CENTRAL NERVOUS SYSTEM

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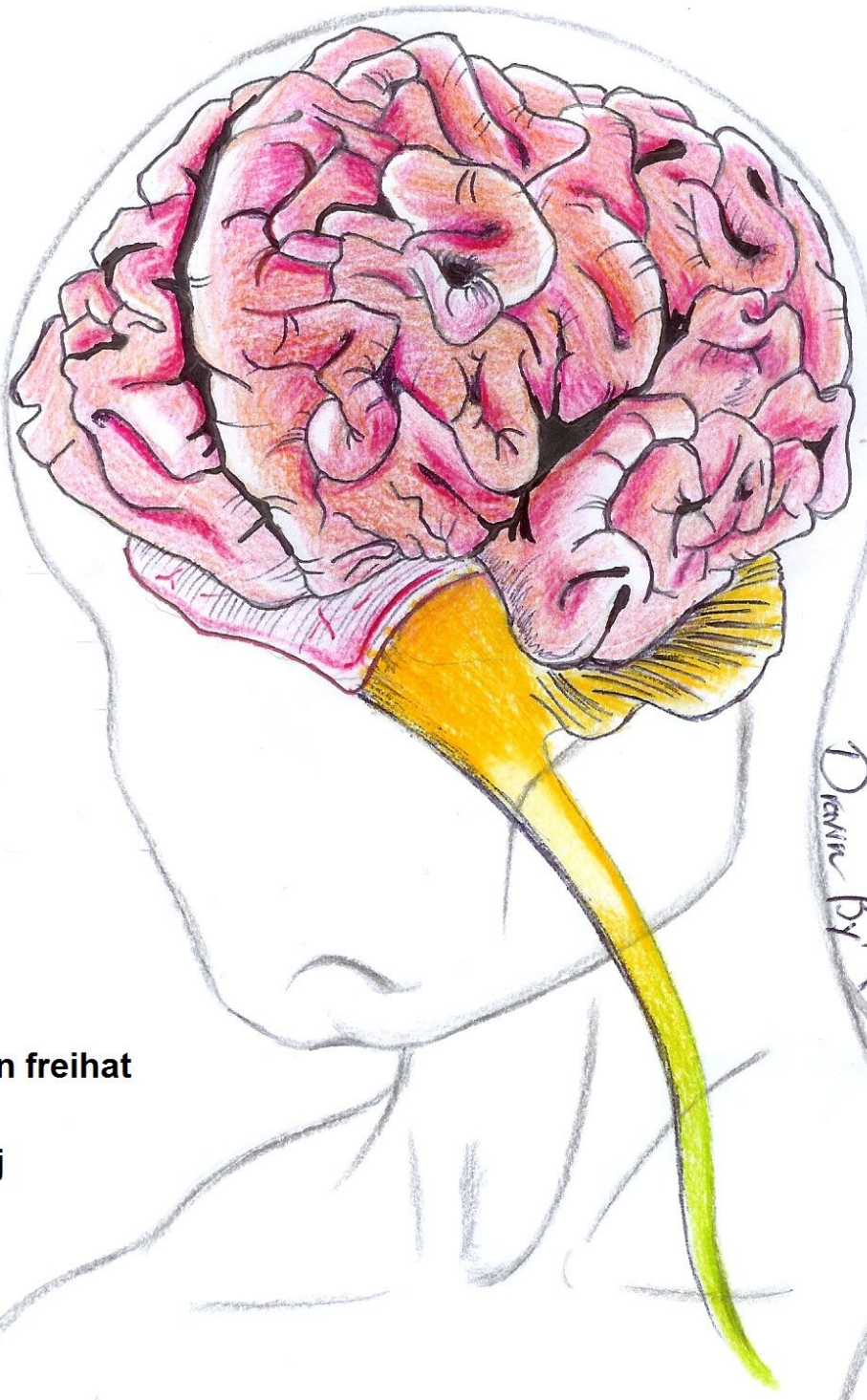

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MOTOR PATHWAYS

Pyramidal Pathway

Yesterday, we talked about 2 major pathways; the corticospinal and corticobulbar and we called them together "pyramidal pathway" because they descend from the cortex down to the pyramid.

REMEMBER that there's what is called pyramidal decussation. 90% of the fibers cross to the opposite side at the level of the medulla. If there's a lesion above the decussation (at the level of internal capsule, brain stem, and vertebral cortex), the effect is contralateral. But if it is below the decussation, the effect is ipsilateral.

* CORTICOSPINAL TRACT

As a result of the decussation; ***two*** pathways are formed : lateral corticospinal tract and ventral corticospinal tract.

