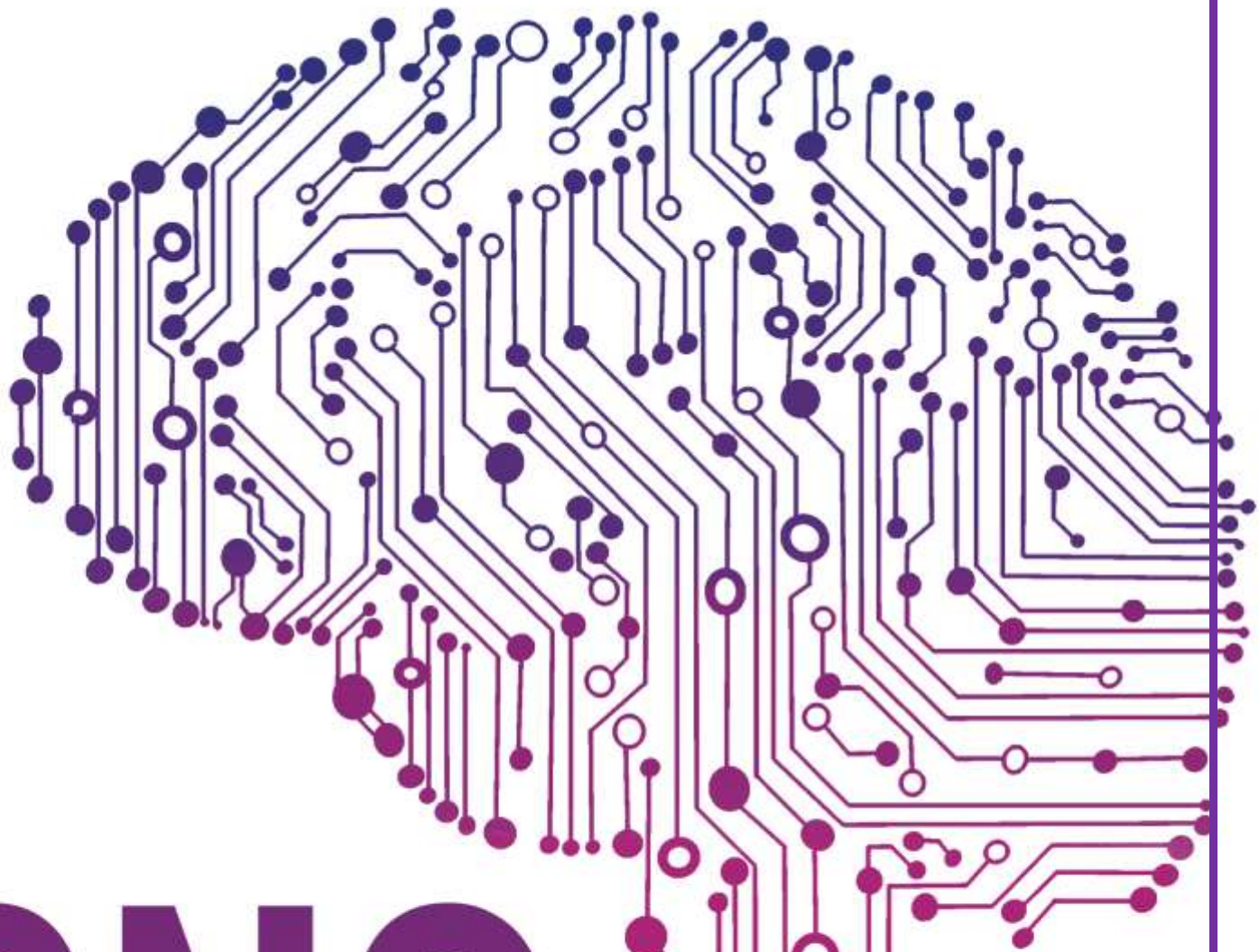

CNS

ANATOMY

6

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Hello everyone, in the last lecture we discussed the descending tracts (motor tracts) that were divided into **Pyramidal** and **Extrapyramidal** tracts, in this lecture we will discuss the differences between (Upper motor neuron lesions and Lower motor neuron lesions).

Comparison between upper motor neuron (UMN) and lower motor neuron (LMN):

Features	Upper motor neuron lesions (UMN)	Lower motor neuron lesions (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

Notes about this table:

-Damage or **a cut to the upper motor neuron** will result in **exaggerated reflexes** (Hyperreflexia) because usually, the effect of the cortex in general on the reflexes is inhibitory.

- Related to hyperreflexia is another phenomenon called **hypertonia** (the muscle tone is increased). **The muscle tone** is a partial state of contraction in the muscle and it is **important in maintaining posture**.

- Hypertonia and Hyperreflexia is the result of an **increase in gamma motor neurons activity**.

- The opposite of these effects occurs in **damage to lower motor neurons because you cut all innervation to the muscle** so **hypotonia (muscle is flaccid) and hyporeflexia** occur and eventually atrophy of the muscle occurs (no atrophy in cut to UMN).

- **Fasciculation** (alternating contracting and relaxation in the same muscle as the twitching of the eyelid) is **present in the case of a LMN lesion and absent in the UMN lesion**.

- **Paralysis occurs in both UMN and LMN lesions**, however **the type is different** if you cut LMN the muscle becomes hypotonic and flaccid (the muscle is relaxed), in UMN lesion the muscle becomes hypertonic and has exaggerated reflexes so there is spastic paralysis, the muscle is rigid (clasp knife).

The skeletal muscle is composed of:

1- Extrafusal fibers (99%): It is the functioning and contractile unit of the muscle, innervated by alpha motor neurons.

