Team Medicine

19#

Approach to localization of a neurological lesion

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The Approach to a Patient with Neurologic Disease:

- Localization is important.
- Investigation modalities differ widely depending upon the level affected.
- Why localization is very important in neurology? Always in neurology we start with the history → physical examination → localization → differential diagnosis.
- It is wrong to start the differential diagnosis "without localization" after only the history and physical examination. So always in neurology we go by history (Hx) → physical examination (PE) → localization → differential diagnosis (DD).
- Differential diagnosis will always depend on accurate localization and temporal profile.
- If you know for example that this is a C-spine lesion and you know that it is acute, then your DD list will be shorter. If you don't localize the lesion, if you go by history, PE and you are not sure about localization (and I will go to examples like this to convince you how much important is localization before differential diagnosis), you will be wrong in your DD or it will be huge "very extensive", that means you will have to do a lot of investigations to reach the diagnosis.
- So you have to have it as a habit, that it's always history, PE then localization. Sometimes, but we do it for neurology residents, we trick them. After history and PE we ask them for DD, we don't remind them to localize the lesion. For medical students we will always remind you that you have to localize the lesion then you mention the DD.

Divisions of the Neuraxis:

1. Cortical Brain.
2. Subcortical area.
3. Brainstem.
4. Cerebellum.
5. Spinal Cord.
6. Root.
7. Peripheral Nerve.
8. Neuromuscular Junction.
9. Muscle.

- These are the vertical localization levels in neurological examination. Each level has specific features in Hx and PE.

Neurologic Examination:

- Higher Cortical Function, like general mental language.
- Cranial Nerves.
- Motor.
- Sensory.
- Deep Tendon Reflexes.
- Coordination.
- Special tests.
- Gait.

(1) Cortical Brain:

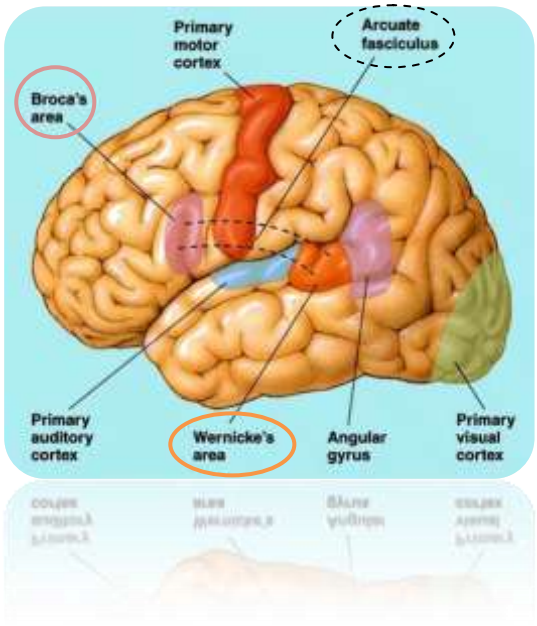
- Depends upon hemispheric dominance.
- Non-neurologists generalize:
  a) Right: visual/spatial, perception and memory.
     - Vision:
       - With occipital cortex: visual impairment or cortical blindness.

