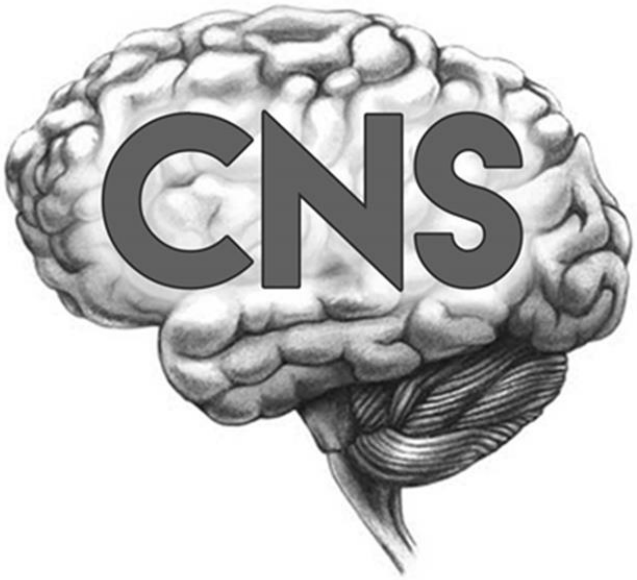


# PHYSIOLOGY

✓Sheet ○Slide ○Handout

Number

6

Subject

The cerebellum

Done By

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Corrected by

Doctor

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Date: 00/00/2016

Price:

بسم الله الرحمن الرحيم

** This sheet was written according to section 1 record (with a different arrangement), slides are included.*

** Lecture outline*:

- **Function of the cerebellum**
- **Summary of Afferents and Efferents**
- **Neural organization of the cerebellum**
- **Cerebellar disorders**
- **Summary of clinical abnormities of the cerebellum**

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## Functions of the Cerebellum

### 1. *Regulation of Equilibrium*

It is the function of the **vestibulocerebellum**, - and part of it is the function of the **vermis**- through the fastigial nucleus.(i.e. when sensory signals are sent to Vestibulocerebellum it will initiate immediate corrective signals and send them to the **vestibular nuclei**, and **RF** to adjust the tone and equilibrate the body.)

### 2. *Regulation of posture*

It is the function of the **vermis** also through the fastigial nucleus.
Remember that posture is controlled by the medial system pathway (which is connected to the axial muscles, antigravity muscles and the extensors); so the **output of the vermis** will control vestibulospinal and reticulospinal tracts-with the tectospinal tract if you're talking about vision- these will regulate the tone and contraction of the antigravity muscles.

### 3. *Regulation (or Coordination) of Voluntary Movements*

It works through different mechanisms:

#### A. Cerebellum is a Comparator

- The cerebellum compares the **intention** with the feedback (the **actual movement**), also it corrects the plan even before the command reaches the skeletal muscle.
- There is short loop of correction between **the cortex**, **the pons**, **the cerebellum** back to **the thalamus** then back to **the cortex**, this occurs specially for **rapid movement** because there is no time for feedback to go and come then correct it.
- A copy of **the intended movement**/the command "*efference copy*" go to the cerebellum through the Cortico- ponto-cerebellar pathway (from the pons)and through the **Ventral** spinocerebellar tract; the one that come from

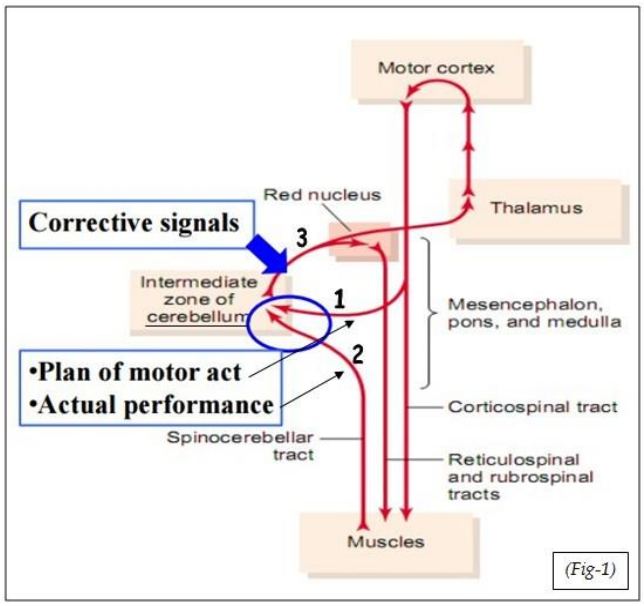
the pons is a copy of the command **directly from the cortex** while the one that comes from ventral spinocerebellar tract is a copy of the signal (the integrated signal) that has to go to the alpha motor neurons. So, in both of them; the cerebellum uses this information to go and do correction right away.