The lateral corticospinaltract extends throughout the spinal cord and is found in the white matter. If you take sections in the cervical, thoracic, lumbar, and sacral regions, you will see the lateral corticospinal tract. The nerve fibers of corticospinal tract synapse with alpha and gamma motor neurons mostly through interneurons, not directly. Other 3% of the fibers, which arise from the large pyramidal Betz cells in lamina V of the cortex, synapse directly with alpha and gamma and enable us to do precise movements,منختار عضلة عضلة.

What is the benefit of those interneurons? To excite a certain muscle and inhibit another that opposes its action. For example, suppose that one interneuron excites alpha of biceps, while the adjacent interneuron will inhibit the alpha of triceps يعني ضربنا عصفورين بحجر (the agonist is stimulated and the antagonist is inhibited). Sometimes, the antagonist is inhibited before the stimulation of the agonist (for example, the triceps is inhibited before the biceps is excited to make a proper flexion).

55% of the lateral corticospinal tract fibers will affect the alpha and gamma MNs of cervical region of spinal cord (brachial plexus of upper limb). 25% of the fibers affect the alpha and gamma of lumbosacral region (lumbar and sacral plexus of the lower limb). This means that if this tract is damaged, the muscles of the upper and lower limbs will be affected (the effect on the upper limb will be more because more fibers are involved). The lateral corticospinal tract acts on distal muscles more than proximal muscles, so if a stroke occurs, the patient won't be able to use his hand properly.

So, if the corticospinal tract is injured in the internal capsule, both limbs will be affected.

The ventral croticospinal tract fibers(10%) act on the medial alpha and gamma of axial proximal muscles on ***both sides***. If it's injured on one side, the alpha and gamma on this side will be stimulated from the other side.

So , If a patient comes to you with a stroke on the right side, the left side will be disabled and skill movements will be lost but he still can elevate his left arm, why? Because the proximal muscles of the left side receive fibers from the other side.

THE LATERAL CORTICOSPINAL TRACT acts on proximal and distal muscles but mainly DISTAL muscles.

THE VENTRAL CORTICOSPINAL TRACT acts on axial and proximal muscles on the same side and opposite side.

* CORTICOBULBAR TRACT

Pyramidal tract (corticospinal and corticobulbar) starts at upper motor neurons in area 4 and 6 but mainly 4, while the extrapyramidal tract is mainly from area 6.

Corticobulbar tract starts from area 4 and 6 and descends to the internal capsule and brain stem, and completes its pathway. Then, it synapses on alpha and gamma that are found in the brain stem and give *motor fibers* from certain cranial nerves such as the motor part of hypoglossal and trigeminal BILATERALLY, this means that when one side is injured, the other side can compensate.

(Remember that corticospinal tract acts on spinal motor neurons, and controls movement of the upper and lower limbs).

Corticobulbar tract fibers act on muscles of head and neck including facial muscles, the tongue, muscles of the larynx and pharynx, etc. Imagine if the corticobulbar tract acts unilaterally, all of these muscles will be affected if one side is damaged so we can't swallow which is very bad.

ALL cranial nerves of brain stem receive the corticobulbar bilaterally **EXCEPT**:

LOWER part of motor nucleus of facial nerve that supplies the lower facial muscles.

The motor nucleus of facial is divided into upper and lower parts. The upper part gives motor fibers to the upper facial muscles such as orbicularis oculi. The lower part gives motor fibers to the lower facial muscles such as orbicularis oris, buccinator, rotator anguli oris, depressor anguli oris, etc.

The perioral muscles act simultaneously. The right muscles pull the mouth angle to the right and the left muscles pull the angle to the left. If the muscles of one side are weakened or damaged, the angle will deviate to the other side.

The input to the upper part of the nucleus is bilateral (contralateral and ipsilateral). If the contralateral input is damaged, the ipsilateral input compensates and all muscles will work normally.

While input to the lower part of the facial nucleus is contralateral. If one side is damaged the lower facial muscles of the other side will be paralyzed.

Hemiplegia and hemiface (HH)

A case where the patient has a stroke in the internal capsule →and this is how the corticospinal and corticobulbar are affected:

If the corticospinal is affected in the internal capsule on the right side → the left hand and leg will be weakened.

If the corticobulbar of the left side are damaged, the right side of lower face will be affected ONLY and vice versa.

If a patient comes with hemiplegia (paralysis in one half of the body), we must examine the lower face by telling him a joke or asking him to show his teeth to notice if there's a deviation in his mouth angle.

So, every patient with hemiplegia, his LOWER face is affected. (HH), HH here is hemiplegia and hemiface not high hallucination :P :P

AGAIN:

- The upper cells of the motor nucleus of facial supply the upper facial muscles. They receive contralateral and ipsilateral corticobulbar fibers. If one side is affected, the other side compensates.
- The lower nucleus supplies the lower muscles. They receive contralateral corticobulbar fibers ONLY. If one side is affected, the muscles of the other side will be affected.
- Now let's consider a stroke affecting the corticospinal and corticobulbar tracts →
  - if the lesion is on the right side → paralysis of the left part of the body and lower face. The left part of the upper face is less affected and vice versa.
- *The most important info to remember here is*, when a patient comes to you with a stroke →and after finding a paralysis of his hand and leg →ALWAYS EXAMINE the lower face!

