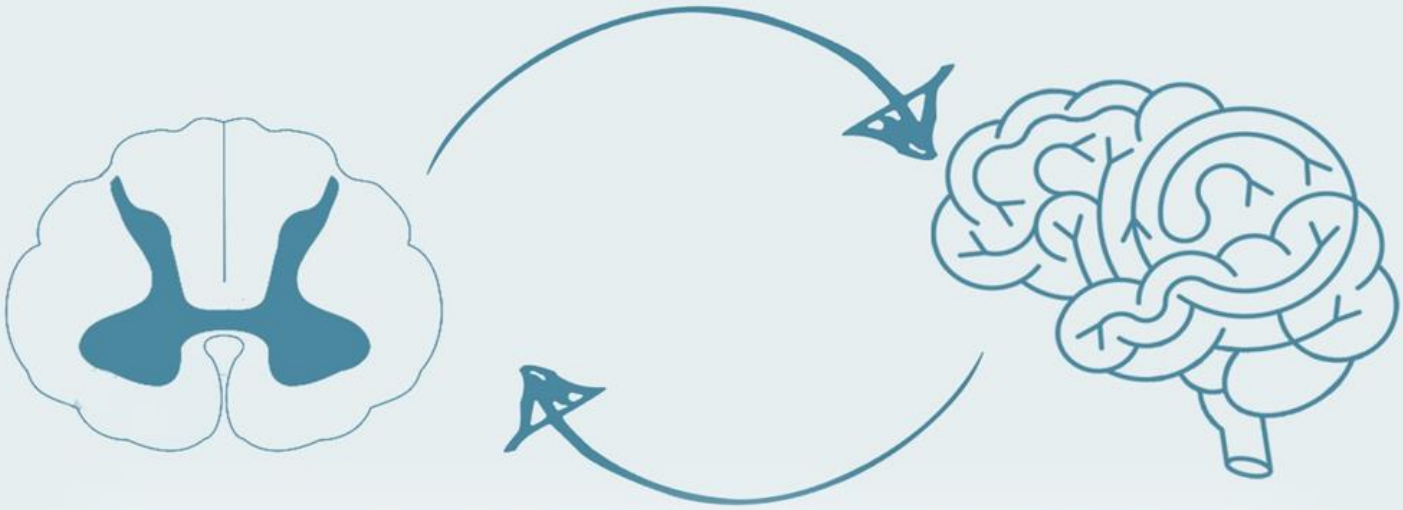

CNS

Anatomy

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number	7
Done by	Mai Ababneh
Correction	Abdullah Nimer
Doctor	Mohammad Al-Salem

Last lecture was about pyramidal and extra-pyramidal tracts.

This table contains a comparison between **Upper Motor Neuron (UMN)** lesions & **Lower Motor Neuron (LMN)** lesions:

COMPARISON BETWEEN UMN AND LMN

Features	Upper motor neuron lesions(UMN)	Lower motor neuron lesion(LMN)
	UMN starts from motor cortex to the cranial nerve nuclei in brain and anterior horn cells in spinal cord	LMN is the motor pathway from anterior horn cell(or Cranial nerve nucleus)via peripheral nerve to the motor end plate
Bulk of muscles	No wasting	Wasting of the affected muscles (atrophy)
Tone of muscles	Tone increases (Hypertonia)	Tone decreases (Hypotonia)
Power of muscles	Paralysis affects movements of group of muscles Spastic/ clasp knife	Individual muscles is paralyzed Flaccid (flaccid paralysis)
Reflexes	Exaggerated. (Hyperreflexia)	diminished or absent. (Hyporeflexia)
Fasciculation	Absent	Present
Babinski sign	Present	Absent
clasp-knife reaction	Present	Absent
Clonus	Present	Absent

***Remember:** the **upper motor neuron** ends at level of the anterior horn cells of gray matter, while the **lower motor neuron** starts from anterior horn cells to skeletal muscles.

- Most of time pyramidal and extra-pyramidal lesions **co-exist**, it is very rare to find a pyramidal lesion without an extra-pyramidal lesion
- **Hypothetically:** A pyramidal lesion results **in hypotonia and hyporeflexia**, while an extra-pyramidal lesion results **in hypertonia and hyperreflexia**. But in real life they usually co-exist, eventually the stronger effect of **extra-pyramidal lesion appears on patient** (hyperreflexia & hypertonia).

So, if we have an **UMN lesion**, it'll result in **hypertonia**, even if pyramidal is involved (most of cases).