- Memory loss is a cortical function, but with acute pathology we don't see it. Although memory loss is non-localizing feature. It doesn't have specific localization.
- Hemispatial neglect, suppose that I am the patient and I have a lesion in the right hemisphere, I don't pay attention to the left hemisphere. Although I can say it. So it is not a visual field defect. I can see the left hemisphere, but I don't pay attention to the left hemisphere as much as I pay to the right hemisphere. So it is not a vision field loss. Students usually mistake it with visual field and blind-spot. Visual field means I don't see in the left hemisphere. Left hemispatial neglect means that I can see but I don't pay attention, I ignore this side, I pay attention to the other side.
- To pay attention to the left hemisphere I need activation from the right frontal lobe. The right frontal lobe makes me pay attention to that side. The left frontal lobe makes me pay attention to the right side. Suppose that there is a stroke, but didn't affect the afferent visual system, so I don't have left hemianopsia. If you do finger counting in the left side I can see it. But if you show me two stimuli (one in the right and one in the left) simultaneously, I will pay attention to the right one and will not pay attention to the left side. We call it hemispatial neglect; it means I can see it, but I'm not paying attention to it. And you can only detect it by providing the patient with two simultaneous stimuli, and you will see that he can pay attention to one side only. It can be permanent and it can be transient. Another way to detect hemispatial neglect, we draw a line and we ask the patient to divide it into two halves, the patient will put it in the right side, he will only divide the right side into two halves, the patient will put it in the right side, he will only divide the right side into two halves, so it will be one quadrant and 3 quadrants. This method is called "line dissection" http://www.youtube.com/watch?v=rB4w_R5APp8&feature=related. Also when they walk they always hit with their left side of the body the doors and the wall, because they don't pay attention that much to this side. And typically we can see it with right hemisphere stroke. So it is affecting the left side more common and more permanent. When it comes with left mca stroke usually it is a transient, it will be in the right side hemisptial neglect and it will be transient, 2 weeks and it will disappear.

b) Left: language and language dependent memory.
   - Language:
     - In right-handed people "almost 97%", it is left hemisphere.
     - And left-handed people, 75% it is still left hemisphere.
     - The other 25% of the left-handed people can be bilateral dominance, that's mean sometimes language centers in the right, sometimes language centers in the left.
     - Very few people have everything in one side. So even with left-handed people, most of them, they have the language centers in the left hemisphere.
     - The second common one is bilateral dominance; it's not right hemisphere language.

- Through detailed examination, neurologists should lateralize and localize within a lobe.
- One of the important functions of the cortex: consciousness
- For the cortex to be impaired with a lesion, you have to have very extensive lesion bilateral.
- The level of consciousness starts for the cortical activating system from the brain stem then goes to the thalami then thalamo cortical junction, so for the cortex lesion to affect the level of consciousness, it has to be bilateral and extensive.

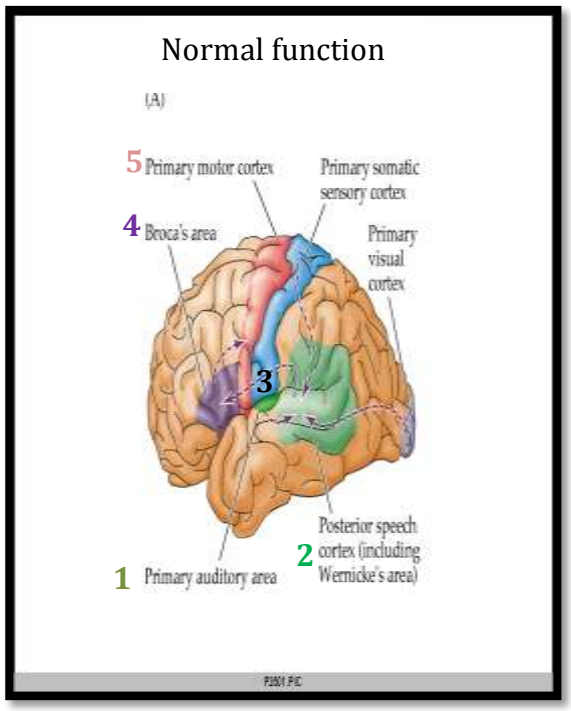
- That's why with stroke we don't see significant reduction in the level of consciousness, even if it is MCA "middle cerebral artery" stroke, which means half of the brain in one site is affected, still we don't see a trauma, because the cortical lesion to affect the consciousness has to be extensive and bilateral.
- Movement is not unique feature of the cortex. Spinal cord and peripheral nerves and brainstem lesions can cause weakness, so it is not specific for the cortex only.
- For cortical functions, we divide them into:
  a) Functions that have specific localization (ex: language "classic example").
  b) And there are functions that have no specific localization (ex: remote memory).Executive cortical functions, like calculation and executive decisions. So depends upon hemisphere dominance as we stated,



I. Frontal Lobe:

- What is specific about frontal lobe? Controlling the behavior. When we say frontal lobe lesion, that means that this is inhibited, he can't do anything, he doesn't pay attention to the surroundings and how much they judge him.

