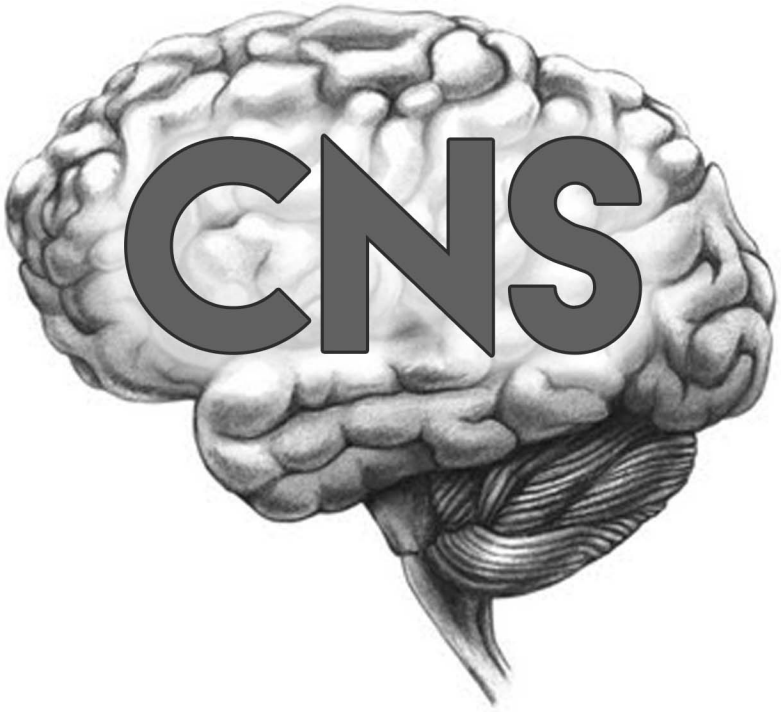


ANATOMY

☑ Sheet ○ Slide ○ Handout

Number

7

Subject

Upper & lower motor neurons' lesions.

Done By

Lina Mansour.

Corrected by

Sara Zayadneh.

Doctor

Faraj Al-bustami

Date: **00/00/2016**

Price:

(sec2 record)

In this lecture we will apply our previous knowledge about the pyramidal and extrapyramidal motor tracts ,their functions & the reflexes associated with motor tracts , by knowing the effect of their lesions.

We are concerned with:

- Upper neurons lesions
- Lower neurons lesions

First, let us put boundaries between what is considered as upper & lower motor neurons lesions.

Look at figure1.

- The **upper** motor neuron lesion might be caused by a stroke any lesion in these areas will create an upper motor neuron lesion:

✓ At the motor cortex (area 4 or 6).
✓ At the internal capsule.
✓ At the mid brain , pons , medulla.
✓ At the level of spinal cord BEFORE the cortical projecting fibers synapse with alpha/ gamma neurons of the spinal cord.

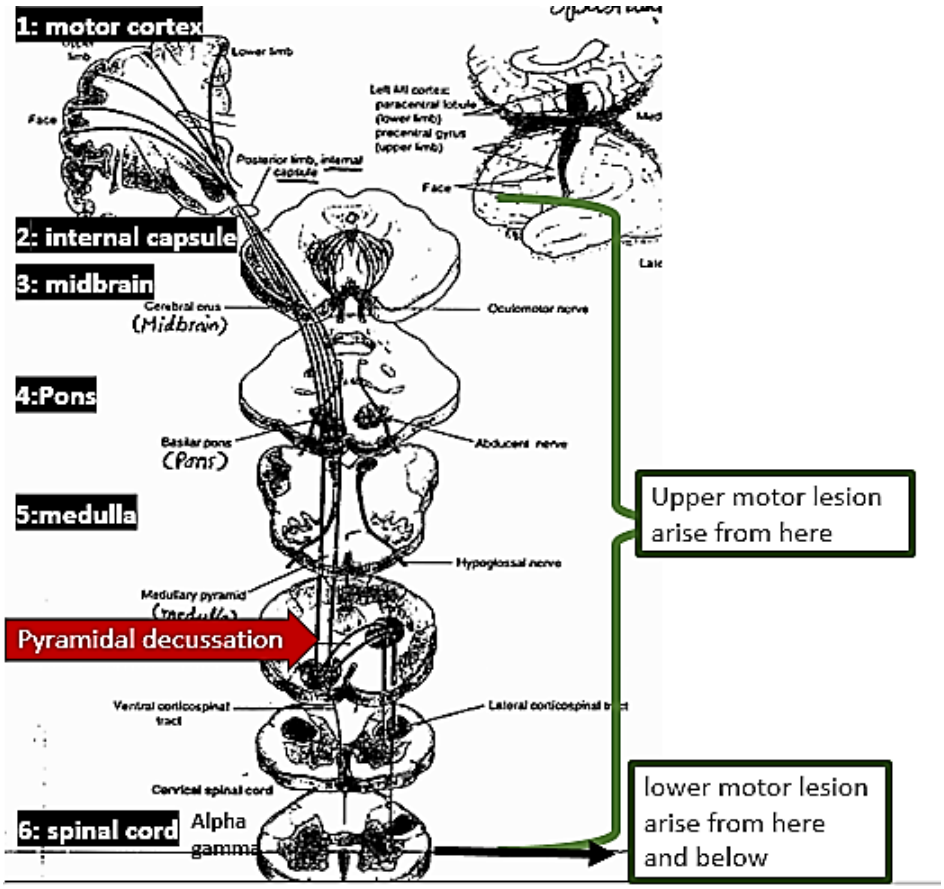
- The **lower** motor neuron lesion can rise if :

✓ Alpha or gamma neurons got traumatized, or any fibers below their level.

