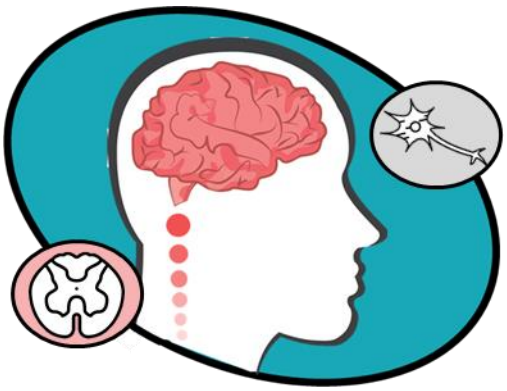

CNS

Anatomy

Sheet Slide

Number

5

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We started comparing upper motor neuron lesion with lower motor neuron lesion

Remember that Upper motor neuron(UMN) extends from the cortex down till anterior horn cells, while the lower motor neuron (LMN)starts from anterior horn cells in spinal cord to end in skeletal muscles.

Now is there a difference between UMN lesions and LMN lesions ?

For a student who didn't know the difference between UMN and LMN his answer will be no ,he will thought that the two types of lesion will lead to paralysis ,but this is not the case , why ?

Due to the facts of presence of reflexes as stretch reflex, so we some differences appear as opposing symptoms between upper and lower motor system .

- Upper motor neurons include pyramidal and extrapyramidal (descending tracts), both of them get affected with the lesion (it is very rare for a lesion to affect one of them and spare the other)

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

In this schedule, the upper motor neuron lesion consequences are due to extrapyramidal tract except for bibinski sign, which is related to pyramidal tract.

- We will start with the reflexes:

In upper motor neuron lesions , **hyperreflexia** occurs , it is because of muscle stretch.

Do you remember stretch reflex ?

We know that in lower motor neurons, alpha motor neuron innervate extrafusal muscle fibers and Gama motor neurons innervate intrafusal muscle fibers (which are composed minority of fibers(1%)) , and we said that the stretch reflex is responsible for muscle tone. Intrafusal fibers contain sensory fibers called muscle spindle . briefly, if a muscle stretched , firing occur in these sensory fibers , they get activated and pass through dorsal root then they activate alpha motor neuron directly without interneuron (monosynaptic),hence muscle contract , it appear as a mechanism to resist stretch in the muscle .now lets use this stretch reflex to explain symptoms of UMN lesion.

If an upper motor neuron lesion takes place, hyperreflexia is the symptom,why?

the upper motor neuron effect on Gama motor neuron is inhibitory, so if the inhibition is released due to the lesion, and the activity for Gama motor neuron increases, and that leads to hyperreflexia (exaggerated reflexes).

- Note: If the pyramidal is affected, hypotonia is the consequence. Extrapyramidal is hypertonia. As we said earlier, that the lesion affects both not one, but the stronger effect is the extrapyramidal tract . So, this results in hyperreflexia followed by hypertonia. This is what called **spastic (rigid) paralysis.**

- Now in lower motor neuron lesions, **hypotonia** occurs. This is because alpha and gama neurons are damaged, so the muscle has no innervation ,thus decrease tone (hypertonia), in this case the muscle is extremely relaxed which mean it is paralyzed **(flaccid paralysis**). As a result, wasting of the affected muscle is the consequence

Note: wasting of muscle mean atrophy or decrease their size. The size of the muscle is determined by its action and nutrition. So injury in the nerve of the muscle will cause atrophy .**however UMN lesions don't cause wasting of affected muscles.**

- Now we move on to rigidity, specifically, clasp knife rigidity.

In upper motor neuron lesion, (as strokes affect the internal capsule which is passage of UMN), clasp knife rigidity occur , it is related to extrapyramidal tract, especially pontine extrapyramidal tract, reticulospinal, and vestibulospinal. Normally, they work as extensors of the lower limb.

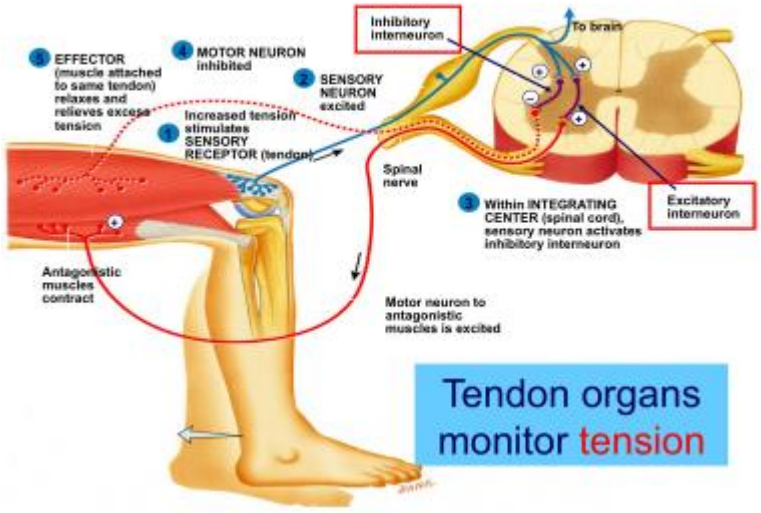
Now, What is the relation between clasp knife and the this type of rigidity ?

