The doctor continued talking about upper motor neuron lesions and mentioned 2 conditions :-

The difference between them is the location of the cut, which will lead to different manifestations.

Please refer to slide 43 in the pdf file or page 30 in the printed handout

FIRST: Patient with "**Decerbrate Rigidity** " : increase tone in the extensors of the upper and lower limb

The damage here in the middle of the midbrain in other words it separated brain stem from cortex, and as we know that Pontine Reticulospinal tract present in the pons, and it's very active, and when it works it cause increase in the tone of extensors and flexors, but mostly on the extensors, and also we know that its inhibition comes from area 6, so if we cut this pathway at any point above pons (in other word we remove the inhibition) it will cause release from inhibition, and will activate alpha and gamma excessively which will cause the stretch reflex to work excessively, and eventually hypertonia and more rigidity and spacticity will occur.

SECOND: Patient with **Decorticate Rigidity**: increase tone in the extensors of the lower limb, and flexors of the upper limb.

The damage here above the mid brain so the red nucleus is intact not damaged so the Rubrospinal tract which originates from **Red nucleus** still works, so it will cause flexion of the upper limb.

Which one of these patients have better prognosis (less dangerous condition)?!

Decorticate patient because the level of cut is above the midbrain which is further from the pons and medulla, while decerebrate patient the level of cut in the midbrain so the cut can affect the vital centers in the medulla because it's closer to it. The further you get from the medulla, the better prognosis you have,.

P.S Rigidity mean: increase in tone.

Now we'll talk about another reflex :-

Please refer to slide 45 in the pdf file or page 32 in the printed handout

****Flexin Reflex (withdrawal Reflex)**

Simply means that when a person steps on a sharp object, the injured foot is withdrawn (removed from danger), we call this flexion reflex because we flex the knee, this reflex is protective reflex but at the same time it's part of walking mechanism (walking mechanism: you rise one leg and the other is supporting you.

Afferent of this type (type 2, 3,4) of reflexion maybe present in the skin or muscle or joint, and considered rapidly conducting, and we call them **Flexor Reflex Afferent** (**FRA**).

This type of synapse also use interneuron and don't directly synapse on alpha and gamma, so this reflex is polysynaptic, remember stretch reflex was monosynaptic. Now those afferent will enter and synapse on inhibitory interneurons, and these inhibitory interneurons will inhibit the alpha which supply the extensor muscle and through excitatory neurons we will activate the Agonist muscle (flexor here), so in this reflex we flex muscle but this flexion must be proceeded by inhibition of the antagonist muscle (extensor muscle), ex: inhibition of triceps must precede stimulation of biceps. Returning to the flexion reflex, the second leg will be extended to us balanced while the first leg is flexed and raised, we call this reflex: flexion crossed extension **(flexion on one side and extension on the other)**.

How this is done on the other side ??

Some of the afferent fibers go to the other side and also use interneurons but to do the opposite and extends the knee joint.

Also if the stimulus is very strong with great intensity (like when a nail enters your foot and goes deep) you may raise your arms we call this phenomenon Irradiation. :- the stimulus is very strong and will be carried by the proprio-spinal tract 》》 which will affect the muscles of the cerviacl region and will make you raise your hands.

Now These inter-neurons are activated and facilitated by pyramidal tract, if the pyramidal tract is damaged, the extensor will be activated and flexor will inhibited (opposite to normal function) or make the effect of both flexor and extensor equal.

****Knee Flexion Reflex:**

Please refer to slide 46 in the pdf file or page 33 in the printed hand out

When I do knee flexion, hamstring muscle will flex the knee, and quadriceps will extend it, (do not forget that before you stimulate the agonist you must inhibit the antagonist)

1. Now higher centers (corticospinal pyramidal tracts) send signals to inhibit antagonist, before stimulation the agonist, through an interneuron we call it Ia inhibitory inter neuron, it will inhibit the alpha of the antagonist muscle (Quadriceps muscle).

2. Higher centers also send other signals to activate the alpha and gamma of the agonist (Hamstring muscle)

3. Contraction of extrafusal by alpha and intrafusal by gamma will occur

(remember from the previous lectures we activate alpha and gamma to initiate a rapid contraction and then maintaining that contraction).