Remember that:

- The upper motor fibers could be traumatize above or below the level of decussation at the medulla (at what is called as the pyramidal/ motor decussation -indicated at figure1), this implies that if the lesion is superior to the medulla, its effects will be contralateral.
- Mostly the pyramidal tract originate from area 4 , while the extrapyramidal originate mostly from area 6 . (Originate= the cell bodies of the neural fibers reside in that area).

***Figure 1** : Upper Vs lower motor lesions possible locations.*



Upper motor lesions

Clinically upper motor neuron lesions will involve the pyramidal and the extrapyramidal motor tracts together. But we can do some experiments to observe each tract loss-of-function-effects alone.
To do so we must think of places where each type of these tracts run alone - without the company of the other tract-

We cannot think of the internal capsule as both types of upper tracts run there together, but at the medullary pyramid, the pyramidal tracts (corticospinal & corticobulbur) run there alone, so if we cut the pyramids in an experimentally animal we can observe **the loss of function effects of the pyramidal tract** , which are:

- ✓ Paralysis or paresis (ضعف أو شلل) .
  - depends on the severity of tissue damage.
  - The patient will mostly present with this sign , he will come complaining of not being able to hold his tea mug or close his clothes buttons.
  - This sign can be contralateral [if the lesion is above the decussation] or ipsilateral [if the lesion below the decussation]
- ✓ **Hypo**tonia.
  - As the pyramidal tract is faciliatory or excitatory (mainly for distal flexor muscles), when its function is lost >> hypotonia resulted.
  - We can examine the tone by observing PASSIVE movements.
  - Flaccid = hypotonia = ارتخاء

Similarly, if area 4 was affected we will have contralateral flaccid **paralysis or paresis.**

- If we want to observe the extrapyramidal loss of function effects alone - experimentally- what area should we cut?
  What are the extrapyramidal loss of function effects:

- ✓ Paralysis or paresis.
- ✓ **Hyper**tonia. Why?
  - From area 6 descend inhibitory neuro- fibers for the pontine-reticulospinal tract (one of the extrapyramidal tracts), when the cortical inhibitory effect is lost because of the lesion the pontine-reticulospinal tract is **disinhibited** (=reactivated)→ this tract will be "super-functional" → a lot of firing impulses are released from it to the alpha and gamma neurons at the spinal cord gray mater (mainly stimulate gamma) → the stretch reflex is really highly active by now. Its effect on the upper limb flexor muscles and lower limb extensor muscles will be very high to the extent that the patient will suffer from → **spasticity/ rigidity** of these muscles.
    Note: stiffness &rigidity are a little bit different, basal ganglia lesions cause **rigidity,** while motor neurons lesions cause **spasticity.**

- WHAT IFs?-

- What if both area 4 and area 6 were affected together?

To answer this question we must be aware of these givings:

- ✓ pyramidal tract originate from area 4 , while the extrapyramidal originate mostly from area 6.
- ✓ The pyramidal lesion will cause >> hypotonia , while the extrapyramidal lesion will cause >> hypertonia.

The answer is that :

In most cases we will have initial **flaccid hypotonia** (pyramidal lost effect) at the first hours of trauma then after a day or two the patient will have **spastic hypertonia** (extrapyramidal lost effects).>>the interval between these two stages is called the **shock stage**.
note: till now no one had explained the underlying mechanism of this stage.

why we say (in most cases) ?
some cases will show with extrapyramidal loss of function from the very beginning.
so at the end all of these patients will suffer from **spastic hypertonia with paralysis /paresis** wither they enter the shock stage or not.

- What if the Lt internal capsule is affected (say by a stroke)?

We can think like in the above situations , in this area both pyramidal and extrapyramidal tracts run, so regardless of the shock-stage-thing the patient will suffer from right spastic paralysis + other things explained later.
Note: the internal capsule is supplied by middle cerebral artery (branch of the internal carotid), if it get occluded >> stroke >> contralateral paresis or paralysis.
Another note: in most cases of upper neuron lesions we are talking about supra-decussation lesion so mostly the effect will be contralateral (for the tracts that do cross), but also u must be aware of the decussation thing.

- So, by now we can understand the clinical S&S of upper motoneurons lesions (pyramidal + extrapyramidal tracts together) :

1. Spasticity / hypertonia
    - Because of the above mentioned mechanism of pontine disinhibition. >>> gamma become hyperactive >>> more and more stretch reflex >>> more tone.
    - This spasticity appear mostly on the antigravity muscles (flexors of upper limb and extensors of lower limb).
    - How to examine this sign ? Refer to figure2
      by trying to extend the hyper-flexed arm (at the affected side), when trying to do so, you will face huge resistance at the beginning of extension then suddenly gives away.
      explanation :
      when we first try to extend the arm , we apply more stretch on flexors muscles (biceps & brachialis) >> the muscle spindle is activated >> stretch reflex >> the muscle tone increase>> that's why we have initial resistance . But after a while the golgi tendon organ get activated >> initiate reverse stretch reflex >> the muscle relax and gives away.
      This phenomenon is called **clasp knife rigidity/ spasticity**.
      الأمر مشابه لفتح "الموس الكبّاس " تبع "الزعران"

2. Hyper-reflexia
    - Examine by doing deep tendon reflex test, which you compare tendon reflex of the normal and the affected limbs.
    - **The deep tendon reflex = jerk** , shown in figure3 , is a type of stretch reflex called dynamic stretch reflex.
    - The underlying mechanism is like hypertonia >> increase in gamma activity due to disinhibition of pontine reticulospinal tract.
3. Clonus
    - Shown in figure4.
    - It is rhythmic contractions of muscles when they are subjected to sudden sustained stretch.
    - How is this test done?
      place your arm on the patient plantar of the affected foot , then we

apply sudden stretch in upward direction with our palm remain supporting the foot planter. If the foot starts a series of dorsal and planter flexion → this is a +ve clonus.