- A copy is coming from the **dorsal** spinocerebellar tract about **the actual movement** in terms of the **length** of the muscle, the **tension**, the **rate** of change in length and the rate of change in tension. The rate is important for the prediction. -better explanation in a bit. -
- Note: the ventral spinocerebellar tract do **not** receive from receptors of the muscles-while the dorsal tract do :D **-** , it receive at the level of alpha motor neurons.
- When there is any "error" in performance or "deviation" from the intended plan; the correction of the signal goes back from the **cerebellum** to the **cortex** then through the **corticospinal tract** to the muscles, or to the **red nucleus** then through the **rubrospinal tract**, the two tracts control movement of the distal muscles/flexors.
- ***(Fig-1)***

1* the way from the cortex to the cerebellum is for plans of motor action

2* feedback from the muscle through the spinocerebellar tract

3* the correction is going back from the cerebellum through the thalamus to the cerebral cortex.




## B. Predictive and Damping Mechanism

- Prediction: Because the cerebellum knows the rate / velocity and the direction; it calculates how much time is needed to reach the destination, then it sends messages to the motor cortex to stop the ongoing movement at the intended position. Why? Because-again- **the cerebellum knows from the rate how much time is needed to reach the destination**.

## C. Planning the Sequence and Timing of Movements

- **Planning** for sequential movements, rapid movements that don't wait for the actual feedback coming from the muscle, also planning for timing, onset and termination especially for programmed movements. These plans are coming from the **cerebrocerebellum**.
- Timing of movement: the **cerebrocerebellum** will find the perfect timing and this is established by computing (calculating) the "**onse**t" and "**termination**" before the movement comes (i.e. adjust the final movement before it is discharged to the lower motor neurons).

### *4. Role of the Cerebellum in Motor learning*

- **Training** is the function of the climbing fibers that comes from the inferior olive, the example of training is **writing**; in the first time the movement is cortical, when the child begins to write he will press hardly on the pen and tear up the paper, here the program is not fine, how it becomes fine? By **learning**, the program is going from the cortex to the cerebellum →the next time it's finer and finer...then it is becoming a **"stored program"** for each learned skill, then the skill will be very fast.
- *From the slides*: *When a person **first** performs a complex motor act, the degree of **cerebellar adjustment** of the "onset" and "termination" of the successive muscles contractions involved in the movements is **almost always inaccurate**, then cerebellar neuronal circuits **learn** to make more accurate movement the next time.*

### *5. Role of the Cerebellum in Rapid and Ballistic Movements*

- *From the slides*: *These movements include writing, typing, talking, running, and many other athletic and professional motor skills.*
- These movements happen so rapidly that is **impossible to depend for their control on the feedback**, so it has to be **corrected in the short loop(**from the **cerebellum** to the pons back to the cerebral **cortex** through the **thalamus)not** through the long loop when we wait for the actual movement to come back from the periphery.

## ❁ Summary of the Afferents and Efferents

### 1. *Afferent Pathways to the Cerebellum*

#### A. From the brain

- They are coming mainly from 2 areas to the cerebellum either through the **Cortico-ponto-cerebellar** tract from the **pons**& from the motor, premotor and sensory areas of the **cortex** and it goes to the lateral hemisphere of the cerebellum.
- Or through **vestibulocerebellar** tract, **reticulocerebellar** tract and from the inferior olive through **olivocerebellar** tract.