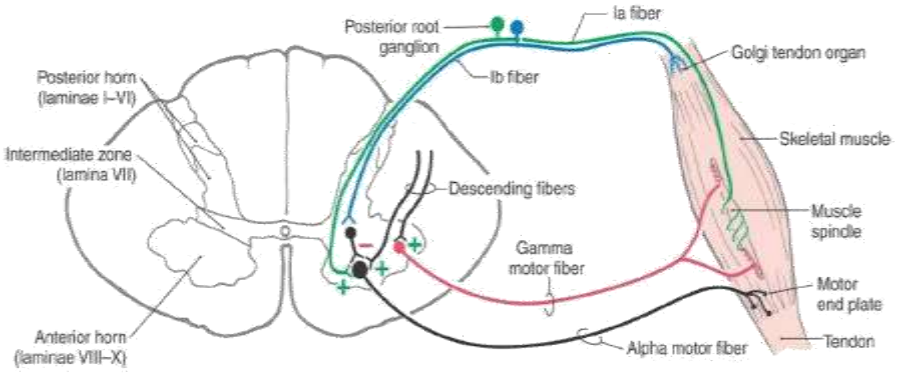
2- Intrafusal fibers (1%): located inside a capsule and contain stretch receptors, innervated by gamma motor neurons.

Muscle spindles (stretch receptors + intrafusal fibers + the capsule) are sensory receptors within the belly of a muscle that primarily detect changes in the length of this muscle.

Each muscle spindle consists of an encapsulated cluster of small striated muscle fibers ("intrafusal muscle fibers") with somewhat unusual structure (e.g., nuclei may be concentrated in a cluster near the middle of the fiber's length).

Now we know that the lower motor neurons emerge from the ventral (anterior) horn of the spinal cord through ventral root to reach the skeletal muscle, It can be either:

- ✓ **Alpha motor neuron.**
- ✓ **Gamma motor neuron.**



Stretch: when the muscle is relaxed or when there is a contraction in the antagonist muscle, the muscle might become overstretched (increase in length).

Example: when there is contraction in the triceps, the biceps becomes stretched and the receptor in the muscle spindle will sense this stretch, and will generate a receptor potential (firing), which will move through the sensory fiber through dorsal root and enter the spinal cord to activate alpha motor neuron that goes to the muscle to contract as reflex.

Activation of alpha motor neurons:

- ✓ **Directly**: from supraspinal centers, through the descending motor pathways (UMN).
- ✓ **Indirectly**: through activated muscle spindles, How ?
  1) Stretch: **Muscle spindle is sensitive to stretch** which means that when the length of the muscle increases it gets activated then it will synapse directly with the lower motor neuron that goes to the same muscle then the muscle will contract. Why we

have such reflex? To preserve muscle tone. Muscle tone indicates that the muscle is always in partial state of contraction because all muscles are shorter than the distance between origin and insertion of it. Muscle tone mainly preserves posture, for example; when you stand up, the partial state of contraction of antigravity muscles like extensors of lower limbs preserves your posture.

2) Gamma loop: Descending tracts activate alpha motor neuron and gamma motor neuron which supply muscle spindle at the same time. Why? First, we have to take a closer look at the muscle spindle.

There are 2 types of intrafusal fibers:

A) Nuclear bag: the nuclei converge in the center like a bag, supplied by dynamic gamma fibers.

B) Nuclear chain: the nuclei converge in the center like a chain, supplied by static gamma fibers.

In both of them, the sarcomeres are located in the periphery while the central area is free of sarcomeres. When they get activated through gamma fibers, the tips will contract while the central area (which has sensory fibers) will stretch → activation of muscle spindle → activation of alpha motor neuron → contraction of extrafusal fibers.
This happens in case of **sustained** contraction.

> ***Extra explanation:*** *when the muscle contracts in response to stretch the muscle spindle will shorten and will no longer be able to sense the stretch on the muscle, but when gamma fibers stimulate the spindle to contract, it increases its sensitivity to stretch and allows it to maintain a steady firing rate for the same stretch, thus the muscle will be able sustain its contraction against that stretch.*

- Gamma fibers activate the muscle fibers indirectly, while alpha fibers do it directly.

- **Alpha fibers give faster but short contraction**

- **Gamma fibers give slow but long contraction**

- For fast contraction: stimulate alpha, while for muscle tone: stimulate gamma.

- For continuous contraction and a certain movement: stimulate both.

❖ Both nuclear bag and chain (where nuclei are clustered) don't contain sarcomeres.

When we look at the muscle spindle, we will find **two types of afferent fibers:**

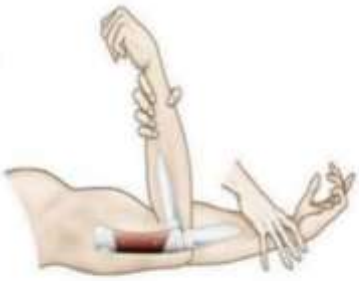
❖ **Primary afferent fibers:** take sensation from both nuclear bag and chain, type 1a fibers according to the old classification, Aα according to the newest one. They have large diameter and high velocity (rapidly adapting) and is responsible for dynamic stretch reflex which

happens in jerks. When you hit a tendon with hammer, the primary afferent will get activated then the reflex will result. Hint: type 1b is found in Golgi tendon organ.

❖ **Secondary afferent fibers:** take sensation from nuclear chain only, type 2 fibers (Aβ). They have smaller diameter and lower velocity (slowly adapting) and is responsible for static stretch reflex which is important in muscle tone. You want the tone to be sustained, so whenever you have a signal, you will have a response. In this way we preserve the tone.

- Clasp knife reaction:

when a clasp knife is opened "fully extended" and you try to close or open it, initially there is resistance but after reaching a specific angle or point it closes suddenly. And the same concept applies to the rigidity that happens in the patient (UMN lesion) where the patient would have a flexed muscle and when the doctor tries to extend the arm of the patient initially there will be resistance but if he persists and applies enough force there will be "sudden release" and the arm will extend.