Muscles of the posterior compartment (gastrocnemius and soleus) of the leg are the muscle responsible for planter flexion.

Why dorsal flexion ?

when there's planter flexion the anterior muscle of the leg will stretch → get activated → induce dorsal flexion. (also my hand which is still placed on the patient's plantar aids in dorsal flexion)

Note: this clonus sign may be normally absent in the patient.

- The underlaying mechanisim is similar to hyperreflexia and hypertonia → which is firing of gamma due to pontine reticulospinal tract disinhibition.

4. Positive **Babinski's sign** :
   - Figure5
   - How to perform this test?

     There are many ways; one of them is to stimulate the skin of the plantar at the affected side with a hard object from the heels forward.حرّك مفتاح على إخمص قدم المريض من الخلف للأمام في الجهة المصابة
   - The normal response is plantar flexion (the toes move downward) if the patient's upper motor neurons are affected his big toe will move upward (halluces dorsal flexion) with fanning of the remaining toes(abducting) باقي الأصابع بتبعد عن بعض. just look at figure5
   - This sign must be examined early (when the patient present) to confirm upper motor neuron lesion when the patient present with hemi-paralysis.

     Note: forget about examining the spasticity and other reflexes at the first day (or even the first week), they won't be +ve. DO BABINSKI!
   - If the patient have hyper-reflexia without +ve babinski's sign → the hyperreflexia is of no value (this hyper-reflexia might be caused by stress).

The underlying mechanism of this sign is the absence of the pyramidal tract, PROVE IT, what is the evidence of this causality!

- This sign is also positive in these situations which also have absence of pyramidal in common:

  - New-born (the pyramidal is not functioning as it's not myelinated yet).
  - During deep sleep (the cortex is not active>> no pyramidal).
  - During General anesthesia (the cortex is not active>> no pyramidal).
  - Coma patients.

- Also , remember in flexion reflex we have said that the pyramidal tract has to do with regulation of interneurons function (as inhibitory or excitatory neurons) we also have said that if the pyramidal is absent these interneurons function will flip-flop (the inhibitory become excitatory , and the excitatory become inhibitory) and that's what is happening here → normally when we stimulate the plantar of the foot some interneurons must initiate flexor reflex to induce plantar flexion , but with the absence of the pyramidal the interneurons function is disturbed (flipped) and instead of inducing plantar flexion other movements are induced (described above).

So, by this we can say that the hypothesis of (+ve Babinski- induced by absence of the pyramidal tract) is:

5. Absence of abdominal flexion reflex at lesion side, normally if u stimulate the abdominal muscle they will twitch, but in case of upper motoneuron lesion this reflex is absent.
6. Absence of cremasteric flexion reflex at lesion side, normally when u stimulate the upper part of the thigh medially, the near testis will erect but

in case of upper motoneuron lesion this reflex is absent. * this reflex is only for males (rarely done).

A typical upper motoneurons lesion patient is showed in figure6.