***These pathways transmit information about intended motion.***

#### B. From the periphery

- **Dorsal spinocerebellar tract:** it's **uncrossed tract** so bring information ipsilaterally. it receive signals from **muscle spindle, Golgi tendon, all large tactile receptors** around the joints to tell the person about the position of the joints is it flexion or extension, and how much flexion or extension and so on .
- **Ventralspinocerebellar tract:** it's a **bilateral tract**. It receives the efference copy from the **alpha motor neurons**.

### 2. *Efferent pathways from the cerebellum*

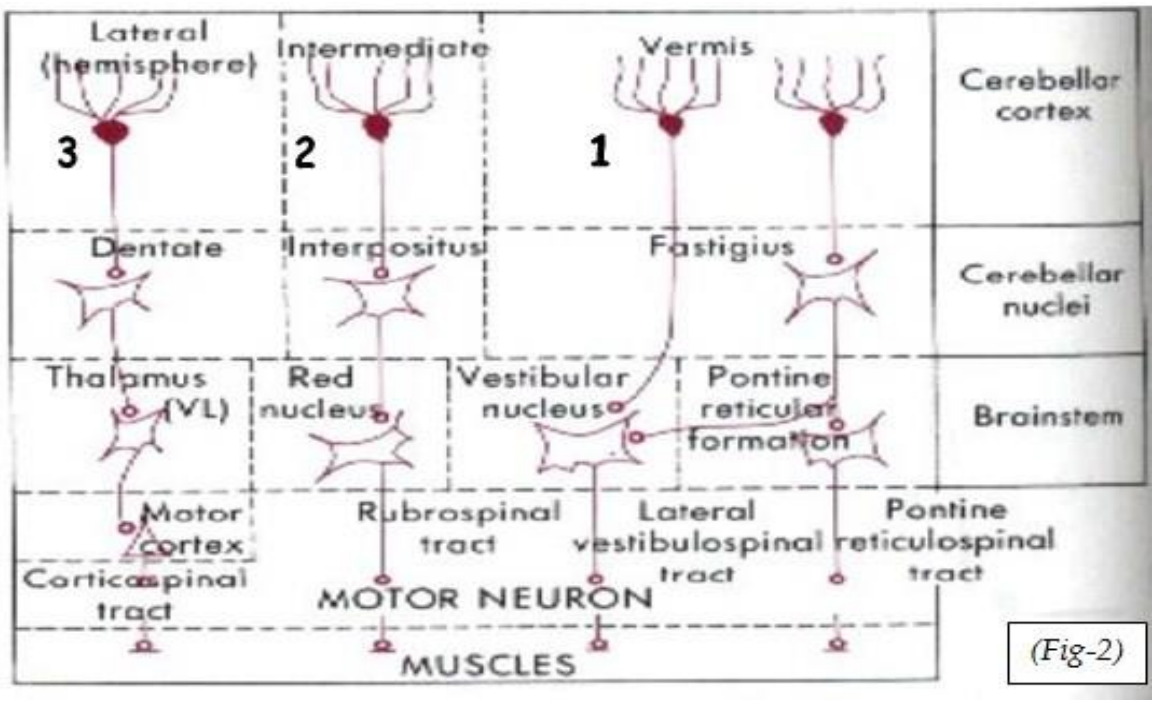
- They have to go out from the deep cerebellar nuclei, remember:

✓ The deep cerebellar nucleus of the **vermis and flocculonodular** lobe →**fastigial nucleus**

✓ The deep cerebellar nucleus of the **intermediate zone**→the **interposed nucleus** (interpositus) with its 2 parts; Emboliform and Globose.

✓ The deep cerebellar nucleus of the **lateral zone**→ the **dentate nucleus**.