If you use clasp knife ,you will force initial resistance ,then sudden release happen .this is similar to rigidity occur due UMN lesions as the rigidity occurs in 2 stages: initial resistance and sudden release.

Initial resistance: because of **overacting pontine reticulospinal** (exaggerated stretch reflex). In this stage, the muscle resists any type of elongation, because of hyperreflexia.

Sudden release: it is thought that it occurs because of activation of golgi tendon. in order to understand it, we must explain Golgi tendon.

Golgi tendon:



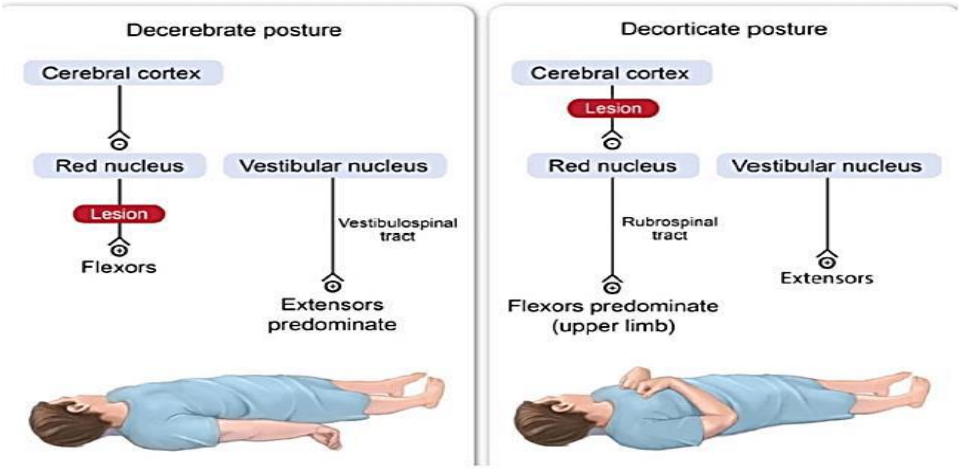
In this example, the 2 involved muscles are quadriceps and its antagonist the hamstring muscles.

If we apply concept of Stretch reflex here, what happen when hamstring muscle is contracted (antagonist muscle) at the same time quadriceps muscle is stretched ,so firing in muscle spindle send their sensory neuron to activate lower motor neuron cause contraction in quadriceps . In the other hand, **inverse stretch reflex (Golgi tendon reflex**)happen , If a contraction occurs in the quadriceps, the receptors which are located in the tendon (Golgi tendon receptor) fire sensory signals to

reach the dorsal root. This will activate two types of interneurons: e**xcitatory,** that sends order to the hamstring muscle antagonist muscle to contract, The other type of interneuron is **inhibitory**, which sends r**elaxation** order to the quadriceps(**same** muscle that contracted at first) telling it that it contracted too much and it is time to relax.

Note: stretch reflex which resist extreme elongation (stretch) in muscle is the **opposite** of Golgi tendon reflex which is resist extreme contraction(tension) in muscle .

There are other types of rigidity: decorticate and decerebrate



both of them are related to **upper motor neuron**. They differ in the level of the lesion accordind to red nucleus in mid brain , at the level of superior colliculus.

- If the lesion is above the red nucleus it is called decorticate.
- If it is lower than red nuclus level, it is called decerebrate.

Decortecate

Recall that the extrapyramidal tract pontino reticulospinal and vestibulospinal are tracts that maintain posture by facilate extension of lower limb and flextion of upper limb. while medullary spinal and rubrospinal oppose them in activity .

Normally, the pontinespinal tract is tonically active, and its under inhibition from cortex . So, in decorticate (as the cortex is inhibited), no inhibition will happen to the pontinespinal tract , the effect will be more activation of this tract , and that will affect the posture. The lower limb is

rigid and fully extended, and the flexors of the upper limb is flexed.this type of rigidty called decorticate .(right picture)

Q: why medullarysoinal tract doesn't cancel effect of pontinespinal tract as they have two opposing effects ?