- Left:
  - Broca's Aphasia:
  - What is the difference between Broca's Aphasia and wernicke's aphasia?
  - Wernicke's aphasia is located in the temporal lobe.
  - House of language:
  - Always remember wernick's is close to the hearing area, that means it is important for what? Comprehension, because it is an input
  - The broca's is part of the frontal lobe, frontal lobe has the motor cortex, and the motor cortex important for the output.
  - What is difference between hearing and comprehension? Like when I hit the table, what part do you use? Hearing, so you didn't need wernick's
  - Wernick's and broca's for content of the language, not responsible of hearing. Hearing the voices is part of the hearing centers.
  - Is hearing unilateral or bilateral? Bilateral, that's why when a patient come with big hemispheric hemiplegic brain SPECT lesion---------- which happens in children , From the two ears there is bilateral innervations to both sides of the hearing centers. So one temporal lobe lesion will not cause hearing loss. So even if the patient has severe aphasia he can hear, unless he has two temporal lobe lesions then he will be deaf "central deafness".



- In higher normal functions: (full comprehension loop)
  1- In order to understand you have to hear first and this will be in the hearing center.
  2- then to wernicke's area to understand the abstracted word, simple comprehension wernick's area can be enough alone by itself, for abstracting thing it has to go to higher cerebral functions for deep understanding.
  3- to talk from higher brocal functions to the broca's area then as spoken language
  4- the repetition loop: to repeat it has to go from the higher center to wernicke's and from wernicke's to the broca's area.
- If the lesion in wernicke's →The patient will have good fluency but he cannot comprehend.
- If the lesion in arcaute fascicula → the patient can comprehend, and talk but he cannot repeat.
- The three major important aphasias are:
  1- wernick's aphasia (sensory or fluent).
  2- broca's aphasia (motor).
  3- conductive aphasia.

- Right:?
- Both:
- Primary motor cortex: motor homunculus
- supplementary motor cortex:
- Voluntary eye field
- prefrontal cortex: personality, initiative

II. Parietal Lobe:

- Right:
  - Left hemispatial neglect, mentioned earlier
  - Dressing and constructional apraxia:

    Apraxia: suppose the patient knows how to drive a car and after a lesion in the brain he lost his skill, he can't drive the car anymore. When you examine him there is no weakness, no visual loss, no dementia, but he lost his credibility to do what he previously learned as a motor skill.

    Somebody who you examined, he is strong in both legs, there is no weakness, no sensory loss, nothing, but he can't walk. If you ask him to move his leg while he is laying flat on the bed, he will do it. If you ask him to walk, he can't walk. This is called **Gait apraxia**.

    Another patient she has no weakness in her eyes, no facial weakness. When you ask her to open her eyes, she can't open her eyes. We call it **eye opening apraxia**.

    There are different kinds of apraxia: dressing apraxia, eating apraxia, etc.
- Left:
  - Gerstman's Tetrad: L/R confusion, finger agnosia, acalculia, agraphia without alexia
  - Werneke's Aphasia (with Temporal lobe)
- Both: cortical sensory modalities

III. Temporal lobe:

We talked about the hearing center. The visual memory is part of the temporal lobe. What will go wrong with a visual memory patient? He will lose his way. For example in his house he will forget the way to his room. This is typical in Alzheimer's patients.