4. now the spindle stimulated, rapidly it send signal through afferent fibers Ia and increase alpha activation(increase contraction) and increase inhibition of interneurons of antagonist muscle (decrease contraction)

5. once the contraction occur, the knee is flexed, so the result of this flexion is the antagonist muscle will be stretched passively, so the muscle spindle will be stimulated, rapidly send signals through 1a to alpha, alpha here in refractory period (in refractory period the cell do not respond to any stimulus) so the alpha will not respond.

Upper Motor Neuron Lesions (UMNLs):

Upper motor neurons are those which present in cerebral cortex area 4, 6 , the lesion may present in the neuron in cortex, or in the descending fibers (axons)anywhere, where anywhere?

First level: after cortex: internal capsule

Second level: midbrain, also pons, medulla

Third level: these axons will continue to spinal cord (before reaching alpha and gamma), all these levels considered UMNLs.

Note: lesion to alpha and gamma or their axons (of the spinal nerve) are considered lower motor neuron lesion.

Note: most of the UMNLs are vascular lesions.

Now in the internal capsule the pyramidal fibers run very close to the extra-pyramidal, if you induce a lesion in the pyramidal tract you cannot avoid extra-pyramidal tract, the symptoms and signs reflect how much the pyramidal and extra pyramidal are affected, but remember most of the signs reflect absence of the extra-pyramidal (we should remember when we say extra-pyramidal just one pathway which is the pontine reticulospinal tract because it's very active, and must receive inhibition to restrict it's activity, so when we induce a lesion to the internal capsule, we loses this inhibition 》》 excessive activation of alpha and gamma will occure leading to excessive work of stretch reflex, which mean more muscle tone and spasticity (rigidity).
Now there are places when we induce a lesion to them, the affected pathway is just pyramidal not both pyramidal and extra-pyramidal (they're both rare to happen):
-First place: pyramid in medulla (we have inside it just the pyramidal tract)
-Second place: area 4 (pyramidal tract originate mainly from area 4)
Now if you induce lesion to the pyramidal tract in pyramid region the effect will be paralysis or paresis on the contralateral side because this lesion above the decussation, and hypotonia (flaccidity) will occur, the reason of

hypotonia is the inhibition of the excitatory effect of pyramidal tract and absence of the stretch reflex.
Note: Also this occurs when we induce lesion to area 4.

Now when we induce lesion in area 6 (remember pontine reticulospinal tract originates from extrapyramidal tract which is controlled by area 6), for this reason Reticulospinal tract is always inhibited by area 6, and when we induce lesion to area 6, we cut this inhibition, so alpha and gamma will be activated >> excessive stretch reflex >> more and more tone, excessively eventually spasticity and rigidity will occur.
So spasticity occurs due to extra-pyramidal involvement.

Now if we induce lesion to internal capsule (both tract affected) like in cases of stroke, what will happen?!
The signs of extra-pyramidal will over shadow the pyramidal how?!
In first few hours flaccid or hypotonia will occur (as a result of absence of pyramidal tract)
After one to two days spasticity or hypertonia will occur (as a result of absence of extra pyramidal tract), so here we can see the absence of inhibition on the brain stem is more important than the absence of excitation.

Now let's come to signs of UMNLs suppose the lesion in the internal capsule as an example, it doesn't matter as long as its before reaching the lower):-
1. Contralateral paralysis or paresis (weakness)

Note: paralysis or paresis depend on severity of the lesion, paresis is when the injury is mild while if it was a severe injury that leads to damage to the internal capsule then paralysis occurs.

Note: contralateral because the lesion above the decussation.

In the first few hours flaccid paralysis and hypotonia will occur on the affected area, after that increased muscle tone or spasticity will occur

especially in certain muscles (antigravity muscles: flexors of the upper limp and extensors of the lower limb and mainly the Extensors of the lower limb, the patient can't open his arm and he can't bend his knee joint, he will drag it across the ground. The increase in tone happens as a result of absence of the extra pyramidal tract, which inhibits the pontine reticulospinal tract.