***Figure 2**: spasticity and clasp knife response*

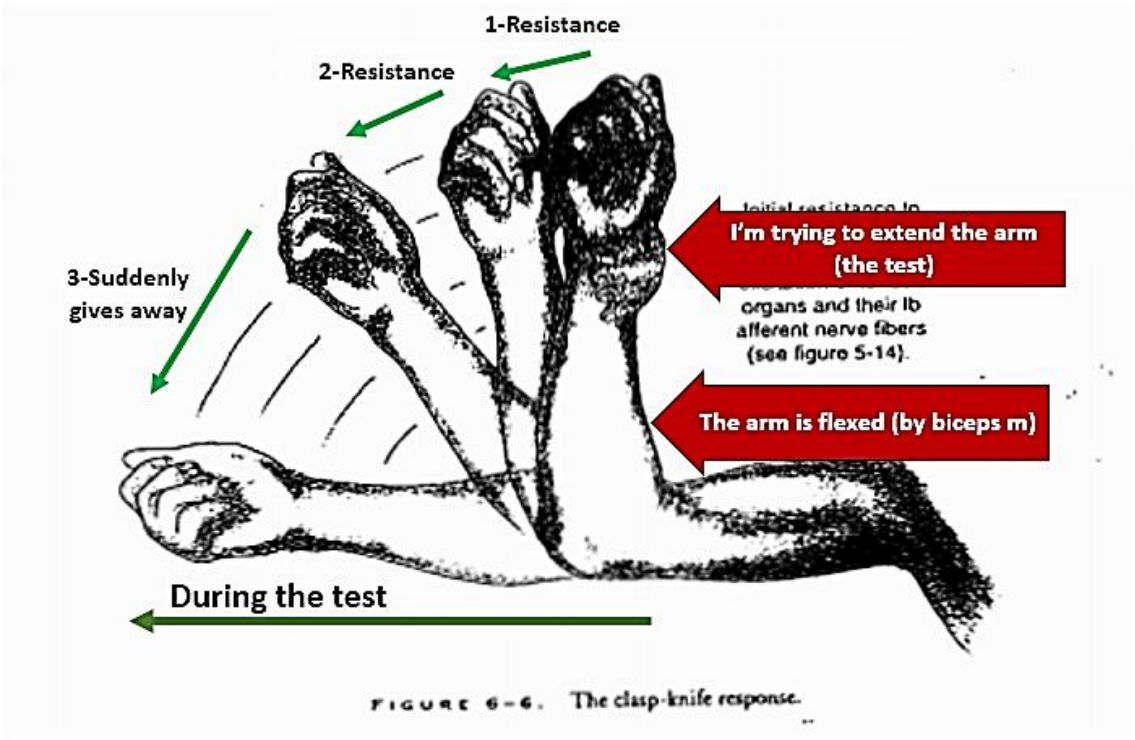


FIGURE 6-6. The clasp-knife response.