- From the **vermis**: fastigioreticular tract and fastigiovestibular tract; for **equilibrium control**.
- From the **intermediate zone**: Interpositorubral tract to the red nucleus and interposito-thalamic tract to the thalamus**.**
- From the **lateral hemisphere:** dentatothalamocortical tract**.**




*(Fig-2)*

- **(*Fig*-2)**

  **1***Fibers pass from the vermis, to the fastigial nucleus, then to the **pons** or to the **vestibular nucleus**, then as the lateral vestibulospinal tract. Notice: there are some fibers from the cortex to the cerebellum that go to the lateral vestibular nucleus **directly** without passing through the fastigial nucleus; this means that **the lateral vestibular nucleus works functionally -at least- as a deep cerebellar nucleus**, because it receive direct input from the cortex and the cerebellum.

  **2*** Fibers from the intermediate zone go through the interpositus nucleus, to the red nucleus, than as rubrospinal tract**.**

  **3*** Fibers from the lateral zone go to the dentate to the thalamus (VA & VL) to the motor cortex then as corticospinal tract.

  » Even the intermediate zone, which serves especially for the distal muscles, fibers go to the thalamus, to the cortex then to the corticospinal tract. *Remember the corrective signal fig 1.*

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## ❀ Neural organization of the cerebellum

### ✓ *Neural organization of the cerebellar cortex*

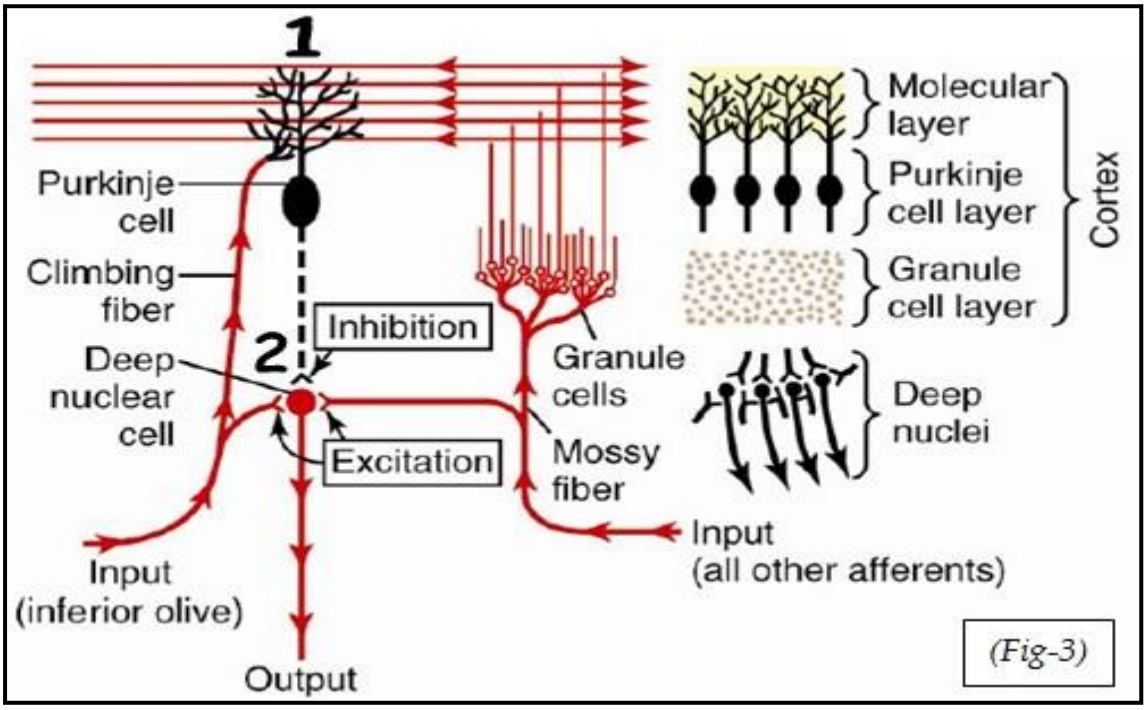
- Microscopic structure or histological structure show three layers of the cerebellum; which are:

Molecular cell layer: it has the parallel fibers, Purkinje cell layer and Granular cell layer. (The 3 layers together →the cerebellar cortex).