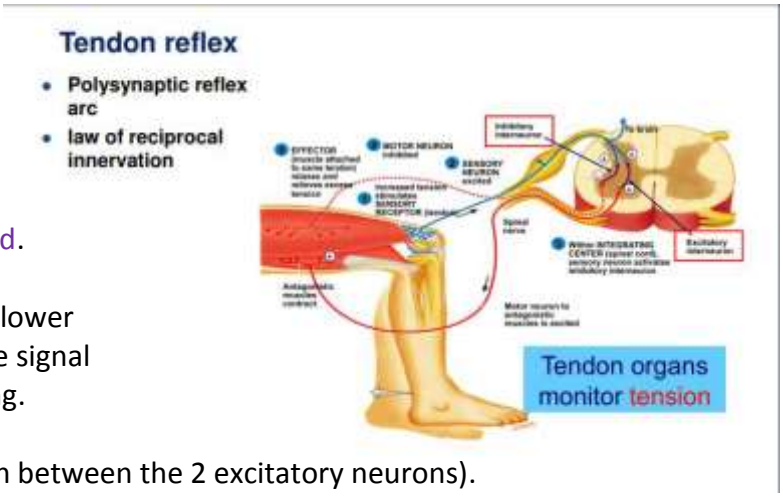


Explanation of why this happens is related to the 2 phenomena:

1) Initial resistance: Exaggerated stretch reflex
The muscle resists stretching, when you stretch a muscle, it responds by contracting and because UMN lesions cause exaggerated reflexes the effect is bigger.

2) Sudden release: Caused by activation of Golgi tendon reflex also called anti-stretch reflex, which resists excessive contraction in the muscle.
(After applying pressure, the tension in the muscle will increase and will be enough to *activate the **Golgi tendon organs*** which will cause the relaxation).

Ex: (pay attention to the figure)
There is a contraction in the quadriceps and this creates tension in its tendon and causes firing of the Golgi tendon organs which are sensory receptor organs, one of the reflexes that happen activates 2 interneurons in the spinal cord.



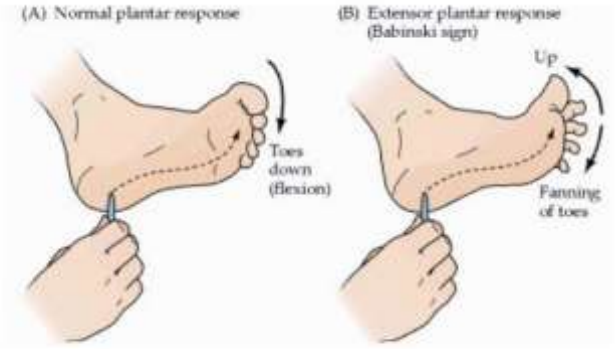
The first interneuron is inhibitory, it inhibits the lower motor neuron that is going to the muscle that the signal originated from to stop it from further contracting.

This reflex is polysynaptic (inhibitory interneuron between the 2 excitatory neurons).

At the same time there is activation of the **second interneuron which is excitatory** and activates the lower motor neuron that is going to the antagonist muscle (the muscle which works in the opposite direction to the original muscle) in this example it is the hamstring, this is what's called the **law of reciprocal innervation**, so that the contraction of a muscle or set of muscles (as of a joint) is accompanied by the simultaneous inhibition of an antagonistic muscle or set of muscles.
It is impossible to have a full contraction of the biceps and triceps at the same time because when there is a contraction in the biceps there is relaxation in the triceps and vice versa (reciprocal innervation).

- **Babinski sign** (present in UMN lesion and not in LMN):

To understand it let's explain the normal response first, When a doctor stimulates the sole of the foot (specifically the lateral aspect) with a blunt object, the normal response is flexion of the toes.
But in cases of UMN lesions what occurs is the opposite (called Babinski sign), when stimulating the sole of the foot what occurs is fanning of the toes, and the big toe is dorsiflexed rather than being flexed and this is suggestive of UMN lesion.



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.

Note: Upper motor neuron lesions most of the time affect both pyramidal and extrapyramidal tracts, it is rare that only one of them is affected, however, the explanation to most of the phenomenon that happens (clasp knife, hypertonia…etc.) is related to the extrapyramidal tract **except Babinski sign which is explained by pyramidal tract**.

Note: in children below the age of 1-1.5 years, positive Babinski sign is normal because full development and myelination of the pyramidal tracts happen after 1-1.5 years of age (when the child stops crawling on 4 limbs and starts standing and moving on 2).

- **Clonus** (another symptom of UMN lesion, not LMN):

In testing for clonus the doctor would attempt to dorsiflex the foot and would face resistance (remember what we said above) and when he applies enough force clonus happens which is **rhythmic contractions and relaxation of muscles when they are subjected to sudden sustained stretch caused by exaggerated reflexes.**

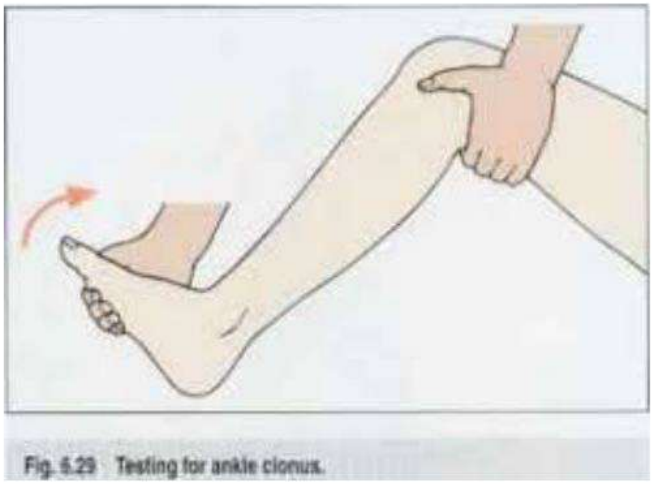


Fig. 6.29 Testing for ankle clonus.