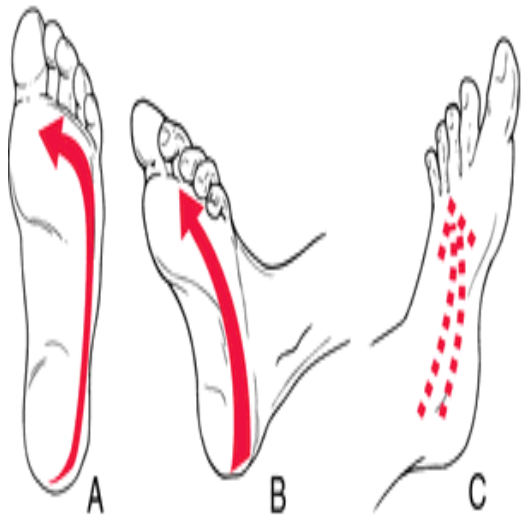
2. Hyperreflexia: when we do knee jerk or biceps jerk to the affected side we expect to have hyperreflexia (the reason here also the absence of the extra-pyramidal tract and very active alpha neurons which result in powerful contraction).
3. clonus :- please refer to slide 57 in the pdf file or page 43 in the printed handout

Clonus is a form of movement marked by contractions and relaxations of a muscle, occurring in rapid succession.
When we suddenly put our hand beneath the foot and suddenly push the foot upwards (Dorsiflexsion), you'll have a series of platar flexion(the foot will move downwards) and dorsiflexion, we call this clonus.
Explanation of this :-
Simply when you hold the foot from the sole and do dorsiflexion, the muscles of the back of the leg (gastrocnemius and soleus) will be little stretched, their muscle spindle will be activated 》》 contraction of these muscles leads to activation of stretch reflex and plantar flexion (the foot moving downward), but you are still holding the foot and you are resisting that movement 》》 so the muscles Anteriorly will be little stretched 》》 their muscle spindle will be activated 》》 leading to Dorsiflexion
And then again and again, this occur in upper motor neuron lesion.
Why doesn't clonus occur in a healthy individual?
because in UMNL patients, their muscle spindle and gamma neurons are overactive and the smallest strech on the spindle activated the stretch reflex activates alpha neurons that cause contraction. So the liberation of the pontine tract caused the #2 + 3# symptoms .

But in, normal people, the pontine reticulospinal tract is not free like in these patients.

4. Babinski sign :-
When you scratch the bottom of the foot from the heel to the fingers
Healthy response :- Plantar flexion of the toes
UMNL :- Dorsiflexion of the big toe and fanning of the other toes

A is line of stimulation or the the line where you scratch
B plantar flexion (normal reaction)
C positive babinski sign (Big toe bends upward, smaller toes fan outward



What causes the babinski sign ??
The absence of the Pyramidal tract,evidence for that ??

If you do this reflex in a newly born -up to six months -it will give positive babinski sign (physiological), why ??

The Pyramidal tract is not yet myelinated so they're not functioning, keep in mind a paitient under anesthesia or in a comma will be also positive as well as a person under stress and tension.

Now do you remember what we said lastly in the" flexion reflex" before we start in the "knee flexion " -page 2- :-

Now These interneurons are activated and facilitated by pyramidal tract, if the pyramidal tract is damaged, the extensor will be activated and flexor will inhibited (opposite to normal function) or make the effect of both flexor and extensor equal.

The same will happen here

Normally :- dorsiflexion

Without pyramidal tract :- extension of the big toe instead of flexion

What about the other toes ??

Here Flexion = Extension 》》 Abduction of the fingers, its midway between flextion and extension.

In babinski sign thes same rules applied here, so this is considered a flexor reflex.

5. -Continuation of the symptoms - absence of abdominal and cremasteric reflex :- with your finger or nail, move it on the skin of the abdomenon on the side that lost the innervation

Normally you'll get a twitch (this is also flexion relfex), in upper lesion you'll get nothing, in obese individual you'll also get nothing because the muscles are already over stretched.