- **Hyperreflexia and hypertonia** are caused by the increase in **gamma motor activity**, an increase in the tone (hypertonia) will cause an increase in the reflexes (hyperreflexia).
- Recall: gamma and alpha motor activity (lecture 6)
  Gamma activates the muscle fibers in the spindle
- Most of time the effect of UMNs on gamma motor neurons is an inhibitory effect, so an upper motor neuron lesion will lead to hyper-excitability in gamma motor neurons → hyperreflexia→ hypertonia→ spasm\rigidity.
- Note that rigidity and spasm differ from each other, explained later
- **Bulk of muscle (size),** as seen in table:
  - **UMN lesions** does **not** cause wasting, because there is hyperreflexia+ hypertonia → muscles are working and contracting so there is no wasting (no atrophy).
  - **LMN lesions** will cause **wasting** of the muscles, muscles are paralyzed (**flaccid paralysis**), no nerve supply to muscles→ no contractions→ wasting (muscle atrophy).
    This could be seen in hospitalized patients not moving for a long time, not using the muscle for a long period of time causes **atrophy**, those patients need **physical therapy** before discharged.
  - Atrophy\muscle wasting is found in LMN not UMN lesions.

*note -> all clinical cases we took in upper limb anatomy are examples on LMN lesions:

- ✓ Radial nerve injury: rest drop
- ✓ Axillary nerve injury: <u>deltoid atrophy</u>, clinically: the greater tubercle becomes palpable (in normal cases you can't feel the greater tubercle of humerus because the deltoid is covering it but in case of deltoid atrophy it can be easily felt).

- **Power of muscles**: both lesions causes paralysis.
- The word paralysis has a general nonspecific meaning: شلل, smaller lesions causes **paresis** (weakness).

- Upper and lower MN lesions causes different kinds of paralysis:
    - UMN → hypertonia leads to **Spastic rigidity\paralysis** (spasticity: (مخشبة. Because of the increased gamma motor neuron activity.
    - LMN → no nerve supply -> **Flaccid paralysis** (مرتخية)-> with time: atrophy, wasting (size decreases).

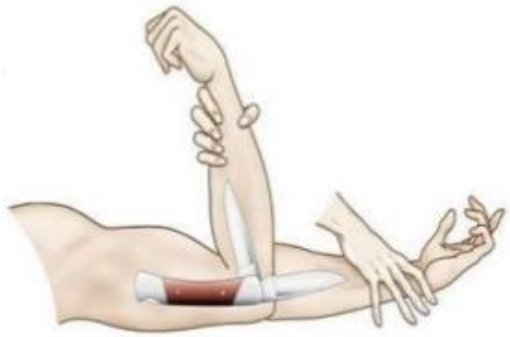
- **Rigidity and spasticity:** commonly people use these two words interchangeably to refer to the same meaning, but they are different:
    - **Rigidity:** means paralysis in 2 opposing groups of muscles (both flexors and extensors\ bidirectional). Found in Parkinson.
    - **Spasticity:** paralysis in one direction\ one group of muscles (either extensors or flexors). That's what we find in case of UMN lesions.

-> Clasp knife reaction:

UMN lesions cause **clasp knife spasticity.**

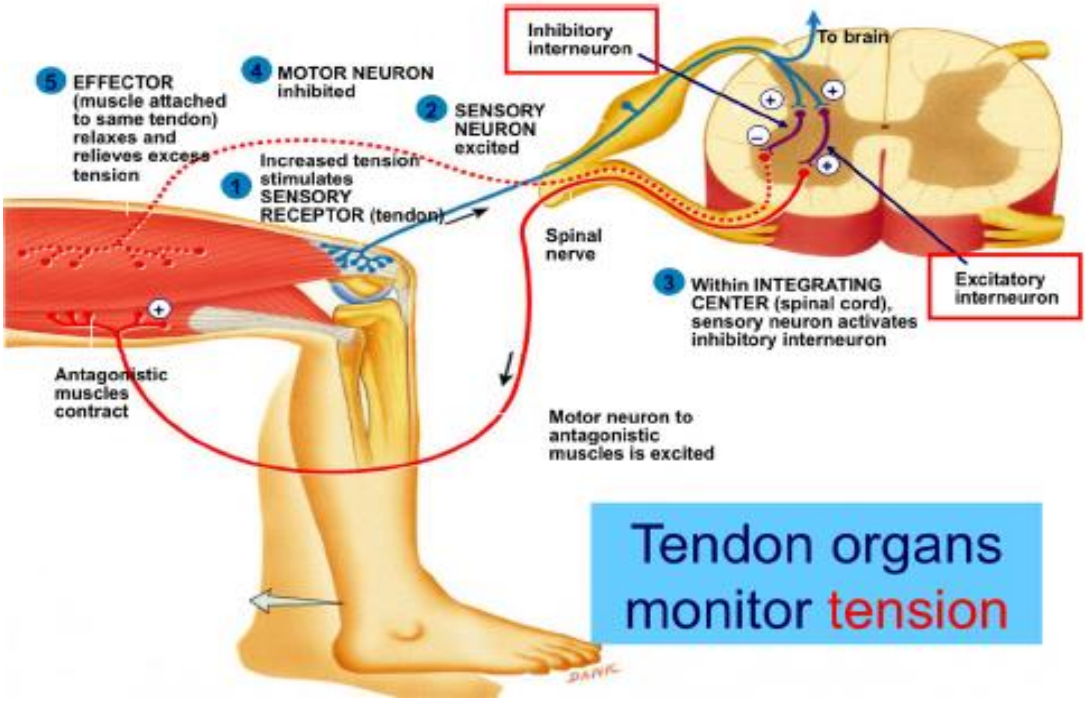
During a neurological examination, the arm of patient is flexed and you're trying to extend it, first there will be some **resistance** for extension (arm tend to stay flexed), but at some point, the resistance stops, and the arm is **suddenly released**.