Rhythmic contractions and relaxation of muscles when they are subjected to sudden sustained stretch

Decerebrate and Decorticate rigidity:

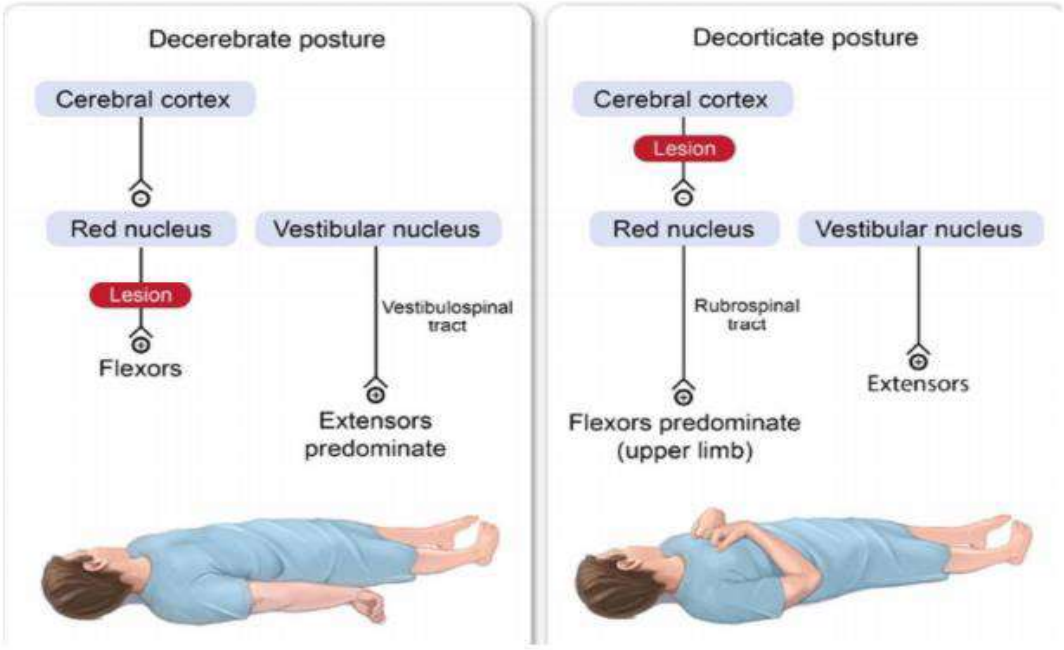
- Both Decerebrate and Decorticate rigidity are related to UMN lesions, the difference between them is the level of the lesion, if the lesion is higher than the red nucleus it's decorticate, and if the lesion was lower it's decerebrate.

- In decorticate there is rigidity in the entire body and the lower limbs are extended while upper limbs are flexed and rigid, in decerebrate, there is also complete rigidity and both the lower limbs and the upper limbs are extended.

- The midbrain (will be discussed next week) is divided into two levels, level of superior colliculus and level of the inferior colliculus, at the superior level there is a structure called the red nucleus (from which the rubrospinal tract descends) a lesion above this nucleus is called decorticate and below it is decerebrate.

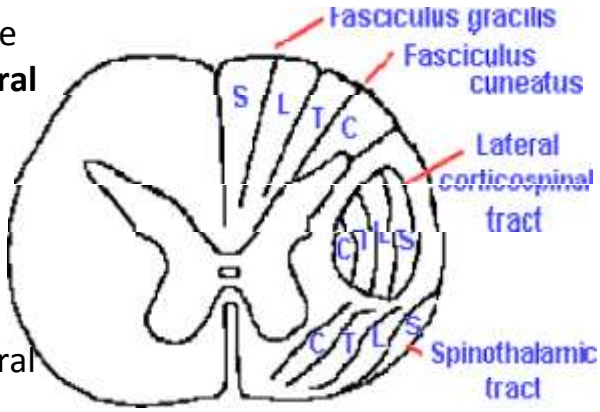
- ❖ **Decorticate posture** (lesion above red nucleus so you affected\removed the cortex, from the name), remember what we said above about the pontine reticulospinal tract and that it is **tonically active** and removing the cortex causes more activation so its effect is more prominent and it causes activation of extensors in the leg and flexors in the arm (antigravity muscles).
- ❖ **Decerebrate posture** (lesion below the red nucleus) the rubrospinal tract is part of the lateral motor system and is responsible for the flexion of muscles in upper limbs so if it is lost, there will be an extension of the upper and lower limbs.

The vital centers (related to CVS and RS) are present in the medulla oblongata and pons (lower part of the brain stem) so a brain stem injury is fatal, if you lose part of the cortex, you lose some functions but brain stem injury is fatal, that is why **decerebrate is worse than decorticate because the lesion is closer to the vital centers so prognosis is worse**



Clinical significance of lamination of the ascending tracts:

- Any **external pressure** exerted on the spinal cord in the region of the **spinothalamic tracts** will first experience **a loss of pain and temperature sensations in the sacral dermatome of the body**



- If pressure increases the other higher segmental dermatomes will be affected

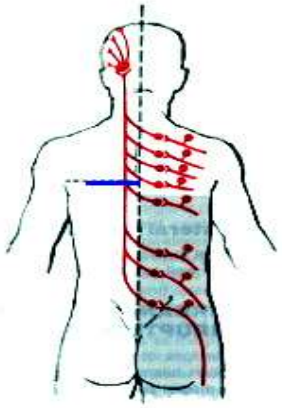
- Remember that in the spinothalamic tracts the cervical to sacral segments are located medial to lateral

- Intramedullary tumor: affect the cervical fibers (Medial)
- Extramedullary tumor would affect lower limb fibers (lateral)
- Sacral sparing: Occur at intramedullary tumor

Clinical application:

1) Destruction of the LSTT (lateral spinothalamic tract):

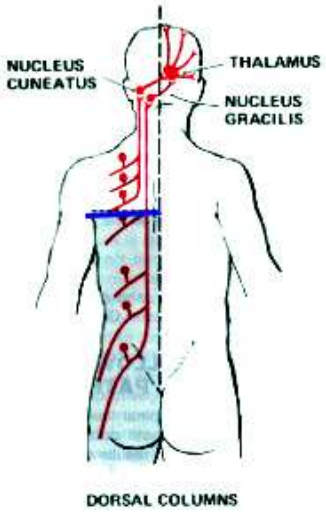
Loss of pain and temperature sensation on the **contralateral side** (due to decussation which happens at the level of the spinal cord) below the level of the lesion.