- Auditory cortex: Heschel's gyrus
- learning and memory: mid/inferior gyri
- olfaction: limbic system
- Left: Werneke's Aphasia

IV. Occipital Lobe:

- Micropsia (they see objects small)/ macropsia (they see objects big).
- Visual hallucinations: elemental and unformed.
- Prosopagnosia: familiar faces, they can't recognize their family faces.
- Cortical blindness: striate cortices, normal pupil rx.
- **How can you tell the difference between the cortical blindness and eye blindness? How you can tell the difference between two patients, somebody who can't see because of severe damage in optic nerve or retinal pathology, and another patient who has severe or large tumor or hemorrhage in the occipital cortex causing the blindness?** The first patient with the retinal pathology he will have blindness in one eye ➔ cortical blindness. if I have left cortical lesion, what kind of visual loss I will have? Retinal anopsia. If I have left or right cortical lesion? It will be left or right hemianopsia. If I have bilateral cortical ➔ it will be bilateral, both eyes.
- **How can you tell a patient with a visual problem either to go to an ophthalmologist or a neurologist?** Anything affecting both eyes at the same time it means it is more central; he should go to a neurologist. Anything affecting one eye "unilateral", he should go to an ophthalmologist, unless it is optic nerve.
- In acute pathology and you are not sure to send the patient to a neurologist or an ophthalmologist.
    - Diplopia ➔ neurologist.
    - Ophthalmoplegia ➔ neurologist.
    - Bilateral visual loss "homonymous hemianopsia or bitemporal hemianopsia" ➔ neurologist.

(2)Subcortical Brain:

- Deep white radiating fibers
    - weakness
    - sensory abnormalities
- **What do we mean by subcortical area? It is a white matter (axons). Is there any gray matter (cell bodies or neurons) in the subcortical area?** Yes, the Basal ganglia, which is composed of caudate, putamen, globus pallidus and thalamus. Some books mention the thalami as a part of the basal ganglia and some books don't mention the thalami.
- **What is unique about subcortical area?** Localization.
- **What things you can see with subcortical which you don't see with cortical?** Suppose there is a patient with sudden weakness, how can I know is it a subcortical stroke or a cortical stroke? Suppose the patient has the weakness in the right side (isolated weakness) versus somebody who has weakness with aphasia, weakness with visual field loss. Which one is cortical? The patient with weakness and aphasia ➔ cortical. Isolated weakness ➔ subcortex. Isolated numbness ➔ subcortex.
- In cortex it is usually bigger lesions with multiple domains (multiple deficits).

- In subcortex it is usually isolated and small lesions. Subcortical lesion can cause weakness, numbness, and attacks to the subcortical area. Typically it is one or maximum two deficits.
- **Which one is more severe, cortical or subcortical?** Cortical.

➢ Visual radiating fibers:
- Deep parietal: bilateral inferior homonomous quadronopsia.
- Deep temporal (Meyer's loop): bilateral superior homonomous quadronopsia.

➢ Basal Ganglia:
- Extrapyramidal signs.

- **The basal ganglia lesion what would it cause? What we call this kind of symptoms or deficits**? Extrapyramidal signs.
- **What is the difference between pyramidal and extrapyramidal?**
  - **Pyramidal:** weakness, spasticity
  - **Extrapyramidal:**
- Rigidity.
- Tremor.
- Chorea: dancing-like movements.
- Dystonia: prolonged muscle contraction, twisting-like movement, it isn't like dancing "chorea", it is more twisting.
  - Sometimes dystonia can be in the neck but it will be tremor-less, we usually see this in Parkinsonian patients or as a complication of Parkinson's treatment.
  - Cervical dystonia can be tremor-less also.
  - Hand and leg dystonia is usually with twisting and sometimes involving the back, which will be twisted backwards with hyperextension of the neck.
  - When you see extrapyramidal signs and symptoms it indicates a problem in the basal ganglia.
  - **Suppose I am a patient with a hemi-parkinsonism, I have Parkinsonism in only my right side, the lesion in my right basal ganglia or my left basal ganglia?** In the left basal ganglia (contralateral), because the motor decussation is in the medulla.
- **The extrapyramidal why is it called "extrapyramidal"?** Because it has an influence on the pyramidal system, they go together.
- **What part of PE is ipsilateral? What kind of deficit**? Cerebellar lesion.