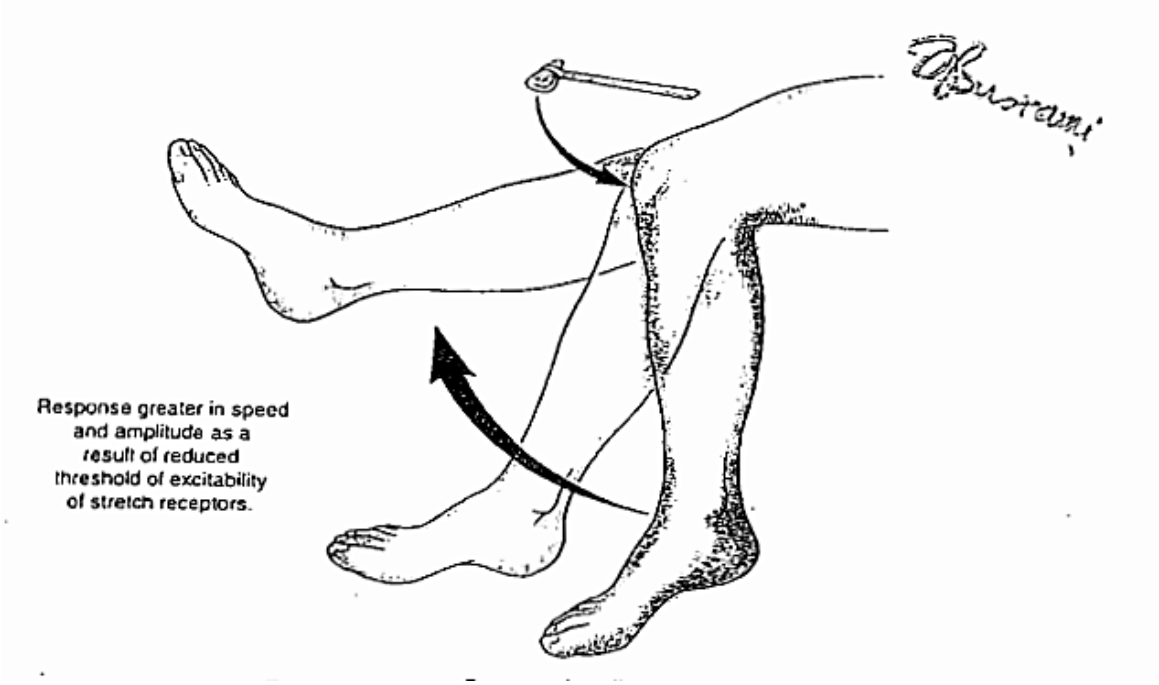


Figure 3: ***deep tendon reflex (patellar tendon reflex).***

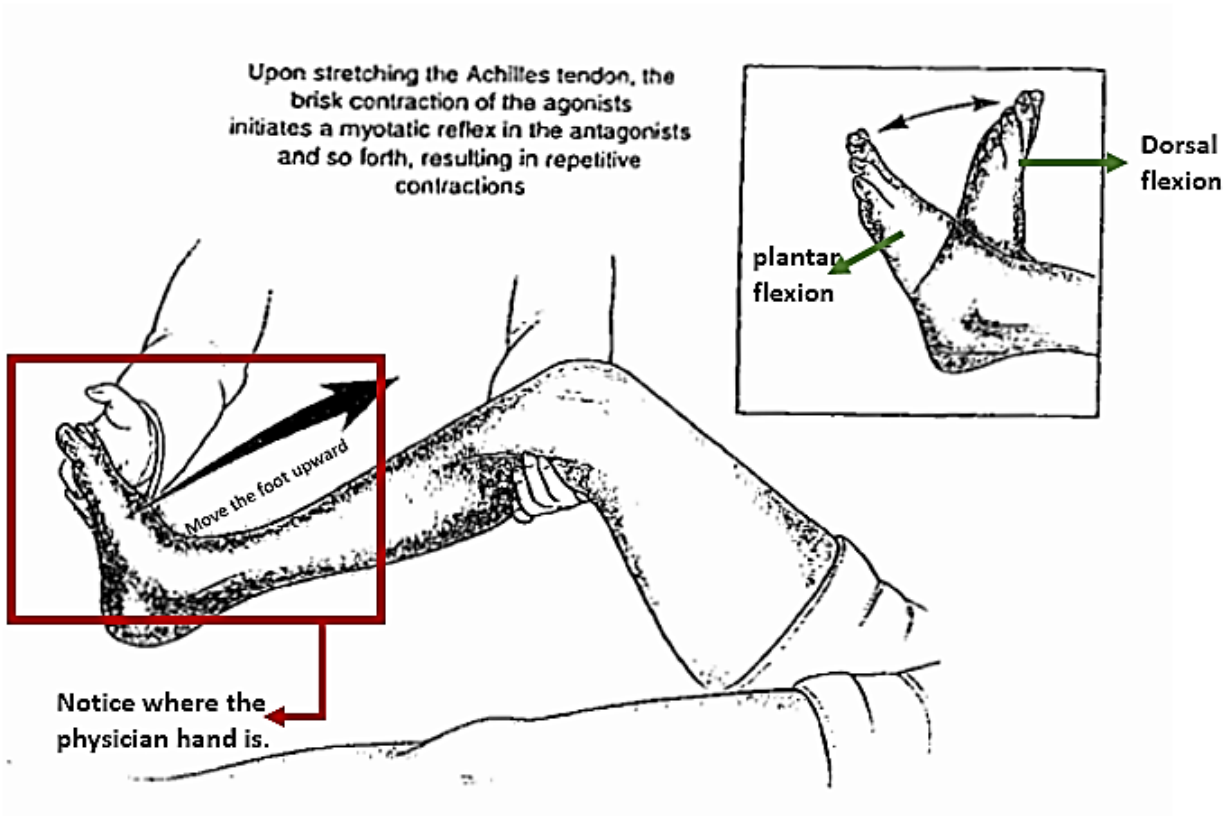


Figure 4 : ***clonus.***

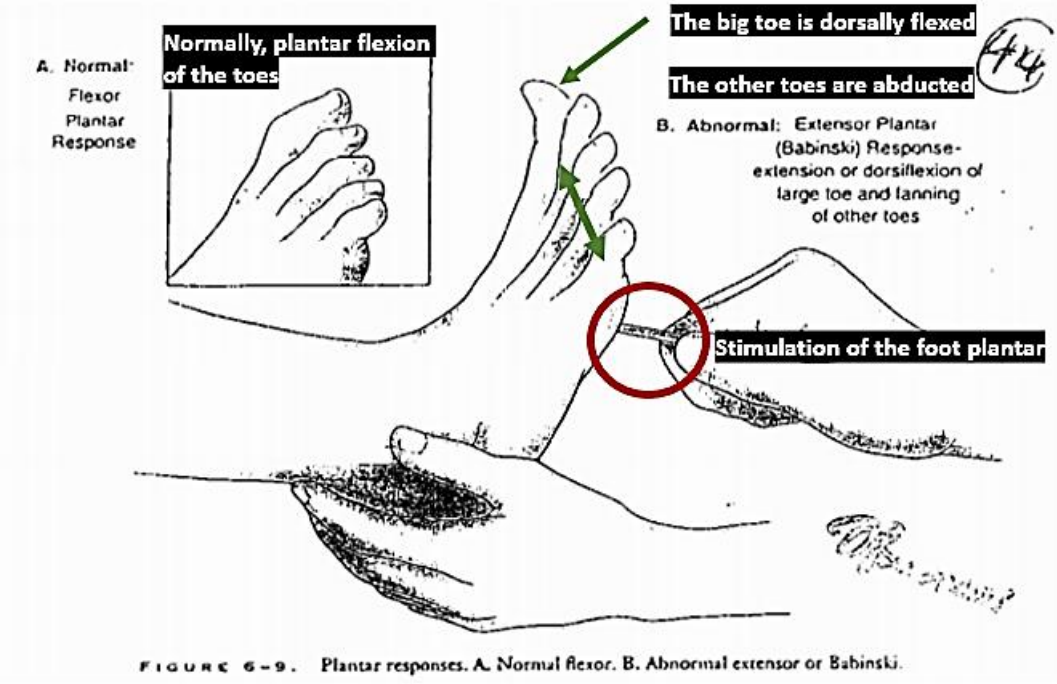


Figure 5 : ***Babinski's sign***

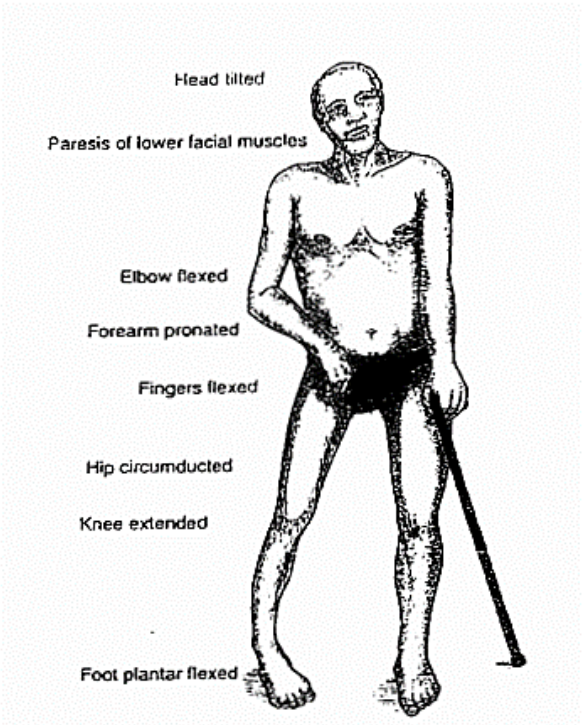


Figure 6 : *the gait of a typical upper motoneurons lesion patient , notice his leg and thigh while walking will be like one piece , his foot is plantar flexed..etc, u must be familiar with all S&S at this figure. can u tell which internal capsule is affected (Rt or Lt) -the answer is at the end of this sheet.*

In CNS lesions u must orient yourself , ask these Qs:

- Where is the lesion?
- What are the underlying S&S?
  Note: the patient presents to u complaining from **SYMPTOMS,** while ur tests on the patient reveal **SIGNS**.
- What is the underlying etiology?
  Is the lesion traumatic, neoplastic, congenital, inflammatory or vascular?
  →Remember strokes have mainly vascular etiology (thrombotic , embolic or hemorrhagic → most dangerous type).