- The last layer deep inside is <u>the deep nuclear layer</u> which contain the deep cerebellar nuclei.
- The afferent fibers that comes to the cerebellum are of 2 types; either **climbing fibers or mossy fibers**.

All the climbing fibers come from the inferior olive (the fibers coming from the inferior olive are called **<u>climbing fibers</u>**).

All other fibers that come to the cerebellum from all parts (from dorsal, ventral spinocerebellar tracts ...) they are forming what we call **<u>Mossy fibers</u>**.




✓ ***(Fig-3) the configuration of functional cerebellar unit*:**

- 1* the **<u>dendrites of purkinje cells</u>** lay in the <u>molecular layer</u> with the **<u>parallel fibers</u>; which are the axons of the granular cells**, i.e. the **axons** of the granular cells **run perpendicularly** to the right or the left forming the parallel fibers (*they **divide** and go a few mm in opposite directions to become parallel fibers*).

  2* the **axon of purkinje synapses with the deep cerebellar nuclei cells**, and from the deep cerebellar nuclei cells the Efferents go out from the cerebellum.

  3* notice the climbing fibers and mossy fibers which are **<u>excitatory</u>**.

- The climbing fibers and mossy fibers **excite** the deep cerebellar nuclei, so the deep cerebellar nuclei **at first** receive **excitatory** inputs to the agonist, after a while the climbing fibers goes and excites the **purkinje fibers** which **are inhibitory**, when the purkinje is stimulated by the climbing fibers or by the

parallel fibers; it goes down and **inhibit the deep cerebellar nuclei**, so the deep cerebellar nuclei first receive excitation then inhibition, excitatory to the agonist and inhibitory to the antagonist.

- So, this is how it works, first excitation then inhibition then excitation then inhibition:
  - ✓ **Excitation of the agonist with inhibition of the antagonist**
  - ✓ **Then inhibition of the agonist with excitation to the antagonist**→ this is called ***reciprocal innervations***: activation of the **agonist** with inactivation of the **antagonist**, and inactivation of the **agonist** with activation of the **antagonist**, this how rapid movement is run down.
- ***So, the deep nuclear cells receive excitatory and inhibitory inputs***:
  1. Excitatory afferent input from **climbing fibers** and from all other fibers
  2. Inhibitory from **Purkinje cells**.

**Notes**:

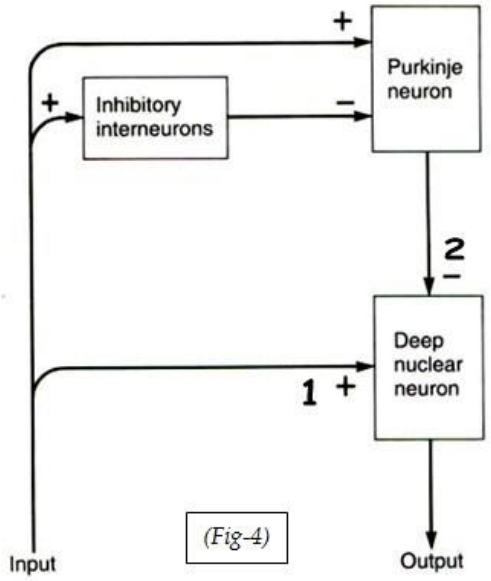
- The granular cells are excitatory and **purkinje cells are inhibitory**.
- Any Movement needs **agonist** and **antagonist**, stimulation of the agonist first, then stimulation of the antagonist to stop the movement.
- There are some interneurons **inside the molecular layer**, these interneurons are **lateral inhibitory neurons** (because they do lateral inhibition), and this is for ***sharpening*** of the signal, i.e. the center is stimulated and outside is inhibited. These of 2 types: ***basket cells*** and ***goblet cells***.