(3) Brainstem:

➢ The Brainstem is basically spinal cord with embedded cranial nerves.

➢ **How you can tell the difference between brainstem and supratentorial?**
- We mean by supratentorial cortex or supratentorial subcortex that it is above the midbrain.
- What's tentorium? Part of the dura, separated from the posterior fossa from the mid and anterior fossa.
- What's below the tentorium? Brainstem and cerebellum.
- So when we say infratentorial it means either brainstem or cerebellum which is the posterior fossa.
- When we say supratentorial it means basal ganglia, subcortical white matter and cortex.

- Cranial neuropathies
  - **What's unique about brainstem?** Cranial nerves.
  - Brainstem is divided into 3 regions: midbrain, pons and medulla oblongata:
  - Cranial nerve number 1 doesn't pass by the brainstem.

- Cranial nerve number 2 (optic nerve) doesn't pass by the brainstem. visual afferent system(optic nerve, chiasm, radiation, visual cortex) , it has nothing to do with brainstem.
- Cranial nerves 3 & 4 are the cranial nerves for the midbrain.
- Cranial nerves 5, 6, 7 & 8 are the cranial nerves for the pons.
- Cranial nerves 9, 10, 11 & 12 are the cranial nerves for the medulla oblongata.
- To make it easy for you to memorize the region, suppose the patient has weakness and diplopia, what is the region involved? Midbrain.
- Suppose there is a patient with weakness, he can't swallow and his gag reflex is absent? Medulla oblongata.
- Suppose a patient has weakness and numbness in the face indicating trigeminal sensory lesion? Pons.
- So we always use the cranial nerve region to help us with the localization.
- Also brainstem reflexes can help with the localization, like light, and gag reflex.
- Always a reflex has an input and an output.
- Light reflex, input: optic nerve, output: occulomotor nerve. So, it's going through midbrain.
- Gag reflex, input: glossopharyngeal nerve, output: vagus nerve. So, both input and output are from medulla.
- Caloric test, input: vestibulocochlear, output: third nerve (because in output we look for nystagmus), caloric test is a big reflex because it is crossing one area from lower pons till the upper midbrain. Suppose a patient with very extensive damage in this area having a bilateral absent caloric reflex, he will have bad prognosis.
- Illustrated video for caloric test: http://www.youtube.com/watch?v=Vo00ZYOXDrQ
- In the brainstem the most important part is cranial neuropathies associated with weakness, numbness, and ataxia, whatever.

- Long Tract signs: (bilateral and crossed)
  - Corticospinal (pyramidal): motor.
    - The corticospinal tract can be affected causing weakness or spasticity.
  - Spinothalamic: pain/temp.
  - Dorsal columns: proprioception (position sensation) /vibration.
  - Autonomic dysfunction (LOC, eyes, mouth, heart, breathing).

(4) Cerebellum:

- Cerebellar Function
  - Pure cerebellar signs ➔ cerebellum
  - Cerebellar and long tracts' signs ➔ brainstem
- We said it is ipsilateral.
- **What will the patient have?** Ataxic gait.
- **If I asked you in the final exam to examine for cerebellum, what will you look for?** Intention tremor, Ataxic gait.
- When you have a cerebellar examination in the final, it has little from every part of the neurological examination.
- **When you assess the cognition and the language, what will you see?** Ataxic speech.
- **What is the difference between speech and language? or aphasia and dysarthria?** If I bring someone from the street and I cut his tongue, is he dysarthric or aphasic? He is dysarthric.
  - **Aphasia** is the language content.

- **Dysarthria** is the mechanical component of the spoken language (phonation and articulation). Somebody has a complication of surgery like hoarseness or injury in the tongue, this is dysarthria. The cerebellum can cause ataxic dysarthria, the content and comprehension are normal, but when he talks the tone goes up and down in unpredictable pattern.