- Some of the following information are repeated (from this lecture and others) but some new information are also present.
  For the upper motor neuron lesions where would the lesion be located?
  1) Area 4
  - ✓ Present at the precentral gyrus + anterior part of paracentral lobule at the frontal lobe.
  - ✓ Clinical correlation, the paracentral lobule represent the lower limb and the sphincters if a tumor arise from the falx cerebri (a meningioma) the patient won't be able to control his sphencters and his lower limb.
  - ✓ The body is presented upside down , precisely but disproportionately.
  - ✓ It is rare to affect all of area 4 collectively , the lesion is often localized to certain area of area 4. So the patient would have **limited paralysis** in one part of his body(the face / upper limb / lower limb..) or **monoplegia** (one limb is paralysis) .. this characteristic for cortical upper motor neuron lesions as the most common etiology is vascular event of the middle cerebral artery >> which emphasis on localization of the lesion (as it's a "branchy" artery)
  - ✓ Broca's area might be also affected as it is close to area 4. When it's affected the patient won't be able to speak "from a motor perspective".برتبط لسان المريض

    Note : here we must take the Rt and Lt side thing into account , if the patient is right handed his broca's is at his Lt hemisphere >> so only a Lt sided stroke will cause him to dysphasic . (the patient must presented with Rt sided mono-limb paralysis to be motor dysphasia).

- ✓ If area 8 (frontal eye field) , say the Lt area 8 is affected , to where will the eye balls move?
  -to the Lt.

2) At the internal capsule

- ✓ Contralateral Hemiparalysis /hemiplegia.
- ✓ The pyramidal and extra pyramidal tracts are affected.
- ✓ U must examine the reflexes , Babinski , the spasticity..etc.
- ✓ U must examine the cranial nerve specially the fascial nerve (lower face).
  - Remember if the patient has a stroke in his Lt internal capsule his mouth angle will be shifted to the Rt .

3) At the mid brain lesion

- ✓ Remember: cranial nerves no. III (oculomotor) & IV (trochlear) originate from the mid brain. So any lesion there won't just affect the pyramidal and the extrapyramidal tracts but also these nerves nucleuses.
- ✓ Signs of oculomotor lesion (oculomotor palsy):
  1. Dilated pupilالبؤبؤ يتوسع.
  2. The eye move down and out بتنحول لبرا.
  3. The eyelid moves downward الجفن بيسحل.
- ✓ U must ask the patient if his oculomotor palsy signs are congenital or has appeared after the stroke.
- ✓ Oculomotor nerve palsy signs appear ipsilateral to the stroke site , while the motor tracts hemiplegia will be contralateral. This is called **Alternating hemiplegia.** شلل الأطراف على ناحية وإصابة العصب البصري على الناحية الأُخرى.

Alternating hemiplegia:
Refers to a form of hemiplegia that has an ipsilateral and contralateral presentation in different parts of the body. (wiki but important)

4) At the medulla

a. Remember the medulla contains CN (9, 10,11, 12) , we are concerned with CN 12 =hypoglossal as it is easy to examine its loss of function:
   i. Ask the patient to show u out his tongue, the tip of the tongue is shifted toward the affected area. (ipsilateral) this is also a case of **Alternating hemiplegia ,** as the hemiplegia will be contralateral.
5) Below the level of pyramidal decussation and above the level of gamma and alpha, the paralysis will be ipsilateral and will affect the limbs present lower than the level of the lesion (if we cut the spinal cord at the level of T10 → the lower limb of ipsilateral side will be paralyzed).

To sum up , cranial nerves palsies can indicate us where the upper motoneuron lesion is located.

- If the patient hemiplegia at the Rt side + oculomotor pulsy at the Lt→ lesion at the level of midbrain (Lt).
- If the patient hemiplegia at the Rt side + hypoglossal pulsy at the Lt→lesion at the level of medulla (Lt).
- If the patient has Rt monoplegia with dysphasia / Rt shifted eye pupil / Lt shifted mouth angle the patient→ have cortical lesion
- Nerves lesions are ipsilateral while paralysis is contralateral (mostly).

- One last thing to discuss concerning hyperreflexia and upper motor neuron lesions.

Four Primary Reflexes

Reflex	Roots Needed for Reflex		Muscle Carrying out the Relex	
Ankle jerk	S1		Gastrocnemius	Planter flexion
Knee jerk	L2, L3, L4	Mainly L4	Quadriceps	Knee extension
Biceps	C5, C6	Mainly C6	Biceps	Arm flexion
Triceps	C7, C8	Mainly C7	Triceps	Arm extension

- To examine the deep tendon reflex (jerk) we knock on the tendon by a special equipment , these tendons are:

✓ To observe ankle jerk: knock on achilles tendon (tendon of gastrocnemius and soleus) when stimulated →induce planter flexion- nerve root S1.