Sudden extension of the arm, same concept as a clasp knife.



- ✓ Happens in **Decerebrate** and **Decorticate spasticity**. (explained later).
- ✓ Clasp knife spasticity: The upper limb resists **extension and flexion** while the lower limb resists **flexion**.
- ✓ Clasp knife: extra-pyramidal pontine.

- ✓ Explanation:
    - A. **Initial resistance:** due to exaggerated stretch reflex, the muscle refuses to stretch.
    - B. **Sudden release:** due to Golgi tendon reflex (explained in physiology)

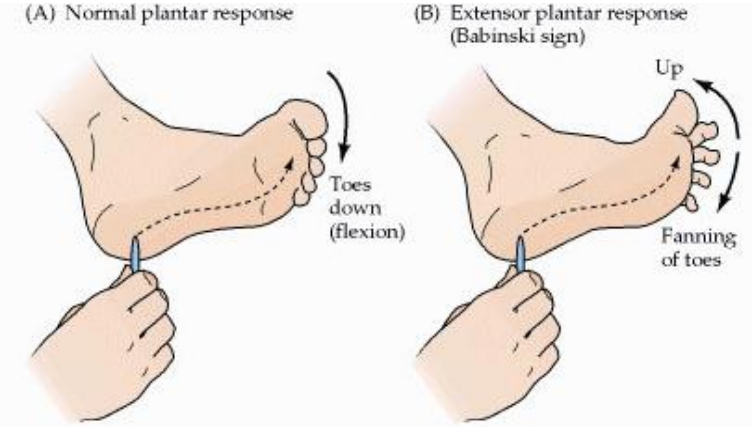


As seen in pic.

Inverse stretch reflex\ Golgi tendon reflex.

1. Receptors are found in the tendon, when quadriceps **contract**, the tendon tension increases.
2. Sensory neuron stimulated, signals go to the brain (blue ascending arrow).
3. Activation of interneuron (excitatory and inhibitory).
4. **Inhibitory signals** will go to quadriceps (same muscle) telling the muscle that it contracted too much and it is time to relax.
5. At the same time there will be an **excitatory signal** to the antagonistic muscle (hamstring muscle) to contract (antagonist muscle contract) according to Law of reciprocal innervations.

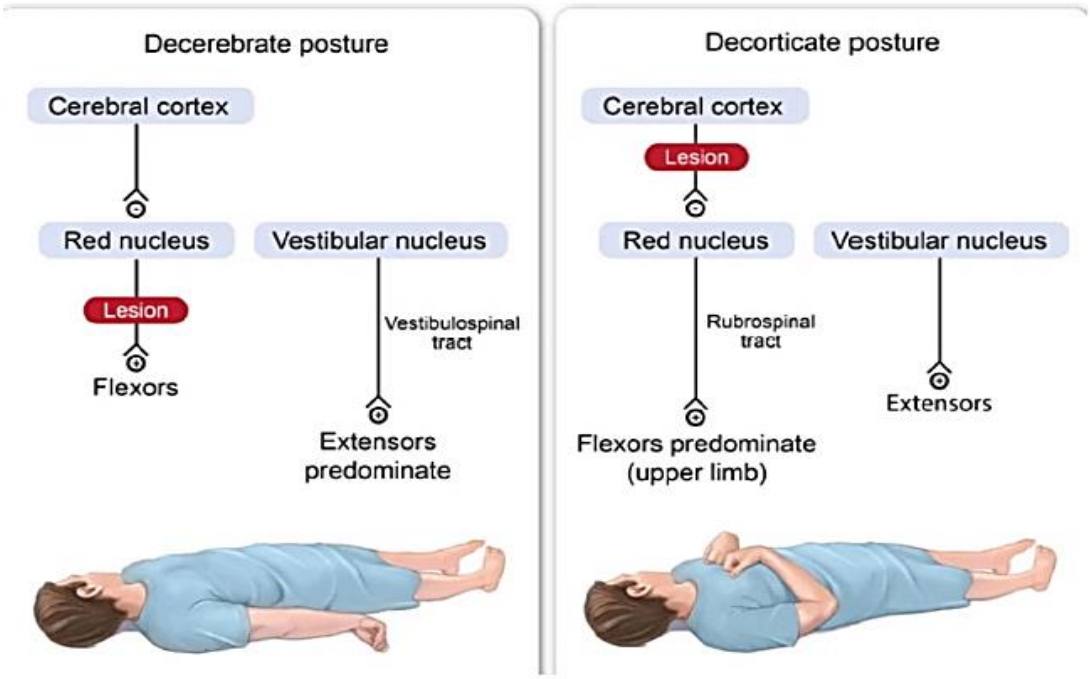
* Babinski's sign:



- **Babinski sign is specifically related to the pyramidal pathway**.
- If you stimulate the planter side of foot by an object, the normal response in adults is **planter flexion**, but in case of an **UMN lesion** (pyramidal) the patient responds by **planter extension and fanning out of toes (dorsiflexion)** especially the big toe.
- **Positive Babinski sign in adults is an indication of UMN lesion**, specifically pyramidal.
- When the **corticospinal tracts are nonfunctional**, the influence of **the other descending tracts** on the toes becomes **apparent**, and a kind of **withdrawal reflex** takes place in response to stimulation of the sole, with the **great toe being dorsally flexed and the other toes fanning out.**
- Until the age of 1.5-2 years old, Positive Babinski sign is normal, because at that age **the corticospinal (**the pyramidal pathway) **is still not well developed** (no myelination yet). that's why we said it is an indication in adults.
- After the development of corticospinal (when babies start to walk)→ the response is **planter flexion.**
- After examining the muscle strength, power and spasticity, Babinski's sign should be examined.

Remember that most of time lesions to both pyramidal and extra-pyramidal **co-exist**, but there is an exception to that:
In early stages of a stroke\initial stage (shock stage), the patient may suffer **hypotonia** (pyramidal) for few hours to one day (even if he\she is an adult), and that's because pyramidal tract is affected earlier than extra-pyramidal tract.

Decorticate Vs. Decerebrate posture:



They represent types of rigidity.
It mainly depends on the **location** of the lesion according to the **red nucleus.**

- Above red nucleus: **decorticate**
- Below red nucleus: **decerebrate**

⇨ **Decorticate:**

- extension (rigidity) in lower limbs by extensors
- Flexed upper limbs because rubrospinal is overreactive.
- mainly pontine is the one that is over reacting (it could be the medullary which gives the opposite effect, but usually it has no clinical significance when damaged), it could also be the vestibular tract.
- For better memorization we say that the lesion causes an increased effect of the pontine tract (Remember: pontine tract→ enhancement of axial extensors)
- the name indicates that **it is under the cortex in the internal capsule** → pontine is lost, inhibition is lost →**over reactivity (rubrospinal activity overcomes pontine activity)**.

⇨ **Decerebrate:**

- lesion below the level of red nucleus.
- Decerebrate: extension in both upper and lower limbs
- The Rubrospinal tract affected
- Remember: Rubrospinal → enhancement of distal flexors especially hand and upper limb flexors
- **Rubrospinal lesion** causes extension in upper limb and hand, lower limbs are also extended
- **Rigidity\ spasticity** not flaccid paralysis

Q: which one has better prognosis?

Decorticate lesions has a better prognosis (why?), decerebrate lesions will be below the level of red nucleus, closer to medulla oblongata where the respiratory centers are found, while decorticate lesions are above the level of red nucleus, away from medulla oblongata.

The most fatal injuries are those which happen in brain stem, because it directly effects the vital centers like respiratory centers, unlike cortex injuries which can disable some functions but might not fatal.

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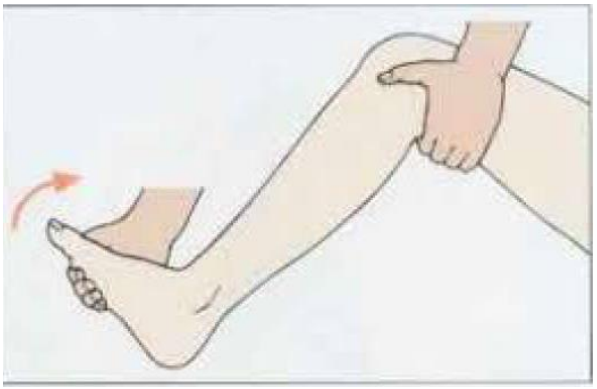
Clonus:



- Clonus is a series of **involuntary, rhythmic, muscular contractions and relaxations.** Seen in **UMN lesions**.
- It is usually initiated by reflex.
- Most important example is **ankle clonus test.**
- Briefly: you support the knee with one hand as seen in picture, and with the other hand you try to do dorsi-flexion of foot.