Because although medullryspinal tract works as an antagonist for the pontinespinal tract ,however the pontinespinal tract is tonically active (its tend to be hyperactive).So, if the cortex is inhibited, the overriding force will be to the pontinespinal tract .

decerebrate:

the lesion is below the level of red nucleus as midcollicular incisions between superior colliculs and inferior colliculs that used in animal research , so the damage will occur to the **rubrospinal.** The rubrospinal tract works as flexor for the upper limb as this tract facilitate flexors movement and inhibit extensors movement of the distal flexor muscles precisely, and have a little effect on the proximal muscles. So, if the flexor is inhibited, this will result in extensors overriding. So, the upper limb is extended and rigid.

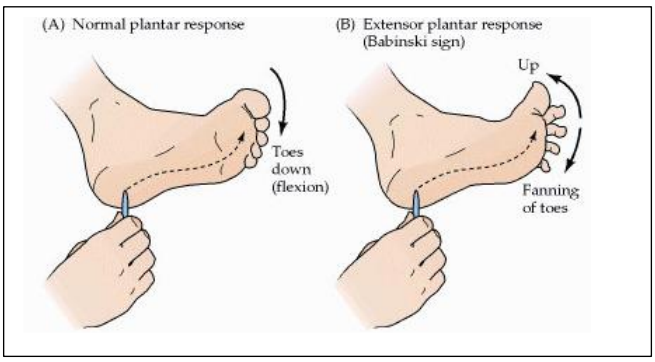
Note: the rubrospinal tract is the only one of extrapyramidal tracts that from lateral motor system with the lateral corticospinal tract

Q: which one has better prognosis?

Decorticate lesions has a better prognosis ,why?

As the **vital centers** are in the lower part of brain stem in the pons and medulla, so if the lesion is below the red nucleus (decerebrate), this is **closer to the vital** centers, so it is more dangerous.

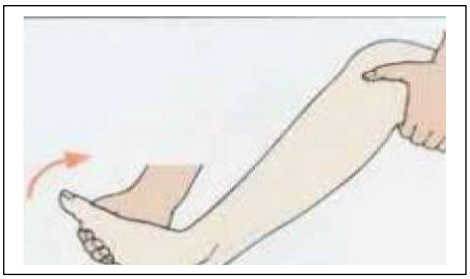
- Babinski sign:



As we said earlier , the upper motor neuron lesion consequences are due to extrapyramidal tract except for bibinski sign, which is related to pyramidal tract.

Normally, in a human being *above the age of one*, if you move a blunt object on the sole of the foot laterally (from the heel to the toes), normal planter reflex happens (toes are planter flexed) .In the case of **Babinski (inversed plantar response),** when you make the same test, the reflex will be **fanning of the toes** and the **big toe will be dorsally flexed**.

- ✓ Positive Babinski sign in adults is an indication of UMN lesion, specifically pyramidal. However, in children below the age of one, the Babinski sign is normal, because the pyramidal track is still not fully myelinated. Full myelination completes when the baby starts to walk
- ✓ Remember that most of time lesions to both pyramidal and extra-pyramidal co-exist, but there is an exception to that:
  Sometimes in the shock stage (after stroke or infarction), hypotonia takes place in the initial first hours followed by hypertonia, although it still the most case suffer firstly from hypertonia .This is due to the fact that the pyramidal tract is affected faster than the extrapyramidal.



- Clonus:

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

Most important example is ankle clonus test.

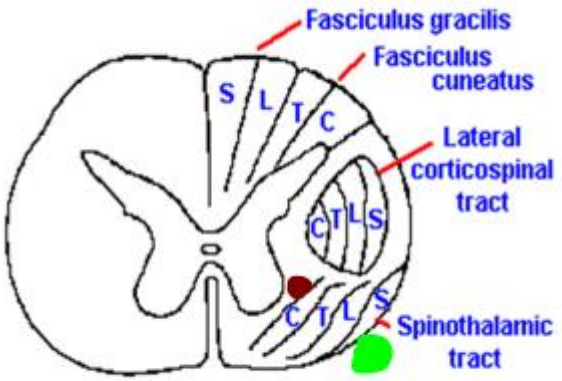
Briefly , the doctor support the knee with one hand as seen in picture, and with the other hand he try to do Doris-flexion of foot. after releasing the force, ankle will get dorsiflexed and plantar flexed in alternating fashion rapidly. This is related to upper motor neuron lesion. it can explained by exaggerated reflex.

- Fasciculation: rapid contraction and relaxation of the same muscle .related ***to lower motor neuron lesion***. The explanation is not required for the exam.