✓ To observe knee jerk : knock on patellar tendon (tendon of quadriceps) when stimulated → knee extension – nerve root L2, L3, L4.

✓ Biceps (flexion of the arm)and triceps (extension of the arm) jerks are mentioned in the table above (memorize it) .

Rigidity Vs spasticity

- Both terms reflect hypertonia.
- The “spasticity” occurs due to upper neuron lesion and affect antigravity muscles which are flexors of the upper and extensors of the lower limbs (mainly).
- We use “Rigidity” term to describe the hypertonia in Parkinson’s disease. (which is caused by a basal ganglia defect) . more details with regard to this disease will be discussed in next lectures , but generally speaking the patient will have bidirectional resistance for both extensors and flexors (of the same limb , bidirectional hypertonia)
- The spasticity is combined with hyperreflexia while the rigidity isn’t. (important = no hyperreflexia in parkinson’s patients).

Lower motor lesions

Occur at the level of alpha and gamma neurons at the spinal cord or at a lower level.
It's simply denervation the muscle of its nerve supply.
The side affected will be ipsilateral to the traumatized side. (If I cut the Rt nerve , the Rt muscles supply by this nerve are affected).

S&S:

- ✓ Paralysis/ atrophy of muscles:
  - Limited to one muscle or group of muscles (which the cut nerve supplies).
  - Sever atrophy of the denervated muscle appears after several weeks of the defect . من عدم الاستخدام
- ✓ Hypotonia (flaccid), also limited to what the nerve supply.
  do u remember the stretch reflex , and the concept of closed circle that if cut any where the whole reflex is gone ? well ,here we do cut the circle (by cutting alpha/ gamma) >> no stretch reflex >> no tone (ie. Hypotonia)
- ✓ Hyporeflexia , due to loss of tone.
  - Clinical correlation , Polio virus attack alpha neurons and induce lower motoneurons lesion. The affected children must be referred to physiotherapist to make sure that the affected muscle won't be severely atrophied (the physiotherapist will keep stretching and moving passively to maintain the size of the muscle)
    Why we do not want the muscle to atrophy despite that the motor fx won't be back what so ever?
    Because muscle support joints and without them the bones will delocalized.
    (Recall how the rotator cuff muscles support the shoulder joint , which are : supraspinatus , infraspinatus ,teres minor , subscapularis).

- ✓ Fasciculation / fibrillation
  - Both terms reflect spontaneous activity of muscle fibers at rest.
  - The difference b/w these two terms that :
    - The Fasciculation can be with unaided eye as the contraction occurs to a group of motor units.
    - The fibrillation cannot be seen by unaided eye as the contraction occur to one motor unit or two.
  - Explanation of this phenomenon : injury potential.
    - Figure7
    - When there's a lower motoneuron lesion , at the area of the lesion (at the cut area) a spontaneous action potential arise due to opening of sodium channels , this AP runs throughout the nerve fiber down to the muscle fibers it normally supply >> group of motor units (which follow this nerve fiber normally) contract spontaneously. This is called Fasciculation.
    - With time Fasciculation will become fibrillation , how ?
      with time the affected nerve fiber degenerate and the injury AP occur at a lower level , at the level of single motor unit (look at the figure 7 ,to understand) انقباض في عدد قليل من الألياف، لا يُرى بالعين المجرّدة.
  - Clinical correlation a polio child will have muscle twitches during his sleep + fever ..