- **Climbing fibers** action potential is **complex** spiky potential, *they send branches to the **deep nuclear cells before** they make extensive connections with the **dendrites of the Purkinje cell.**(follow the course of these fibers on fig- 3 to get this)*
- **Mossy fiber** action potential i**s simple** spiky potential. The **granular cells** are excitatory and they **receive their inputs from mossy fibers**; and these fibers also excite the deep cerebellar nuclei.

- **The inferior olive is important for learning**, why? Because when it goes and stimulate the deep cerebellar nuclei and stimulate the purkinje fibers **it changes/alters the sensitivity** of purkinje cells (their action potential or their excitatory postsynaptic potential), so **the purkinje cells learn from the previous excitation** that come from the climbing fibers, so the next time its sensitivity is changed or altered. So by this way the purkinje cells learn the next time.

- That's why the inferior olive is very important; also, because it receives feedback, so it works as if it's **a cerebellum** by itself → it **sends excitation** to the deep cerebellar nuclei and it **receives feedback**, so it do **comparison** between the actual and the intended function; it receive Corticospinal and sensory from the muscles. So, if there is a mismatch; it corrects it, and send it through the climbing fibers. (Some signals pass with correction, other without it).

### ✓ *(Fig-4) Deep nuclear cell activity*



(Fig-4)

- 1* the inputs are excitatory to the deep cerebellar nuclei, so first there is excitation by both climbing and mossy fibers.
  2* this is followed by inhibition from the purkinje cells -which is inhibitory-.
  - » **Normally the balance is in favor of excitation**.
- so, this is how it works:
  First, the intended movement come from **corticospinal tract** to the **muscle**, then back through the **climbing fibers** from **the inferior olive** to **the deep cerebellar nuclei** then goes back to **corticospinal tract** (through the thalamus then to the cortex). It will enforce the force that is done by the corticospinal tract, so the ***tone is increased*** by this mechanism.
  → *Direct motor pathway (via corticospinal tract) is **enhanced by cerebellum**, by **additional signals to the tract** or by **signals back to the cortex.***
  That's why if the **cerebellum is diseased** or destroyed there is **hypotonia**; because the **tone that comes from the cerebellum** to enforce the tone that is coming from the corticospinal tract **is lost**→ hypotonia.

### ✓ *The Turn-On / Turn-Off Function(the doctor read the following)*

- *At beginning of motion there are excitatory signals sent into motor pathways by deep nuclear cells to enhance movement, followed by **inhibitory signals milliseconds later.***
  - *Provides a **damping function** to **stop** movement from overshooting its mark*
  - *Resembles a delay-line type of electronic circuit for negative feedback*
- *Cerebellum contributes to the rapid **turn-on signals for agonist** muscles and **turn-off of antagonist** muscles at beginning of a motion.*

- *Then it **times the opposite sequence** (**turn-on signals for antagonist** and **turn-off of agonist**) at the end of the intended motion.*
- *Mossy fiber input also to **Purkinje cells** which **activates** them after a few millisec. , this results in an **inhibitory signal to the deep nuclear cell (by the purkinje)**this **inhibits** the **agonist** muscle **which stops the movement.***
- **So, agonist activation is directly from the deep cerebellar nucleus, while the inhibition of the antagonist is coming from the purkinje.**

***That was a trial trying to explain what happens, but nothing is 100% accurate.***

### ✓ *Motion Control by the Cerebellum*

- Cerebral cortex is inexact, most of its **motions** are **pendular** and have **inertia** (it don't stop on the intended position), what's going to stop it? **The cerebellum**, it will damp the movement (stop it at the right position).
- To move a limb accurately it must be **accelerated and decelerated** in the right sequence, **cerebellum calculates momentum and inertia and initiates acceleration and braking activity.**
- What happens without the cerebellum(when its destroyed):
  - The movement remain ***pendular***; the leg will **keep on moving forward and backward**(like a pendulum) when we tap the patellar tendon (knee jerk).
  - ***Past pointing***; while the patient is closing his eyes, he cannot put his thumb on the tip of the nose, he passes it.
    (He doesn't have cerebellar correction).
  - ***Rebound phenomena****(Explained later in this sheet)***.**

- Remember the **predictive function** of the cerebellum, which is to **calculate the time that is needed to stop your movement at the intended position**.