- In case of ankle clonus there will be a state of **alternating dorsi-flexion and planter flexion caused by exaggerated reflexes.**
- Mainly **extra-pyramidal**

Summary of somatic motor control:

- **Basal ganglia and cerebellum** are very important for **motor activity** and motor system. That will be explained later.
- What happens before movement is the idea of movement (creating the order) which is taken from the cortex (mainly the **prefrontal cortex**).
- **Pre-frontal cortex** is what makes you a human being, everything related to personality, beliefs, thoughts, decisions, social behavior etc. is found there.
  That's why the frontal lobe (motor lobe) is also called **executive lobe** (responsible for actions).

- **Motor association areas**: their role is consultation, includes: **prefrontal, supplementary motor, frontal eye field.**
  Those areas consult basal ganglia (basal nuclei) to make a motor response, because motor ganglia are **previously programmed on habitual motor responses**, so they give instant motor responses without thinking of movement (the first time you walk you create a program of a complex of movements, then you don't have to create another program every time you walk, instead, you use the previous program).

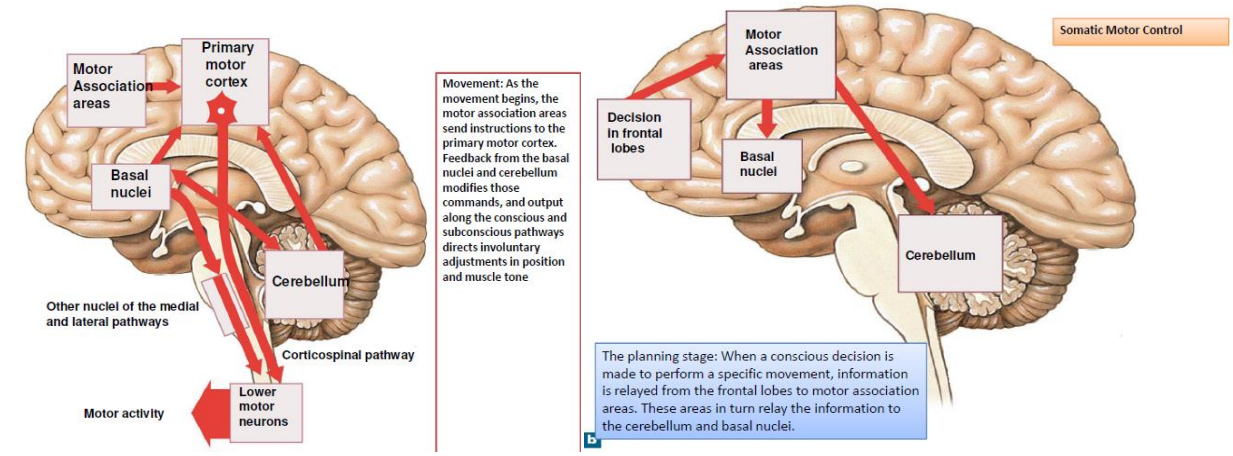
  Same goes for **cerebellum** which is responsible for balance, it has previously stored data related to balance and it has a relation with vestibular nucleus and spinocerebellar tract (remember: spinocerebellar tract→ proprioception).
  So, the motor association areas consult cerebellum to know the current position of the body before movement in order to make a motor plan.

  Then, information (feedback) goes from **the basal nuclei and cerebellum** back to the **cerebral cortex** by motor lobes going through the thalamus (even though it's motor)

Note: Cerebellum and basal ganglia are not connected to spinal cord; instead they deal with cortex directly.

Next it goes down in the descending tract and finally to lower motor neuron (sometimes called the final common path)

All of this will be explained in more details when we talk about cerebellum and basal ganglia.