2) Destruction of the posterior column (fasciculus gracilis and cuneatus):

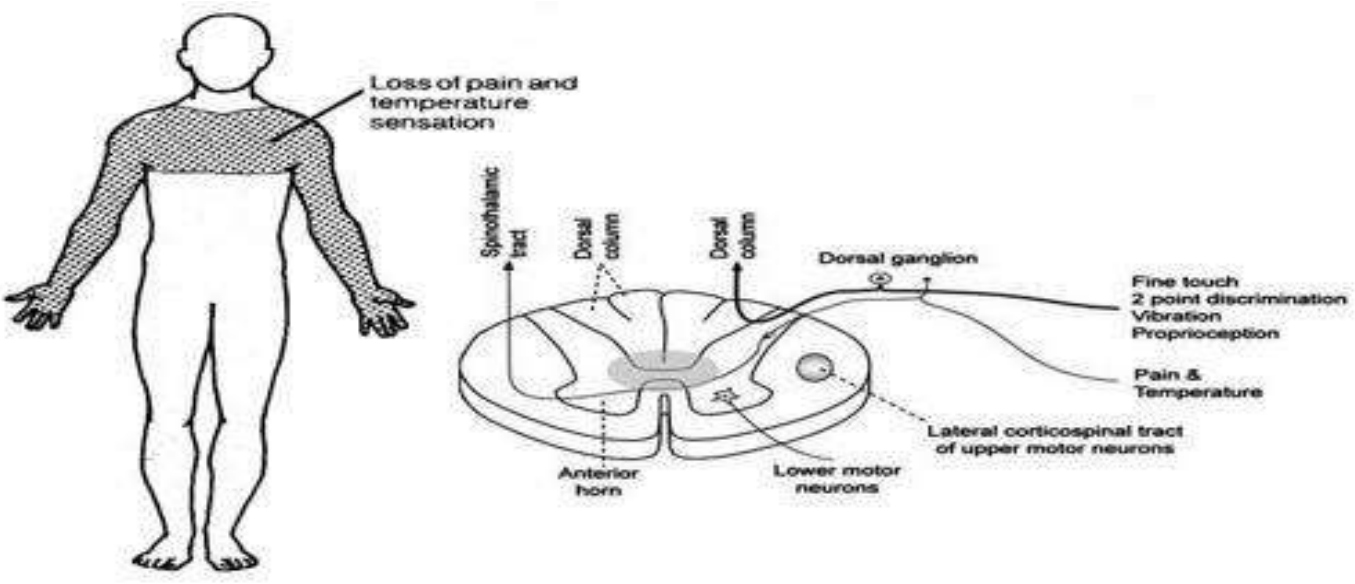
Loss of muscle-joint sense, position sense, vibration sense, and tactile discrimination **ipsilaterally** (because the decussation happens above at the level of the medulla oblongata, so the damage happened before the crossing over) below the level of the lesion.

Note: it is extremely rare to have a lesion of the spinal cord to be localized as to affect one sensory tract only.

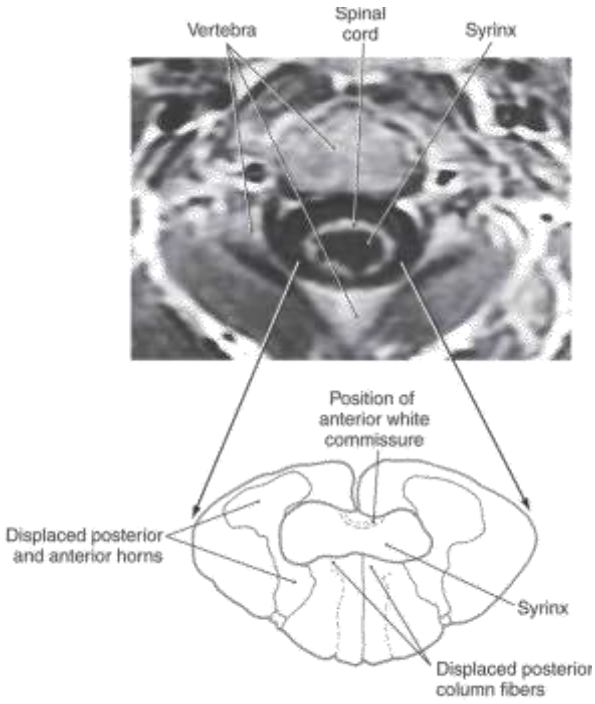


3) Syringomyelia: Cavitation of the central canal in the spinal cord (increase in size of the canal) could be due to any reason, this cavitation will damage the fibers crossing in the anterior white commissure in both directions, this will cause **bilateral loss of pain and thermal sensations.**

example: if the syringomyelia is located at the C4-C5 levels of the spinal cord, sensations are lost in the configuration of a cape draped over the shoulders and extending down to the level of the nipple.



Note: fibers don't cross over in a horizontal manner, they are a bit tilted, for example let's say the first order neuron entered at the level of C3, it will synapse with the second order neuron at the level of C3 and as its crossing in the anterior white commissure (anterior to the central canal) it will become at the level of C4 and by the time it finishes crossing over it will have reached C5, so the process of crossing over doesn't happen horizontally.



In some cases this cavitation extends to the anterior horns, causing **muscle weakness and even paralysis sometimes**, if the syrinx (cavity) extends to one anterior horn, this will cause an ipsilateral weakness if both anterior horns are involved, the weakness will be bilateral.