Cremasteric reflex :- in a a male, when you scratch the upper skin of the thigh

The corresponding testis will be pulled , How ??

The touching of the skin 》》 sends impulses 》》 alerted a muscle named Cremasteric muscle found in the spermatic cord, in patients with upper lesion 》》 No response.

Note :- Hyperreflexia without babinski sigh 》》 insignificant

Babinski sign will appear before the symptoms that are related to reflexes appear

So if a a patient was brought to you with stroke, you don't start with reflexes because they appear after a day or two, Babinski appears in the earliest stages. If it's a stroke, you try to know the cause of it, embolic, thrombotic or hemorrhagic. If it was caused by an embolus, we do ECG because it can originate from a mural thrombus the heart, we can also check the internal carotid as a thrombus can originate from there. Further embolism in these cases can be prevented by giving anticoagulant, while if you give anticoagulant to a patient who have hemorrhagic stroke, it can lead to death.

Now let's continue:-

Please refer to slide 56 and page 42 ...

The doctor describes the patient in the picture

This patient has upper lesion in the left and the damage is in the right

His right hand is partially flexed and the tone is increased in the flexors in the upper limb and Extensors in the lower limb - this patient will walk using a crutch and he will drag his right leg as a one piece.

When we try to open his arm or bend his knee joint, "Clasp-knife rigidity " will occur

Remember from previous lectures initial resistance then it gives away. In the early resistance, the stretch reflex is activated since we've pulling the muscle spindle, leading to more contraction and resistance which activates the golgi tendon organ leading to relaxation and it gives away.

Internal capsule contains descending motor and ascending sensory fibers, so if the capsule is cut, in the first hours we will lose sensory too Contraleterally, we call it **Contraleterally hemianesthesia temporarily** (complete loss of pain, touch or temp) but it's temporarily, why ??

Because the thalamus helps in analyzing sensory information, crudely not carefully and expertly as the cortex.

So in internal capsule cut we'll have Hemiplegia, Temporarily hemianesthesia and hemiface (lower part is affected); muscles of the lower face get innervated by one corticobulbar tract on each side.

Muscles of the lower face pulls the angle of the mouth towards it, an UMNL to the right side will cause weakness of lower left face, arm and leg and the mouth gets pulled to the right by healthy muscles.

Area 4 and 6 in the cortex are wide so it's hard from a single vascular injury to take it down once, so if we have a vascular injury there we'll have limited paralysis, monolegia (a hand or leg or a lower lart of the face) 《 《 one of them will be damaged, in contrast to internal capsule damage, where all of them is damaged.

Area 4 and 6 are in the frontal lobe and are close to motor speech area, so :-

If you saw a patient with monoplegia, in upper limb specially, and if the injury is on the left side of the cortex and the right arm is damaged the more probably motor speech area is damaged too.

If the injury is in the brain stem :-

Let's say in the medulla

The manifestation of the Pyramidal and Extrapyramidal when they are damaged while descending here are Contraleterally, while the manifestations of the damage in the cranial nerves are Ipsilaterally (mid brain contains neurons of CN III AND IV)

Examples

So you'll see a patient :-

-Have left Hemiplegia and his right eye, the pupil is shifted outward

So he has an injury on the right at the level of midbrain (where the 3rd cranial nerve is)

》》 before decussation 》》 left H-H》》 the CN damage is ipsilateral

So the right eye is affected.

-have left H-H and paralyzed tongue 》》 Hypoglossal is down

the Hypoglossal is in the medulla, so the level of injury is in the medulla.

** This is called Alternating Hemiplegia :- H-H on one side and nerve damage on the other side.

Cranial nerve damage is considered lower motor neuron lesion; at the level of Alpha and gamma neurons, where as the Pyramidal and Extrapyramidal are considered upper, even though they are both being cut at the same level.

Written by :-

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FUAD ZAYED

Dedicated to you all, especially to

قاسم شجراوري

مهند المومني، ليث الحواجرة، أحمد خان

سلام نصيرات، محمود سعيد، عمر حكيمو

والشيخ مينا بطارسة