- The lateral cerebellar hemisphere communicates with **premotor** and **sensory cortex** and corresponding area of the **basal ganglia** where the **plan originates**. **(**where does the plan of the movement occur exactly is not known, but action potential and activity of the cerebellum and **basal ganglia** were recorded **before the movement occurs, so certainly the basal ganglia have a function in the plan of movement)**
- The lateral hemisphere **receives** the plan and **times** the sequential events to carry out the planned movement, remember: **cerebrocerebellum** is important for **rapid and ballistic movement**.

## ❁ Cerebellar Disorders

### 1. *Flocculonodular lobe (vestibulocerebellum) disorders*

It is manifested by **disequilibrium** (sometimes with nystagmus); the patient is:

A. **Swaying down** during standing with a tendency to fall down, the patient is unable to balance and unable to maintain equilibrium.
B. **Having Unsteady (staggering, drunken) gate,** it's tested by ***the tandem test***(heel to toe test). When the traffic police suspect an alcoholic driver, they test them by asking the driver to walk heal-to-toe in a straight line; he won't be able to do that if he's drunk.

### 2. *Vermal disorders*

It is manifested by **inability to maintain the upright standing posture** due to **failure to adjust the tone**→ disequilibrium**.**

### 3. *Neocerebellar syndrome*

» It results from vascular **strokes**, **degenerative disorders**, or**tumors**/neoplasm.

» It is manifested by: hypotonia, asthenia, and ataxia.

#### A. Hypotonia

- Remember: when the deep cerebellar nucleus is excited by the climbing fibers and mossy fibers, it sends these excitations back to the corticospinal tract ***to enhance the tone*** so, when the cerebellum is diseased →decrease in the **tone** of skeletal muscles due to decreased **facilitation of the γ motor neurons** as a result of decreased **supraspinal facilitation**.
- It's associated with ***hyporeflexia***→ decreased stretch reflex.
- ***The knee jerk becomes pendular*** like the pendulum; it doesn't stop because the cerebellum is what does calculation to stop it.

When we take our physiology lab we will learn sensory and motor examinations, one of these examinations is reflexes.
We-as beginners- won't be able to tell if this is hypo-reflexia or hyper-reflexia, but by experience we will know that **++** is ***normal*** reflexia, **+ or zero** is ***hypo*** reflexia and **+++** is ***hyper*** reflexia (This is how the deep tendon reflexs are graded)

#### B. Asthenia

There is **weakness** because of enhancement loss, the **muscles will fatigue** more readily than normal, **as a result of interruption of the activating effect** of the cerebellum on the cerebral cortical motor areas.

### C. Ataxia(or Asynergia)

Cerebellar ataxia can manifest itself in many ways:

#### 1) Dysmetria

- **Errors in the range and direction of the movement**, because of improper measurement of the distance, **i.e. abnormal calculations** in the higher range or in the lower range**.** That will result in ***past pointing or hypermetria***→ the patient will **"overshoot"** while doing the thumb – nose test because the movement is cortical, but if there is a **cerebellum** it will **break** it → no overshot, it will **stop** at the intended position.
- So these errors result from **failure of the "comparator" and "damping" functions of the cerebellum** that normally adjust the course of the movement and bring it smoothly to the desired position.

