule** cell is the main cell in this layer that sends its axon to the molecular layer(1st layer)>>this axon will bifurcate **as "T"** in the outer layer.

- ꕥ It means that the axon of the granule and dendrites of the purkinje in the same layer will**SYNAPSE** (axodendritic synapse).
- It also synapses with the dendrites of the golgi cells and the dendrites of the basket and stellate.
- {in other words ; if you stimulate the granule cell>>all other cells(purkinje, stellate ,basket ,golgi) will be stimulated}.
- note : in the granular layer there are ***gromeruli***(they're synaptic nodules):
  the main cell that contribute to this synapse is the granule cell ,where Golgi also participate in this synapse .

{So , the dendrites of the granule cell participate in the synapse of the gromerulus in the inner layer /and its axon goes to the outer layer and synapse there.}

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B. **vestibulocerebellum: (the only part found in fish ;because they only need balance)

The smallest region, (anatomically) the flocculonodular lobe, is often called (functionally) the vestibulocerebellum. It is the oldest part in evolutionary terms (archicerebellum) {archi:old} , and participates mainly in balance and spatial orientation (the only part of the cerebellum found in fish). {it sends output and receive input from the vestibular apparatus In the inner ear and vestibular nuclei in the brainstem}.

**the input consists of two:

1)***primary*** vestibularfibers(that comes from the balance receptors in the inner ear ; labyrinth ⟶ these receptors are (semicircular canal / utricle and saccule ). Fibershere continue from these receptors through the inf. cerebellar peduncle toward the flocculonodular lobe.

2)***secondary*** vestibular fibers:if these fibers start from the receptors of balance then continue by passing through ***the vestibular nucleus*** vestibulocerebellar lobe.

➡ These receptors of the vestibular nerve get stimulated when the head moves >>and theses fibers will enter the cerebellum as **MOSSY** fibers to stimulate granule cells (of the flocculonodular lobe) and in turn it will stimulate purkinje cells(of the flocculonodular lobe)>>>>>
Then the output of the purkinje will inhibit the nucleus of the flocculonodular lobe.

**WHAT nucleus ??

→ the***vestibular nucleus***{mainly**the lateral** one but also involving (sup./ mid./ Inf.) nuclei} (exception to the rule)>>>this nucleus was in the cerebellum, and during the development, migrated out into the junction between pons and medulla (correct me if I was wrong about the location).

➡ **then the lateral vestibular nucleus sends down the ***lateral vestibulospinal tract*** (part of the medial motor system>>> that stimulate mainly axial and proximal muscles)>>so it will stimulate alpha and gamma of the *extensors(gait and posture*).

**Recall:the function of cerebellum is coordination between agonist and antagonist.**

I.e. when you stand , extensors mainly work; that's why for you to be balanced you need the extensors mostly &coordinate between the axial muscles (muscles of the vertebral column).

⟶ The **lateral**vestibular nucleus also sends fibers to the nuclei of cranial nerves ( CN#3/ #4/#6) through ***MLF*** (*descending and ascending fibers in the brainstem and upper part of the spinal cord*)to deliver information from the cerebellum and vestibular receptors to CN #3 /#4/#6>>these nerves are responsible of ***vestibuloocular reflex***.


→ I.e. if you move your head to the right side >>movement of the head will stimulate vestibular receptors >>and through the ***vestibuloocular*** reflex >> your eyes will move to the opposite side (in this case to the left)

☞ MLF(medial longitudinal fasciculus) coordinate head movement with eye movement.

→ Note:**MLF** is affected early in demyelinating diseases like (**MS**).

{ Recall>MS ; when the demyelinating is affecting the pyramidal tract the result is paralysis}.

➔ **vestibulocerebellum:**

1) **Balance: by controlling axial and proximal muscles.**
2) **Coordinating between eye and head movement (moves opposite toeach other's).**

C. ***Spinocerebellum:(anatomically) vermis and paravermis , regulates axial ,proximal and distal muscles, and its activity during actual movements.***

- 1st input :spinocerebellar tract (ant. & post.): brings information from the muscle spindle and golgi tendon about the actual movement. And it enters as **mossy**fibers.

- 2nd input: olivocerebellar tract: it enters as **climbing**fibers: it should be called (corticoolivocerebellar)>it brings information from the cortex(area6 and association cortex) to the cerebellum through the inf. Olive.

☞ From area 6means: information about the **planned or intended**movement.

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So we conclude that this part(spinocerebellum)compares***between the actual movement and intended movement*** .

If it finds any mistake in the movement or incompatibility with the planned movement **after the execution of the movement**, it will correct it through its output(that exits from*interpositus* nuclei ,sometimes cooperates with*fastegial*)and this output should coordinate between the agonist and antagonist. (axial, distal and proximal muscles)

1)***axial muscles***: here the output is directed to ***the RF***that in turn will send ***the reticulospinal tract***(part of the medial motor system) to regulate axial and proximal muscles.

2)***distal muscles***: here the output is directed to the ***red nucleus*** ;then sends ***rubrospinal*** tract(part of the ***lateral*** motor system) to regulate ***distal*** muscles and part of the proximal muscles.