Spinal cord lesions:

Note you need to study the first lab lecture to understand lesions .

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



Clinical importance

- Intramedullary tumor(brown): affect the cervical fibers (Medial) , usually it less possible to affect sacral ,and this what its called sacral sparing .
- Extramedullary tumor (green): would affect firstly sacral fibers (lateral) as the tumor will expand it will affect at end cervical fibers .
- Sacral sparing: Occur at intramedullary tumor (because the sacral fibers are far away from the lesion)

It is very rare that one tract only is affected, but we will discuss these imaginary situations.

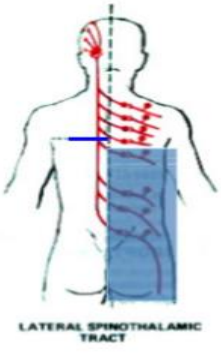
Extra note :Spinal cord injuries: whatever is partially or completely damaged, the effect appears below theinjury level, neither the sensory goes up, nor the motor goes down.

At the level of injury we have two motor pathways (pyramidal & extra-) and two sensory pathways(spinothalamic & dorsal column).

<u>If the lateral spinothalamic tract is affected at the blue line (second order neuron), the symptoms are:</u>

Clinical application
destruction of LSTT
- loss of
  - pain and thermal sensation
  - <u>on the contralateral side</u>
  - below the level of the lesion

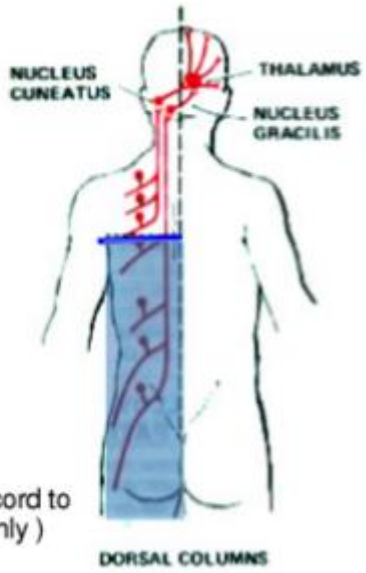
patient will not recognize hot and cold



If the posterior column system is damaged, the lesion will lead to:

Clinical application
destruction of
fasciculus gracilia and cuneatus

- loss of muscle joint sense, position sense, vibration sense and tactile discrimination
- on the same side
- below the level of the lesion



(extremely rare to have a lesion of the spinal cord to be localized as to affect one sensory tract only)

Syringomyelia:

It is a Cavitation of the central regions of the spinal cord (may occur due to meningitis and other causes).

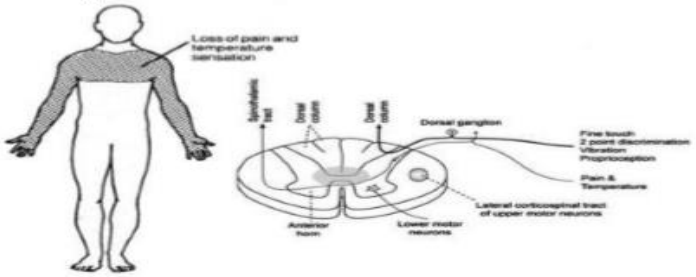
This will cause damage fibers of spinothalamic tracts that crossing in the anterior white commissure in both directions (from left to right and vice versa), which will cause Bilateral loss of pain and thermal sensations

Note: Syringomyelia won't affect the posterior column system because it does not cross over the midline.

☒ Important note: fibers that cross the midline don't cross horizontally. The highest dermatome affected indicates the lowermost cord segment occupied by the lesion because the fibers cross over two or three segment. Let's assume that fibers enter at C6 , they ascend to discussit anterior white commissure at C5 level fibers .then reach the other side at level of C4 .

Another important example:

- When it is located at the C4 to C5 levels of the spinal cord sensory losses in the configuration of a cape draped over the shoulders and extending down to nipple level



If syringomyelia extends **into one anterior** horn results it will cause **ipsilateral motor deficit**; if it reaches the right side, it will cause weakness of the muscles of the right side. if **both anterior horns are involved, the weakness is bilateral.**

Brown-Séquard Syndrome

Functional hemisection of the spinal cord (half of the spinal cord is cut at one side), it's very rare, this results in damage to the lateral corticospinal tract , ALS, posterior columns.so the symptom is related to each tract as

- ✓ Contralateral loss of nociceptive and thermal sensations over the body below the level of the lesion

• Ipsilateral loss of discriminative tactile, vibratory, and position sense over the body below the level of the lesion

• Ipsilateral paralysis of the leg or leg and arm, depending on the level of the hemisection