4) Brown-Séquard Syndrome:

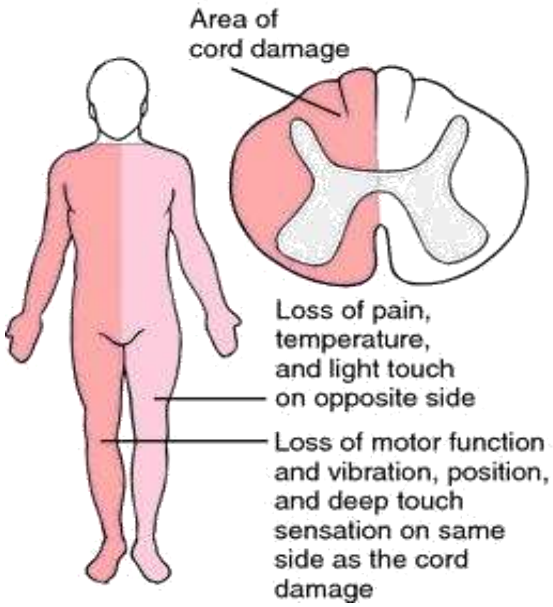
Functional hemisection of the spinal cord (damage that involved half the spinal cord), this will cause damage to the corticospinal tract, ALS, posterior columns.

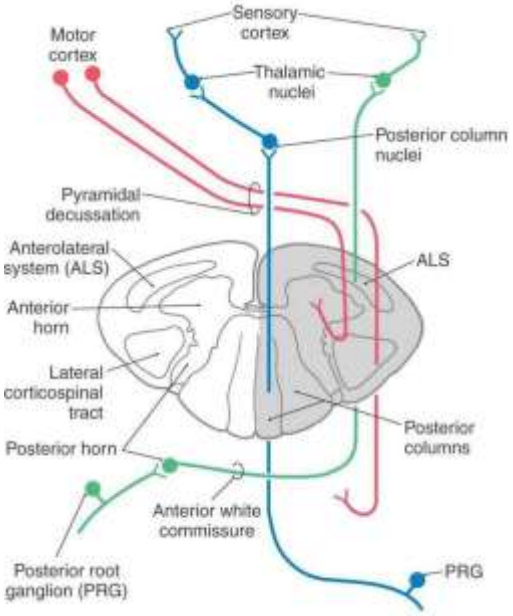
What will happen?

1) **Contralateral** loss of nociceptive and thermal sensations over the body below the level of the lesion.

2) **Ipsilateral** loss of discriminative tactile, vibratory, and position sense over the body below the level of the lesion.

3) **Ipsilateral** paralysis or weakness (hemiparesis, hemiplegia).





Arterial blood supply of the brain:

- Brain is supplied by **pairs of internal carotid artery and vertebral artery.**

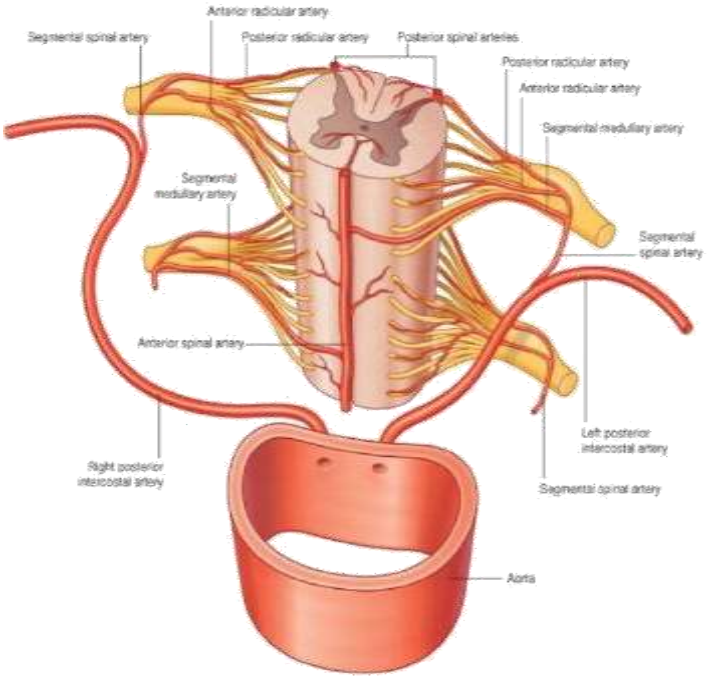
- Internal carotid artery enters the skull through carotid canal "in the petrous part of temporal bone"

- Vertebral artery enters the skull through foramen magnum "in the occipital bone"

❖ Their branches anastomose on the inferior surface of the brain to form **Circle of Willis.**

- The four arteries lie within the subarachnoid space. The two vertebral arteries will unite forming **basilar artery** that **gives posterior cerebral artery** and **receive posterior communicating artery**.

Arterial blood supply of the spinal cord:



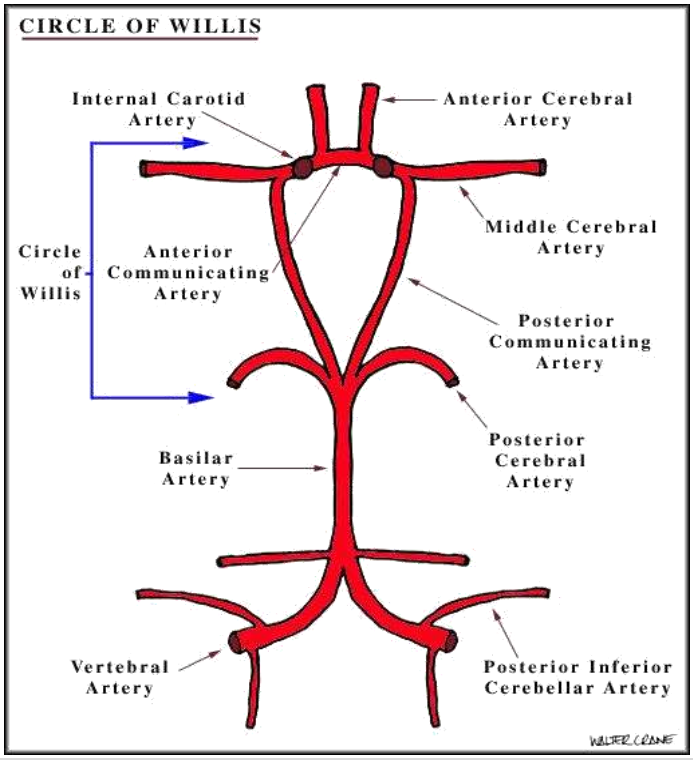
1) Longitudinal arteries:

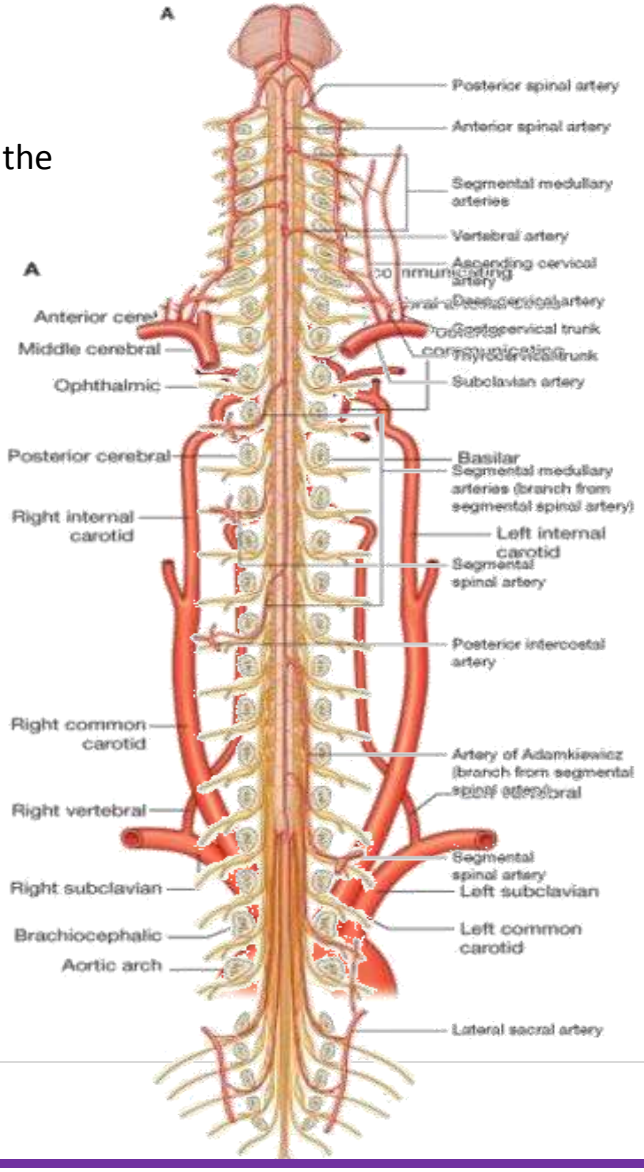
A) <u>One</u> anterior spinal artery: arise from the vertebral arteries (runs in the anterior median fissure).

B) <u>Two</u> posterior spinal arteries: arise from the posterior inferior cerebellar artery branch from the vertebral artery (in the posterolateral sulcus).

2) Segmental (horizontal) spinal arteries enter intervertebral foramen, arise from:

- Vertebral arteries and deep cervical arteries (in the neck).

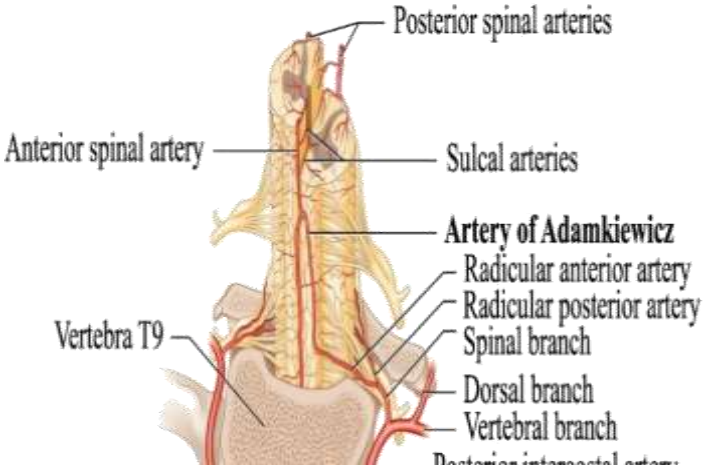




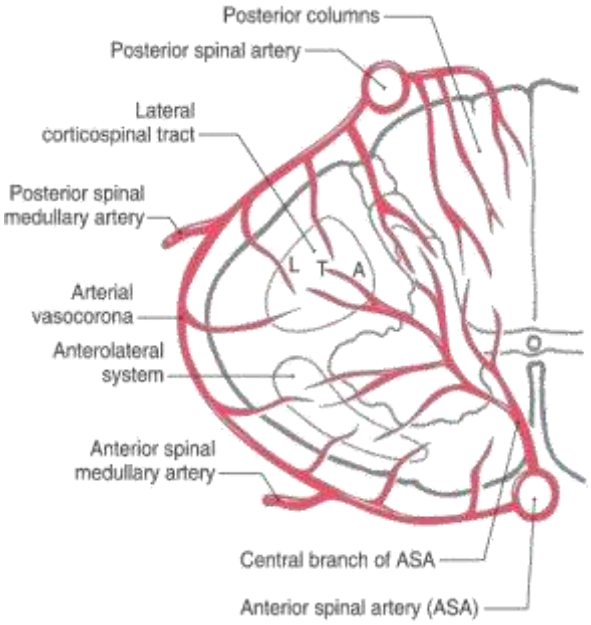
- Posterior intercostal arteries (in the thorax).

- lumbar arteries (in the abdomen).

Now every segmental artery gives three branches:



- Posterior radicular artery (runs with posterior "dorsal" root to reach spinal cord).
- Anterior radicular artery (runs with anterior "ventral" root to reach spinal cord).
- Segmental medullary artery (anastomose with anterior spinal artery).



3) Artery of Adamkiewicz:

- usually <u>on the left side</u>, from the left posterior intercostal artery at the level of the 9th to 12th intercostal artery, which braches from the aorta and supplies the lower
two third of the spinal cord.

- This will <u>reinforce the arterial supply to the lower portion of the spinal cord</u> (far from circle of Willis)

- Anastomose with anterior spinal artery.