3) output to the cortexthrough the ***thalamus***>>toward ***area4 ,6*** which send the ***pyramidal*** tract(part of the ***lateral Motor system***)to regulate mainly ***distal*** muscles .

- ✓ so ,the ***spinocerebellum*** affect *axial ,proximal and distal* muscle during the *actual(during the execution)* movement. Just likevestibulocerebellumas they act through ***feedback*** mechanism (receive input and sends output).
- ✓ ***vestibulo***- and ***spino***-cerebellum affect the muscles in a typical ***feedback***,not by *affecting* alpha and gamma directly. إلا عند بعض الناس ☺

***D. Cerebrocerebellum :lateral Part of cerebellar hemisphere , the largest and most important part of thecerebellum::: it acts as feed forward::: it regulate the movement before it happens.***

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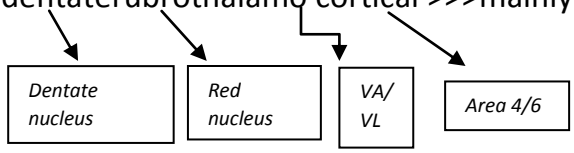
- It's a part of the ***motion triangle*** that *plans* for the movement (area 6 / basal ganglion /cerebrocerebellum)>>>they all participate in the *programming* of the movement.
- it receives input from wide areas of the cortex (sensory ,motor and **mainly *association***)>>so it deals with ideas ; you need an idea before the planning and execution of the movement.>>it deals with ***ideas*** about the movement ***before*** it starts .
- A tract descends from cortex is called (***corticopontocerebellar*** tract)=CPC>>cortical fibers will **synapse** with pontine nuclei >>then pontine fibers will enter the cerebellum through ***middle cerebellar peduncle as mossy fibers.{it brings information about the intended movement}***

☞ The*middle cerebellar peduncle* is formed by the axons of *pontine nuclei* of the ***opposite*** **side**.(so, the mother neuronal cells of MCP are in the pons not the cortex).

- CPC tract enter as mossy fibers>>synapse with granule cells>>to purkinje of the cerebrocerebellum>>>then it gives output to the dentate nucleus which sends output through ***sup. Cerebellar peduncle*** as (***dentatorubrothalamocortical***tract)to regulate the ideas not the movement itself .
- Again: the input through middle peduncle and the output through superior peduncle.
- Injuries to the *superior peduncle* is the most dangerous!!
- dentaterubrothalamo cortical >>>mainly to ***area 6,4***(because they deal with ideas and intentions).




- it acts as feed forward>>so correcting the ideasbefore the execution >>that's why if this tract is not working the result is **cerebellar ataxia**.

☞ ***The worst*** cerebellar ataxia; which results following a lesion in the sup. Cerebellar peduncle>>(because this peduncle carries the correction tract for the ideas) .
(Ora lesion in the red nucleus; as it's a part of this tract).

ꕥ The function of cerebrocerebellum is planning, initiation and timing of sequential movement. So, it's concerned with ***learning and storage*** of ***sequential component*** of motor activity.

{ E.g. typing: needs very rapid flexion and extension of the fingers >>and *the sequence of events for this skilled activity* (typing/drawing) is learned, then stored in the cerebrocerebellum . }

☞ ***Lesions of the cerebellum don't produce paralysis or paresis.***

- Again , Lesions could be cerebrovascular ,tumor ,inflammatory,traumatic ,etc..
- mostly ,especially in the *acute* stage ,produce ***hypotonia***.

→{Recall: The tone is produced by the stretch reflex ,which is produced mainly by gamma,>>what regulate the activity of gamma ?higher centers(area 4,6).>>>so, if the cerebellum doesn't regulate these higher centers (due to a lesion)>>these centers will not regulate gamma>>and gamma is responsible for the tone >>so diseases of the cerebellum will result in hypotonia} .

***Hypotonia*** is due to the absence of the *cerebellar control* of the cerebral cortex , as the cerebral cortex is always under the control of the cerebellum .

☞ Diseases of cerebellum can result in :
A) **disturbance of the equilibrium** . B) **hypotonia.** C)**Asynergia.**

A) The main function of the cerebellum is coordination (for example between the flexors and extensors)>>that's why it's responsible for the equilibrium of the body >>so any disease in the cerebellum will ***disturb this equilibrium***.

B) As we said ,diseases of the cerebellum produce ***hypotonia*** in human beings, while in some animals, there is increase in tone.

C) Also, diseases of the cerebellum produce ***asynergia***:means impaired coordination that result from errors in the rate, force, range and direction of the movement.

***Lesions of vestibulocerebellum: (mostly by tumors affecting mainly children(medulloblastoma) which will hit the roof of 4th ventricle to attack the deep nuclei).***

If these nuclei affected by the tumor (which are the most important structure in the cerebellum )>>**the manifestation** of diseases in the cerebellum will appear:
*(1+2+3 are the manifestations of lesions in the vestibulocerebellum.)