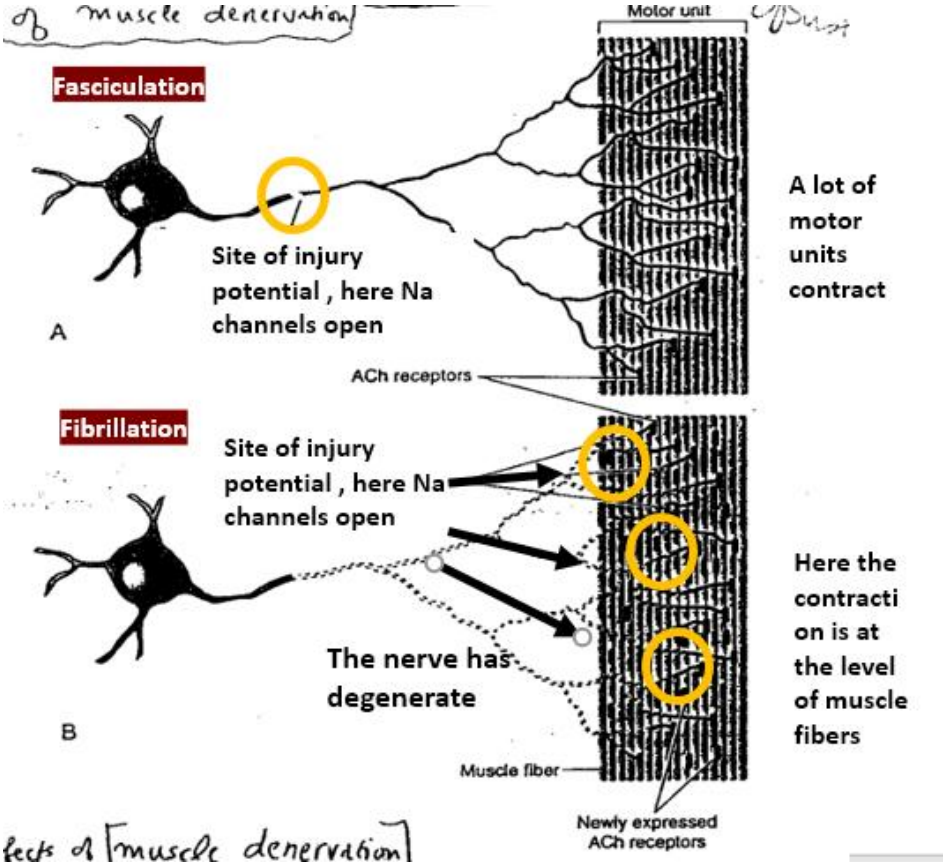


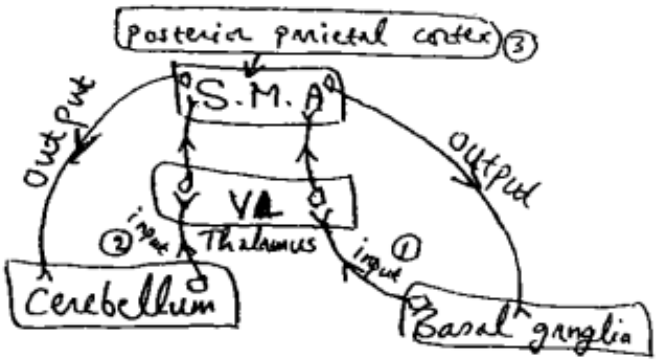
Figure 7 : ***Fasciculation will become fibrillation***

These information were mentioned by the Dr in Anatomy_7th lecture (sec-1):

<u>Area 6 & upper motor lesion</u>:

About area 6:

- Composed from premotor area (laterally) and supplementary motor (medially)
  both control axial & proximal muscles. (While the 1ry motor area control more distal muscles).
  Generally area 4 is responsible for motion execution (for simple movement), while area 6 concern with motion program of complex movements , also the basal ganglia , cerebellum (its lateral part) and the secretary ,the thalamus, aid in motion program. مثلث الحركة



✓

Also remember that any complex movement need an idea first before its program & execution , these motivative ideas come from the association cortex present in each lobe (frontal association, parietal association..) these ideas are translated into motor plans.
This motor plan is what really determine which muscles are needed for the movement, which muscles should be activated and which should be inhibited.
So, the idea arise in the association cortex , the program and planning in area 6 & the execution in area 4.
<u>Note</u>: area 4 can do simple movements by its own, or execute the area-6-planned movements.

Another note: it's area 6 (SMA) which interplay تتعاون with lateral cerebellum and BG not area4.

Area 6 (premotor area) receives a lot of inputs , as it's the programmer >> it need to know the state of the body to know what motor program is needed , it receives input from **the posterior parietal cortex** (which are **area 5 & 7** in the parietal lobe , just posterior to area 3,1, 2) in the past they used to call this area association sensory cortex .

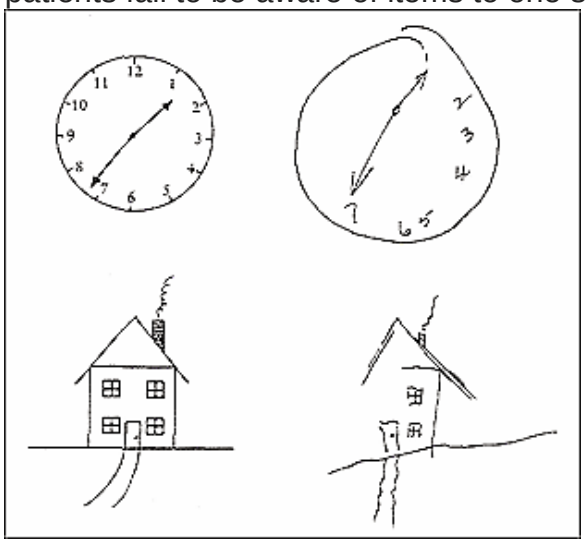
This area itself reserves its inputs from the skin (cutaneous) , proprioceptive (muscles and joints) , visual , hearing & also from motor areas. When **the posterior parietal** cortex receive all these information it can now generate **conscious map of the body** بترسم خريطة للجسم then this map is sent to the premotor area and the SMA (area6) so that they can create the suitable precise motor program.

- ✓ If the posterior parietal cortex (area 5+8) is affected (has a lesion) the patient will suffer from what is called **Hemineglect,** in which the patient ignore/ Deny the presence of one half of the body.

read more about this disorder from here (The Bizarre Disorder of Hemineglect)

(wiki)

Hemineglect, also known as **unilateral neglect**, hemispatial **neglect** or spatial **neglect**, is a common and disabling condition following brain damage in which patients fail to be aware of items to one side of space.



Note: the premotor area receives similar information from the cerebellum.
Another function for area 6 :

- ✓ **Premotor area** prepares the body to DO the motor action, like postural preparation for coming movement. يمكننا ملاحظة هذه الوظيفة بمراقبة وضعية لاعبي التنس قبل استقبالهم للكرة
- ✓ **Supplementary motor area** is responsible for complex bilateral movement (like these involving both hands). يعني مثلاً حارس المرمى لازم تكون هذه المنطقة شغالة منيح عنده

Finally,

.. Lesions of the supplementary motor area result, for example, in the inability to orient the hand correctly when reaching for a target or to coordinate the hands during bi-manual tasks.

END OF TEXT.