Blood supply of the spinal cord (at the level of the segments):

❖ Terminal branches of the spinal medullary arteries join to form **Arterial vasocorona**.

1) Posterior spinal arteries and arterial vasocorona: the posterior columns and peripheral parts of the lateral and anterior funiculi.

2) Anterior spinal artery: Most of the gray matter and the adjacent parts of the white matter.

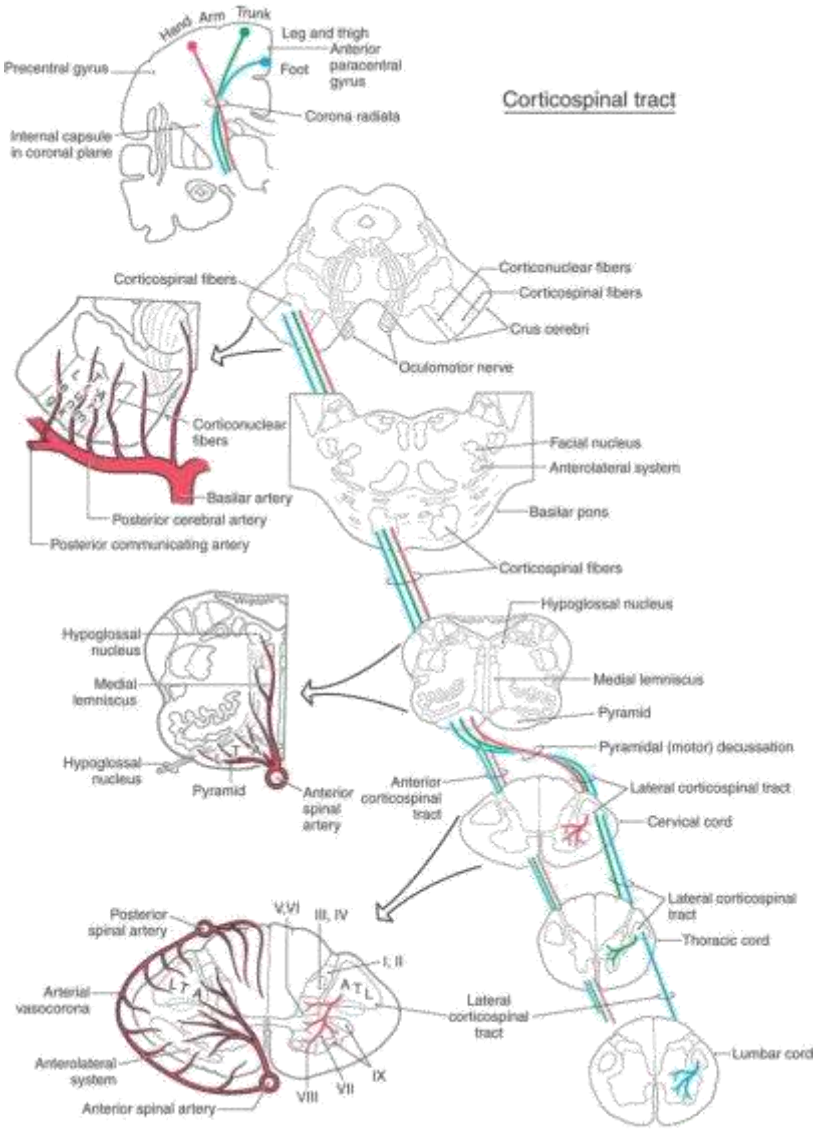
Note: It is very important to memorize the distribution of these arteries and the regions they supply, for the lecture after the next one when we talk about lesions.

Venous drainage of spinal cord:

1) Two pairs of pairs on each side.

2) One midline channel parallels the anterior median fissure **(Anterior spinal vein).**

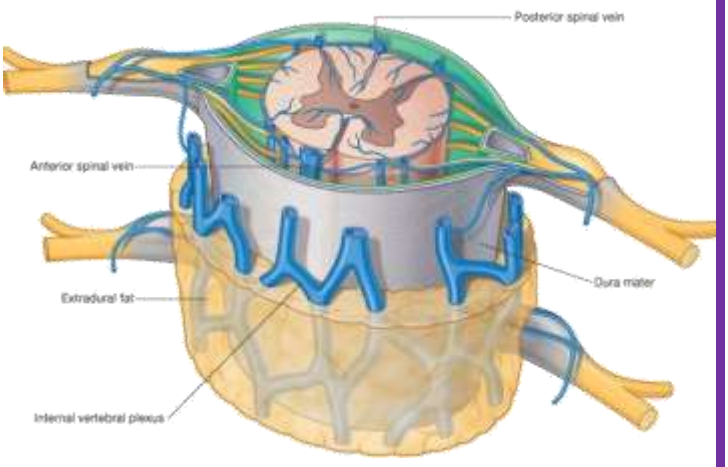
3) One midline channel passes along the posterior median sulcus **(Posterior spinal vein).**



Those veins will drain into an **extensive internal vertebral plexus in the extradural (epidural) space of the vertebral canal**, then drains into **segmentally arranged vessels** that connect with major systemic veins like <u>azygos system in the thorax or intracranial veins.</u>

Central Cord Syndrome:

- Occurs in case of **occlusion in the blood supply of the anterior spinal artery**, which often occur in the **case of neck hyperextension.**
- This results in **bilateral weakness in extremities, more in upper than lower extremities.**
- Why Bilateral weakness? Because remember that we have <u>one anterior spinal artery that supply both right and left side</u>.
- Why upper extremities are affected more than lower? <u>Because the origin of the anterior spinal artery is from the vertebral</u>



artery, so its blood supply is coming from above so its affected more, furthermore the lower extremities receive blood supply from other sources (like Artery of Adamkiewicz)

- Also, its characterized by **bilateral pain and thermal sensation loss, bladder dysfunction.**
- Compromise of blood flow in the **posterior spinal artery** results in:
  Ipsilateral reduction or loss of discriminative, positional, and vibratory tactile sensations at and below the segmental level of the injury.

Good luck