1) Disturbance in the equilibrium : inability to stand without support***:***
***inability to maintain upright posture .***

2) ***Staggering ataxic gait***: with tendency to fall toward the lesion side.
(ataxicgait: *like drunk gait).*

3) ***Nystagmus***: when the patient is trying to fix the gaze(تحديق) on an object, he will have *jerky eye movement* (in which the eye moves slowly in one direction then rapidly on the other direction) .*This happens due to loss of coordination between extraocular muscles.(i.e. loss of coordination between CN#3 and CN#6).*

- *Remember; CN#3 for the medial Rectus >>**adduction** of the eyeball*
  *CN#6for the lat. Rectus>>**abduction**.*

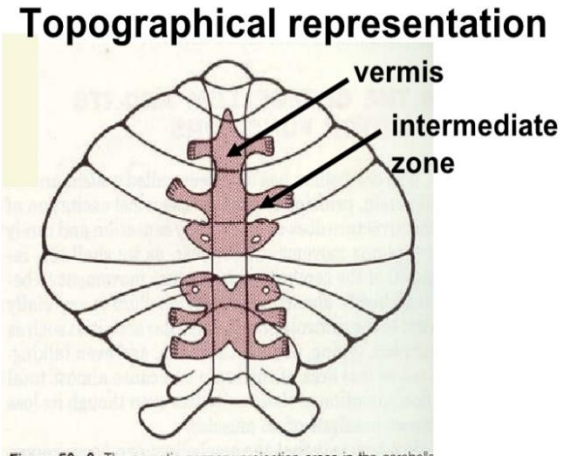
- Note : AS the vestibulocerebellum sends output to the ***MLF***, any lesion in MLF alone will result in ***nystagmus.***

- Clinical case :

*A child came to you with ataxic gait .He also can't stand without support /and he might have nystagmus. A CT showed a brain tumor hitting the roof of the 4th ventricle. what's the most likely diagnosis ?*
*Dx. :medulloblastoma with vestibulocerebellar nuclei lesion.*

## ℘ *topographic representation of the body in the cerebellum:*

- *Ant.lobe*>>the body is represented *upside down* .
- *Post.lobe*>>the representation is *erect* .
- In the *vermal* region>>the representation for the *axial* muscles.
- In the *paravermal* region(intermediate zone)>>the representation of the *limbs and facial region*.
- **In the *lateral zone***>>*NO representation* (it doesn't deal with the actual movement /but with the IDEAS>>>so coordination of sequential pattern of muscular activity).




☞ If you stimulate the cerebellum at any region by an electrode >***no motor movement*** or sensation will result .WHY???

→ *Because the cerebellum doesn't deal directly with alpha and gamma*
*That's why the cerebellum is called the* ***silent area.***

## ℘ *Signs of spino- and cerebro- cerebellar lesions :*

- *old books stated that we can't differentiate between spino- and cerebro-cerebellar lesion. Recently, it's stated that we can differentiate between them.*
- signs are :
  1)***dysmetria***: hand ***overshot*** or ***undershot*** *(either one finger will reach and pass the other finger >>overshot(hypermetria) /or that finger will not reach the other one and stop before reaching the target>>undershot(hypometria)*
  2)***dysdiadochokinesia***: inability to do *two successive* movements.
  (Supination&pronation /or flexion &extension).

☞ These two signs are due ***to absence of proper timing***.

- What stops the movement? The antagonist >>if there was a ***delay*** in the stimulation of the antagonist (It should be stimulated at the end of the movement in order to stop the movement)>>the result is ***hypermetria or overshot***>>you will exceed the target.
- In ***dysdiadochokinesia***: if the agonist -at the beginning of the movement-was ***delayed***>>the *initiation* of the movement will be *slow.*

→so ,***dysmetria***>> delay in the *termination* of the movement due to delay in the intervention of the *antagonist*.

→***Dysdiadochkinesia***>>delay in the *initiation* of each successive movement due to delay in the activation of the *agonist*.

3) ***Intention tremor:***
e.g. during **finger- nose test**>>putting his finger on his nose>>it either stops before reaching the nose or passes the nose(overshot or undershot).

e.g. holding a glass of water >>the patient will try to approach the glass but he can't hold it (because his hand will start shaking >>moving up and down>>tremor)

- *Intention tremor*>>disease of the *cerebellum*>>intention; means tremor that happens when the patient try to do *purposeful* movement.
- *It's absent at rest (it happens only at the end of purposeful movement*). Opposite to that ;***rest tremor***:it happens at rest,due to basal ganglia lesion , i.e.Parkinson's with pill-rolling tremor).
- Intention tremor is mainly due to defective feedback control from the cerebellum on cortically-initiated movement. This tremor is the worst if it result from superior cerebellar peduncle lesions(again and again ; because it sends corrective command about the ideas before the initiation of the movement.)
- Intention tremor : mainly , Is due to absence of the damping function of the cerebellum
- All our motor activities that arise from the cortex are of higher momentum/higher force (more than needed), e.g.In finger-nosetest ,the flexion in the biceps is strong enough to exceed the nose approaching the eye , where the damping function of the cerebellum limits the movement.
- So , when there is a disease in the cerebellum ,>>no damping >>the patient will be pass-pointed (overshot).>>intention tremor.

Thanks to Hassan Saadi