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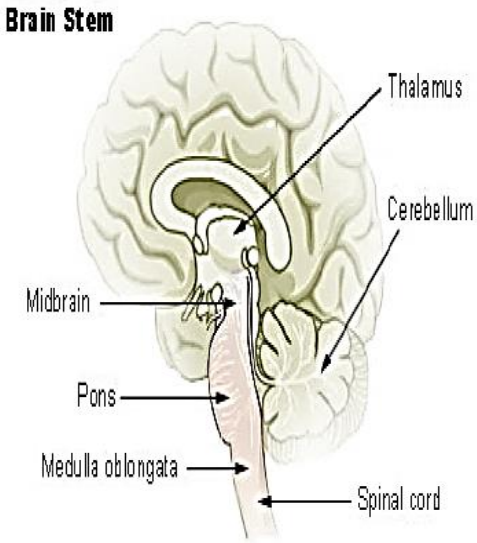
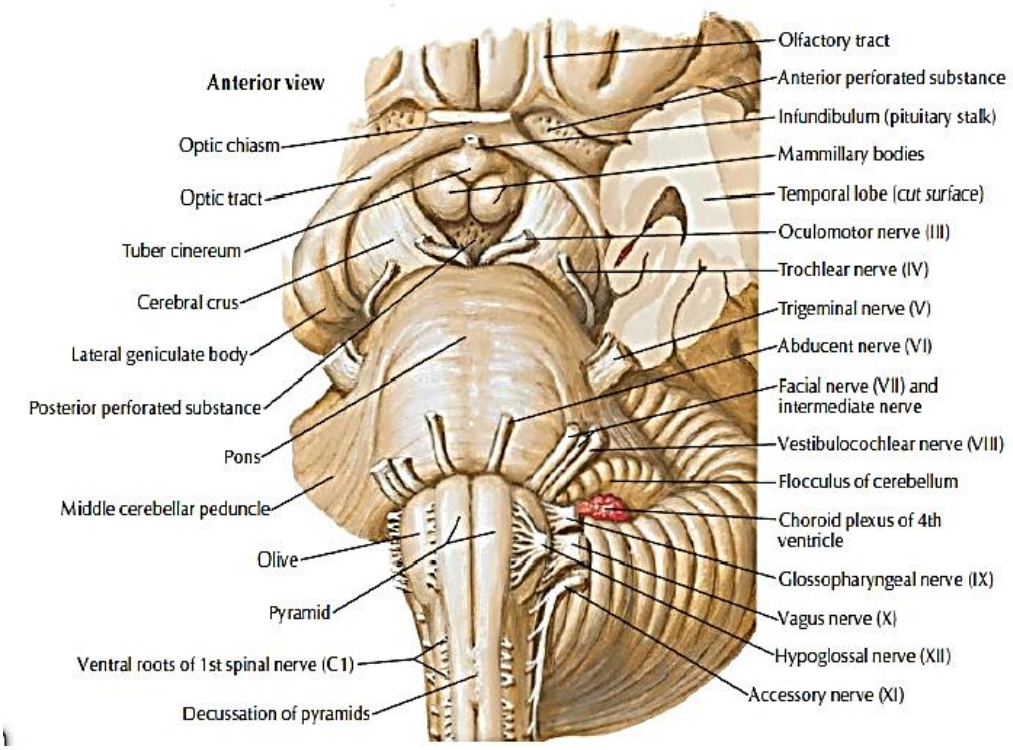
Brain stem:

- **Hindbrain:**
  - Medulla
  - Pons
  - Cerebellum
- **Brain stem:**
  - Medulla
  - Pons
  - Midbrain



- Gross/external features: (explained in lab)



- **The Pyramid two bulges around the midline**, while **the olive away from midline.**
- The picture Shows the order of cranial nerves arising from brain stem.
- The first two cranial nerves**, olfactory (I) and optic (II), are not related to brain stem** they arise from the **forebrain**. Both are sensory.
- Oculomotor (III) arises from the floor of the mid-brain, and it is a motor nerve.
- Trochlear N. (IV) is the only one that arises from posterior\dorsal aspect of brainstem, it is a motor nerve.
- Trigeminal N.(V) largest cranial nerve, arises from mid pontine area, divides into two sensory branches (ophthalmic and maxillary) and one motor branch (Mandibular)
- Abducent (VI), Facial (VII) and Vestibulocochlear (VIII) nerves arise from **pontomedullary junction**
- Glossopharyngeal (IX), Vagus (X), accessory (XI) → arise from the groove between the olive and inferior cerebellar peduncle.

- Hypoglossal (XII) arises from the groove In between the pyramid and olive

Q: what is the cavity found in the hindbrain:

The Fourth ventricle, it is related to both **pons and medulla oblongata**

It has the shape of a tent, with a roof and floor

- the floor of 4th ventricle is formed of **the posterior aspect of pons of medulla oblongata**
- **the roof is toward the cerebellum**
- **the upper part of medulla oblongata is related to the lower part of the 4th ventricle (the cavity)**
- a cross section in the lower part of medulla oblongata will show the central canal

Everything will be explained in more details in the lab.