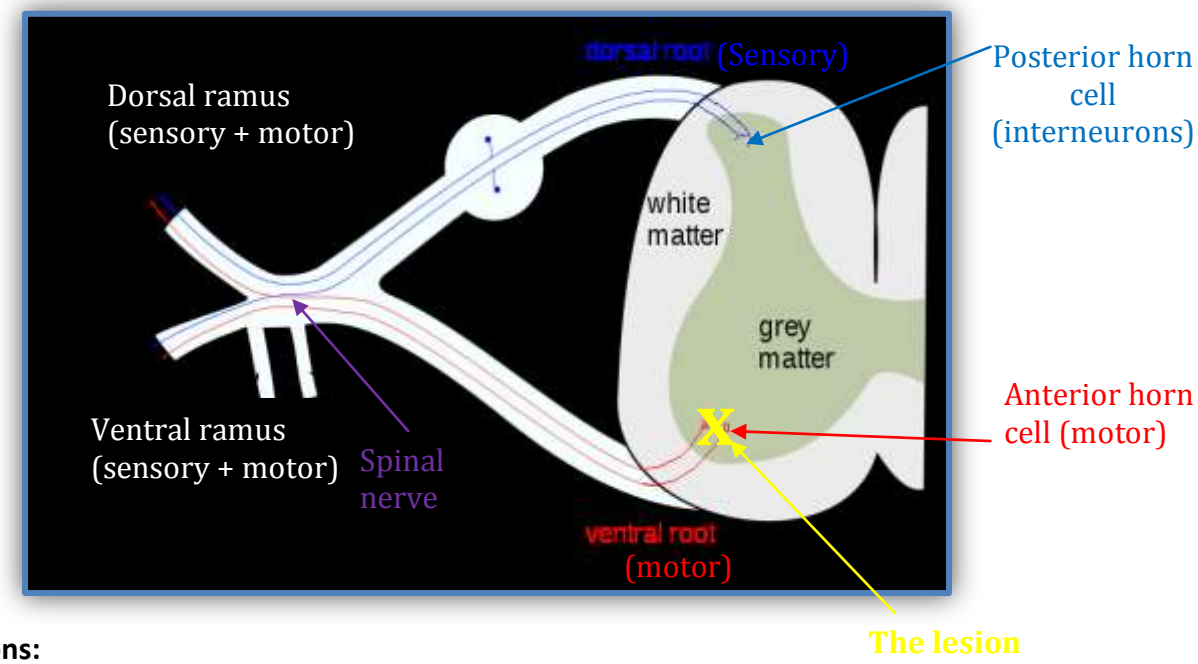
- **In cranial nerve examination, what will you see in somebody with cerebellar lesion?** Nystagmus.
- **How many types of nystagmus we have?** You can divide it by the direction, horizontal and vertical. Always there is a rapid phase and corrective phase, if the nystagmus doesn't have a rapid and a corrective phase we call it **pendular nystagmus, rotatory nystagmus**. You can see all kinds of nystagmus in YouTube. Cerebellar lesion can cause any kind of nystagmus.

(5) Spinal Cord:

- 3 Functions:
  - Motor ➔ UMNL
  - Sensory
  - Autonomic
- **Key ? sensory level**
- **What's unique about spinal cord lesion localization? What's the hallmark? When you bring a patient with a spinal cord injury what is the most important question?** Sensory level or the motor level (the level of injury).
- So the hallmark for the spinal cord injury to have a motor, sensory and autonomic deficit, this is the complete classic picture.
- Anything affecting the legs and sparing the arm, or it has a sensory level to the nipple it means spinal cord injury.
- Whenever you examine a patient and you are suspecting a spinal cord injury, try to identify the spinal level of injury, whether it is sensory level, anterior or posterior.
- Suppose a patient have weakness in both legs and normal upper, he also has spasticity in the legs with hyper-reflexia, where is the lesion? Dorsal or thoracic. It is not lumbar, because there is no spinal cord in the lumbar area, it is cauda equina (it is nerve roots so no spinal cord injury), so it will not cause hypertonia, it will cause hypotonia – nerve root injury-.
- If you see the legs are affected with spasticity, then you know it is CNS lesion.
- If the arms are spared, then you know that the C spine is spared.
- **What part of the CNS is below the c spine and above the lumbar?** Dorsal or thoracic spine.
- You can identify it by the sensory examination, you do the sensory examination and you go anterior posterior for like pinprick and temperature until you identify the level.
- You have to go by the map for the dermatomal and sensory level.
  - The level for the nipple is T4 – T5.
  - The level for the umbilicus is T10.
- **What about anterior horn cells, where are they located?** Anterior horn of the spinal cord. **What's the function?** Motor. **Is it upper motor neuron or lower motor neuron?** Of course lower motor neuron.
- When we say first and second order neuron, suppose I want to move my thumb here, the order has to come from the motor cortex in my right hemisphere, it will go down through the brainstem until it reaches C6 in the anterior horn level in the c spine (neck), then the anterior horn cell from there