#### 2) Intention (action/ kinetic) tremor

- When the patient **extends** his arm, the cortex will think that the movement is very rapid so the patient will **take his arm back**, which is also seen to be very rapid then the patient will try to **extend** his arm again and **so on**.
- The cerebellar patient is **normal without movement** and he will have these **tremors when doing a movement (*i.e. they appear when the patient performs a voluntary motor act, and not seen when the muscles are at rest*).**
- **Note: basal ganglia patient (e.g. Parkinson patient) will have *resting tremors*, it disappears while doing a movement.**

#### 3) Decomposition of complex movement

- *The motor action is carried out as **several fragmented steps** rather than a smoothly progressing movement.*
- *For instance, in reaching for an object by the hand, the cerebellar patient may first move the **shoulder joint,** then the **elbow**, followed by the **wrist** and **fingers** → **simulate movements of a "robot"**.*
- Again, the cortical movement is **slow** (the patient moves the **shoulder** then the **elbow** then catch the cup for example), so the movement is **decomposed/broken down** (as the slow motion of a robot) because there is **no cerebellar control.**
- **Talking** is a rapid movement, (rapid contraction of agonists and antagonists), when it's decomposed the patient will have **slurred speech→*dysarthria*** (dys: abnormal, arthria: articulation).–refer to point 7 next page.

### 4) Rebound phenomena

- The cerebellar patient is **unable to stop the ongoing movement rapidly** due to **failure of the predictive and damping functions** of the cerebellum. This can be observed in what is called ***"rebound phenomenon".***
- When there is a flexion of the forearm against resistance (provided by the examiner's hand), the cerebellar patient **cannot stop the resultant inward movement** of his limb in due time following its release, and the forearm flexes forcibly and may **strike his body with considerable violence.**

(امسك يد المريض واحكيلو يشد , ولما تفلت يدك فجأة بترتد يد المريض عليه و بيضرب حاله لأنه ما قدر يوقف الحركة ).

### 5) Dysdiadochokinesia

- The **inability** of the patient **to perform rapid alternating opposite movements (*e.g. rapid repetitive pronation and supination of forearm*)**
- The movements will be slow and irregular. But if he cannot do it at all →**Adiadochokinesia**
- It results from failure to adjust precisely the proper **timing** for the onset and termination (Inability to calculate the time).

### 6) Nystagmus

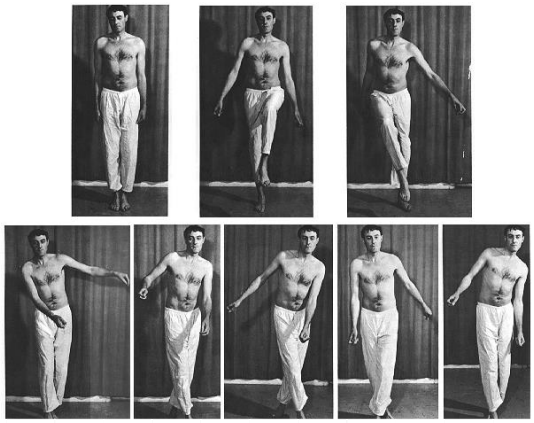
- Cerebellar nystagmus is a tremor of the eyeballs (rapid eye movement) as a result of "dysmetria" of the eye movement.

### 7) Scanning Speech (Dysarthria)

- It's the **inability to connect the words together**; each word is **fragmented** into several syllables, *producing "scanning" or "staccato" speech, like someone trying to speak an obscure foreign language for the first time.*

### 8) Unsteady Gait

- The gait is broad-based due to dysmetria and kinetic tremors of the lower limb muscles.



## ❁ Summary of Clinical Abnormalities of the Cerebellum

- All signs of cerebellar diseases are ***ipsilateral*** since there is double crossing- from cortex to pons and back to cortex.
- **Basal ganglia diseases are *contralateral*.**
- Ataxia and intention tremor
  - Failure to predict motor movement, patients will overshoot intended target, ***past pointing***.
  - Dysequilbrium- ataxic (staggering) gait (drunken gait)
- Dysdiadochokinesia (Adiadochokinesia)
  - failure of orderly progression of movement
- Dysarthria
  - Failure of orderly progression in vocalization
- Cerebellar nystagmus
  - Intention tremor of the eyes when trying to fix on object.

❁ ***Neurology is very easy, if you see a patient with unsteady gate, intention tremor, dysarthria, or nystagmus →cerebral disorder.***

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***That was it, good luck :D***

***Shout out to Sanal, Nadia and the squad<3***