(32:12)

Internal structure of medulla

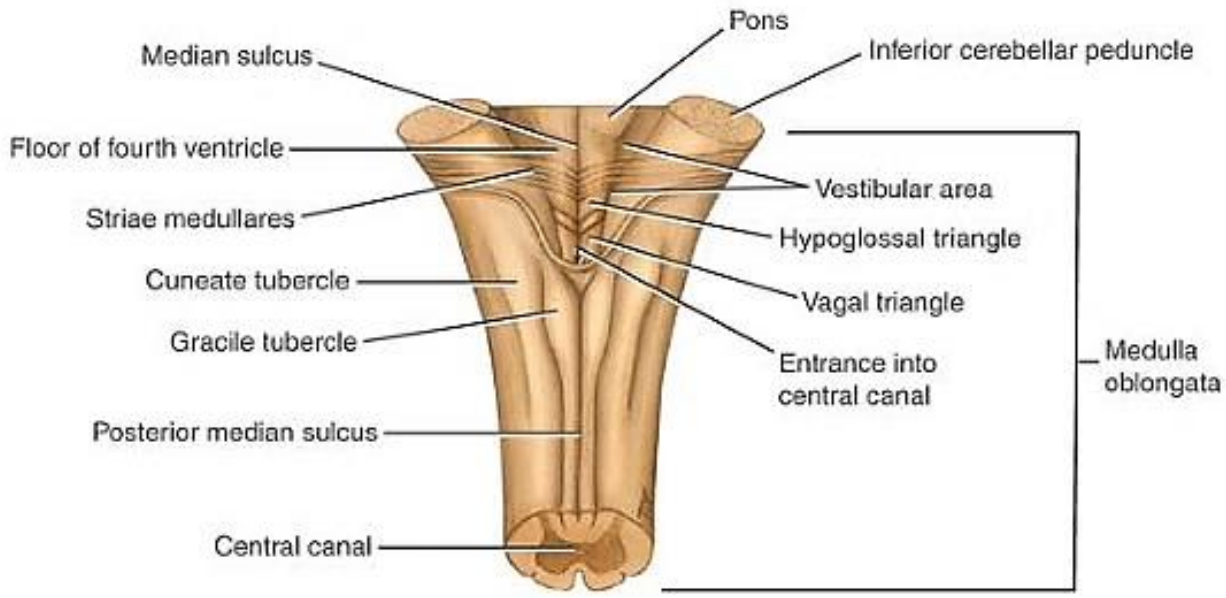
All brainstem will be studied **in 8 sections**, 4 of them from **medulla oblongata**, 2 of these 4 are on **the lower medulla**, where the cavity is the central canal

- if the section has a big cavity → open medulla (2 sections)
- if you see a central canal in the section → close medulla (2 sections)

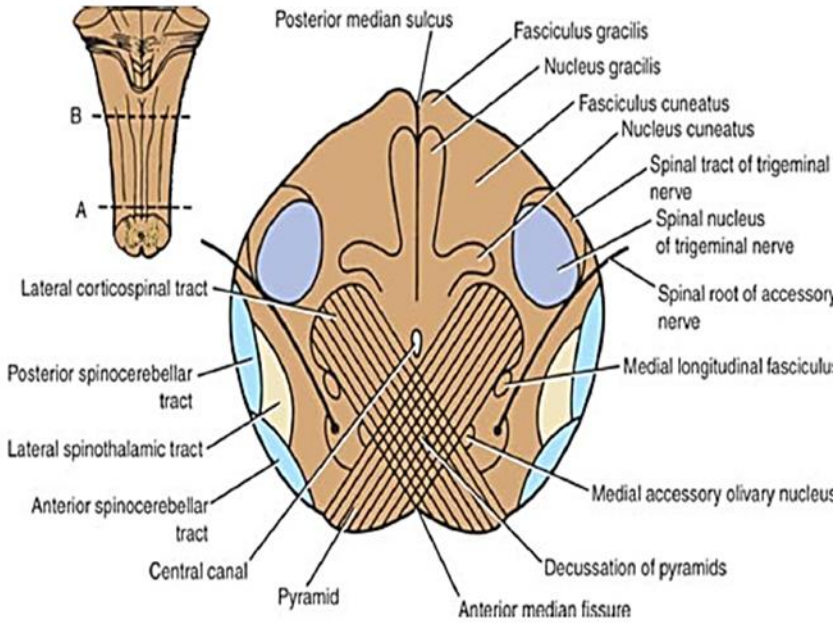
From downward to upward

- level of pyramidal decussation (close medulla / motor)
- level of decussation of lemnisci (close medulla / sensory)
- level of olives (open medulla)
- level just inferior to the pons
- practically they are 3 decussations because 3rd and 4th decussations are too similar

➔ Level of decussation of pyramid:



- This is a **posterior view.**
- You can see the floor of the 4th ventricle which has a rhomboid shape (معين) (two triangles)
- The lower part of it (the lower triangle) is the part that is related to medulla oblongata
- Both sections A & B in the picture below will cut into the closed medulla which mean that they will show the central canal.