will make the radial nerve to the extension of the thumb. So all what I need is two levels, only single synapse in the spinal cord.

- **What's the hallmark of anterior horn cell lesion or pathology like lower ALS (Amyotrophic lateral sclerosis)** – common disease in the states- **?** Flaccidity and muscle atrophy and sensory examination will be normal.
- This is the way you can differentiate between anterior horn cells or motor neuron disease from peripheral neuropathy. The sensory examination is normal, but in different neuropathy we will see motor and sensory deficit going with single peripheral nerve distribution or multiple nerve distribution. In anterior horn cells you will see pure weakness, fasciculation and atrophy, but sensory examination is totally normal, why? (see the pic. bellow)



- **Motor neurons:**
  - Pure motor deficit(s)
    - Upper (primary Lateral sclerosis) in motor cortex
    - Lower motor neurons in spinal cord
  - Spread:
    - leg ➔ arm ➔ bulbar
    - bulbar ➔ arm and leg
  - Of course there is a pattern for the anterior horn cell weakness, fasciculation and atrophy, it is either bulbar then it goes arm and legs or leg arm and bulbar , so always there is a pattern. It doesn't occur like left leg then after 2 years right arm, after 2 more years bulbar, it doesn't go like this. Typically there is a spread.

(6) Root/Radiculopathy:

- **In a case of disc affecting the motor root, what the patient will feel?** Radicular pain starts in the neck and goes to the arms.
- **Why it is radicular pain?** Because the nerve is going to the hand, it is referred pain.
- **What pattern of weakness?** It is lower motor neuron. If you have two patients. One with big c spine disc radiation compressing c spine. Another patient has c spine disc compressing nerve root

C6. What will be the difference between the two patients? In c spine injury the weakness will be at the level and below, so everything will be affected the hands and the legs. In C6 injury above the level of C6 will be spared which gave us sensory level and the kind of the weakness will be myotomal distribution (C6 innervating muscles will be affected) this means that C5 , C4, C7 will not be affected, it will be single root. Sometimes two discs or a one large disc compressing two nerve roots this is called Polyradiculopathy. So, in case of polyradiculopathy the weakness will be in C6 and C7, while C5 and C8 will be spared and also legs will be spared. So, if you got a patient with neck pain (radicular pain) and weakness in C6 muscle like the extensor this is nerve root ,while if you got a patient with neck pain and he came paralyzed or tetraplegic this is spinal cord injury (compression).

- Radicular pain: hallmark.
- Sensory abnormalities in dermatomes.
- Weakness in a myotomal distribution.

(7)Peripheral Nerve(presuming nonfocality):

- What's unique about peripheral nerves? Motor and sensory and sometimes autonomic.
- Everything will fit in single peripheral nerve distribution, it doesn't fit with myotomal or radicular distribution and it doesn't fit with central distribution.
- **What's central distribution?** Suppose you get a patient with hand numbness, how can I tell this hand numbness is median nerve (peripheral nerve), C6 (nerve root) or stroke, how can I tell the difference? By assessing the distribution.
    - If it is 3 fingers and a half and above the wrist is spared, I will think about median nerve and carpal tunnel syndrome.
    - If it is affecting also the forearm, I will think about nerve root.
    - If it is both sides of the hand but up to the wrist, so it doesn't fit with median, doesn't fit with ulnar, it can be stroke.
    - So stroke or central pathology doesn't really go by single dermatomal or myotomal distribution, it will affect like half of the limb. So you have to think about the distribution.
- Weakness (LMN)
- Numbness
- +/- autonomic

➔ All are consistent with PN distribution

(8)Neuromuscular Junction:

- Fatigability: hallmark.
- Weakness: proximal and symmetric.
    - muscles have normal bulk and tone.
    - EOMs, bulbar, arms, and legs.
- Sensation: preserved (normal).
- Autonomic will be normal.

(9) Muscle:

- Weakness
    - Symmetric
    - Proximal
    - + atrophy & absent DTRs

- What's unique about myopathy? A pure motor deficit, muscle weakness, pain, cramps and almost always proximal bilateral (both thighs, both deltoids and biceps), rarely it can start distally then spread proximally. The common pattern that I want you to always think about is proximal symmetrical.
- Sensation is normal
  - though patients complain of cramping, & aching
  - myalgia
- Autonomic will be normal

Few Things to Remember:

- Accurate Hx and PE are needed.
- Some neurologic diseases hit more than one level in the neuraxis ➔ include all involved. Always in localization you have to be inclusive, if it could be a muscle and a nerve, I will put the two as possible localization then I will start differential diagnosis. You have to be inclusive not exclusive. If it fits in 2 localization say two localizations are possible , it can be here it can be there, differential diagnosis for this localization is 1- 2- 3- , differential diagnosis for that localization is 1- 2- 3-. So always be INCLUSIVE.
- Never fabricate part of the exam, if you have PE that is incomplete or wrong, your localization is wrong, your DD and management is wrong.
- Always localize before making DDx list.
- The localization plus the tempo of progression allow one to narrow a differential diagnosis.

Case-1:

32 y/o male with reduced endurance and mild weakness in his legs. His older brother has weakness too. P/E: mild atrophy in legs with fasciculation but increased muscle tone. (Normal) sensory exam. Localize?

Atrophy ➔ lower .

increased muscle tone ➔ upper.

Mix. so you think about ALS.

Motor Neuron Disease

➔ Familial ALS

Case-2

55 y/o lady with sudden diplopia and weakness in Right (Face, Arm, Leg). Localize?

Brainstem, where in the brainstem? Midbrain, because of the diplopia. Because it is sudden most likely stroke

Midbrain lesion

➔ Ischemic stroke

Case-3

75 y/o lady with increased misnaming stuff over 2 years (she has problem with naming). P/E: decreased lexical fluency and some paraphasic errors (problems in the language). No other neurological signs. Localize?

Cortex, where in the cortex? Left frontal.

Why it is not temporal? Because we didn't comment here about comprehension, everything is about fluency, everything is output. What is the part? broca's.

Lt frontal lobe

➔ Frontal dementia

Case-4:

35 y/o man with Rt blindness, dysarthria, dysphagia, and weakness in Rt arm and leg plus Lt leg and sensory level at T-4. Localize?

Right blindness, I should think about optic nerve.

Dysarthria and dysphagia, I will think about medulla.

Sensory level T4, immediately think about spinal cord.

This is multi-level.

Multiple lesions

➔ At least brainstem and T-spine

➔ MS (multiple sclerosis)

This is why I was telling you, if localization didn't fit in one area, try to be inclusive. It is not always a single lesion in neurological lesions, sometimes it is multiple

Case-5:

16 y/o girl with gait ataxia and poor coordination in 4 limbs + nystagmus when looks to Rt side. Localize?

Cerebellar

➔ Spinocerebellar ataxia

Case-6:

50 y/o lady with diplopia on/off worse at evenings

In exam: partial ptosis in Rt eye. Localize?

NM junction

➔ Myasthenia gravis