- first thing to notice is the **central canal** (make it your reference point)
- **Pyramidal decussation is anterior to central canal.**
- This decussation is composed of corticospinal tract fibers crossing each other; right to left& left to right, **except** for the **anterior corticospinal fibers that go ipsilateral.**
- If you look posterior to central canal you will see:
  - **Gray matter:**
    - Nucleus gracilis: close to midline
    - Nucleus cuneatus: lateral.

    Remember: <u>nucleus</u> indicates gray matter
  - White matter:
    - Fasciculus gracilis
    - Fasciculus cuneatus

Just to remind you when we took the posterior column system:

The fibers that go up are divided to:

- Medially **gracilis**
- Laterally **cuneatus**

  Both are white matter in spinal cord, and they go up to the lower part of medulla and their you'll see

- ✓ The 2 nuclei (gracilis and cuneatus) and their fibers (white matter Fasciculi)

- In brainstem: you'll see the traces of both ascending and descending fibers, all will pass through the brainstem.
- The brainstem consists mainly from the nuclei of the cranial nerves (sensory or motor)

➔ Spinal nucleus of trigeminal nerve:

- **trigeminal Nerve:** originates from **mid pontine area (pons)**
  - **mainly sensory:** head, neck, nasal cavity, oral cavity and face (except for the angle of mandible)
  - **motor:** muscles of **mastication** (tensor tympani, tensor veli palatine, mylohyoid, ant. Belly of digastric)
  - has **four** nuclei: one motor, three sensory
  - sensory nuclei of trigeminal:
    - **I. main sensory (primary sensory):** found in pons
    - **II. mesencephalic:** name indicates it's up in the brain
    - **III. spinal nucleus of trigeminal Nerve:**

- spinal nucleus of trigeminal is named so because it extends **along the brainstem from mid pontine down to spinal cord**, it is an extension of the upper cervical segment substantia gelatinosa (lamina II)
- you'll see the **spinal nucleus in upper and lower sections in brain stem**.
- note it doesn't extend along all the brainstem, it starts from mid pontine and below, while above the mid pontine it is replaced by **main sensory**.

Q: why 3 sensory nuclei for trigeminal N.?

Related to modality:

- **spinal:** pain and temp.
- **mesencephalic:** proprioception
- **main sensory:** crude touch

- Spinal nucleus of trigeminal is surrounded by fibers from trigeminal (white matter) trying to reach nucleus.

- **Important:** the anterolateral system in the spinal cord doesn't change very much when reaching the medulla oblongata, fibers come closer but they still have the same position related to each other, so you should pay attention to the few things:
  1. Nucleus gracilis and nucleus cuneatus (not found in spine)
  2. Fasciculus gracilis and Fasciculus cuneatus
  3. Pyramidal decussation

- **Important**: spinal nucleus of trigeminal is not only trigeminal fibers, the only reason to call it trigeminal is that most of fibers are trigeminal fibers (predominant fibers) but all cranial nerves that have pain and temperature modality (facial, glossopharyngeal, vagus) all relay to spinal nucleus of trigeminal (for example, the glossopharyngeal fibers send input to spinal nucleus of trigeminal).

Medial longitudinal fasciculus:

Vestibular nucleus gives a bundle of white matter that connects the vestibular nucleus with **the motor nucleus of the 3rd, 4th and 6th cranial nerves** (oculomotor III, trochlear IV, abducens VI), to synchronize the movement of eye ball, that bundle of white matter is called **medial longitudinal fasciculus**.

The idea of the link between the three cranial nerves is to coordinate the movement of right and left eye:

When you move your eyes to right or left direction, in one eye it's the medial rectus muscle that contracted but for the other eye it's the lateral rectus that contracted, and for this to happen at the same time we need the connection between the three motor nuclei.

What about the **vestibular nucleus**? The connection between motor nuclei and vestibular nucleus is to coordinate the movement of head with movement of eye (vestibular provides information about gravity) to maintain the visual field while walking and moving your head. لما تثبت عينيك على شيء معين و انت ماشي

we will see the Medial longitudinal fasciculus more clearly from **upper sections

Remember:

Nerves that control the movement of the eyeball:

1- **Oculomotor (III):** all external muscles except superior oblique and lateral rectus.
2- **Trochlear (IV):** supplies superior oblique.
3- **Abducent (VI):** supplies lateral rectus.

Thank you