The facial nerve passes through the parotid gland before it reaches the face. In cases where the nerve itself gets injured, for example, if there's a tumor in the parotid gland, the nerve will be damaged. Here You should be thinking about how to differentiate? Well, here the upper and lower face will be affected → BELL'S PALSY

- The best way to diagnose Bell's palsy if the lesion is considerable →
- Ask the patient to close his eyes → he'll not be able to close the eye on the affected side.
- Ask the patient to smile → the angle of the mouth will deviate to the other side. If the <u>left side is damaged → the mouth will deviate to the right.</u>

I think the doctor forgot to talk about the other nerve that receives contralateral corticobulbar fibers which is <u>the part of cranial nerve XII that supplies the tongue</u>.

Extrapyramidal Pathway

* RUBROSPINAL TRACT (rubro means red)

It originates in the red nucleus in the midbrain. The axons of this red nucleus go down and *cross immediately* and descend in the midbrain, pons, medulla and spinal cord. Then, they synapse with alpha and gamma.

EVERY motor pathway starts in the upper motor neurons and ends in the lower motor neurons where they synapse on the alpha and gamma.

The rubrospinal tract is very close to the lateral corticospinal tract in the spinal cord. They form →the LATERAL MOTOR SYSTEM. They affect the motor neurons laterally (the lateral alpha and gamma) that supply the DISTAL muscles mainly with little effect on the proximal muscles.

Like corticospinal tract, rubrospinal tract synapses with alpha and gamma through interneurons. We have excitatory and inhibitory interneurons. If the red nucleus is stimulated, the flexors will be excited through the excitatory interneurons and the extensors will be inhibited through the inhibitory interneurons.

The input to the red nucleus comes from area 4 and 6 but mainly 6 of the cortex. The rubrospinal tract should be really called cortico-rubro-spinal

AGAIN : the lateral corticospinal and rubrospinal form together the lateral motor system because they affect alpha and gamma laterally that supply the DISTAL muscles mainly.

* RETICULOSPINAL TRACT

It originates from the ***reticular*** formation in the pons and medulla. Accordingly, we have two reticulospinal tracts:

1- Pontine (medial) reticulospinal tract
2- Medullary (lateral) reticulospinal tract

These two pathways apparently begin in the pons and Medulla, respectively, but in fact they receive inputs from the cortex so they are actually CORTICORETICULOSPINAL.

BOTH pyramidal and extrapyramidal pathways begin in the cortex.

Both reticulospinal tracts descend ipsilaterally from the upper motor neurons and terminate in the lower motor neurons through interneurons within laminae VII & VIII.

Pontine reticulospinal tract fibers have excitatory effect on both extensors and flexors but mainly on extensors.

Remember that rubrospinal tract affects the lateral region of spinal cord that supplies distal muscles but reticulospinal tracts affect the medial region that supplies the axial and proximal muscles.

LATERAL REGION OF SPINAL CORD → DISTAL MUSCLES OF THE LIMBS.
MEDIAL REGION OF SPINAL TRACT → AXIAL MUSCLES AND PROXIMAL MUSCLES OF THE LIMBS.

THESE AXIAL PROXIMAL MUSCLES are responsible for the posture such as standing & sitting and automatic movements such as walking and running.

Pontine reticulospinal tract apparently descends ipsilaterally but at the level of spinal cord, it crosses and affects alpha and gamma of the other side also. It has an excitatory effect on extensors (and flexors) of the shoulders and thighs for example.

Medullary reticulospinal tract has an inhibitory effect on extensors mainly (and flexors).

So, there's a **BALANCE** between both pathways**,** one pathway excites the extensors and the other inhibits them.

PONTINE reticulospinal tract originates from **pontine reticular formation** (nucleus pontis caudalis and nucleus reticularis pontis oralis "NOT IMPORTANT TO KNOW")

MEDULLARY reticulospinal tract originates from **medullary reticular formation** (nucleus reticularis gigantocellularis "NOT IMPORTANT ALSO")

** DON'T FORGET **
Reticulospinal tracts give fibers bilaterally (contralaterally and ipsilaterally).
Both pontine and medullary reticulospinal tracts are actually "CORTICORETICULOSPINAL"
BOTH pyramidal and extrapyramidal pathways start from the cortex.

* VESTIBULOSPINAL TRACT

VESTIBULO → BALANCE

Lateral vestibulospinal tract

Cranial nerve VIII → vestibulocochlear. It has 4 nuclei in pons and medulla. One of these nuclei is called lateral vestibular nucleus (Deiter's nucleus) that gives axons that synapse with alpha and gamma medially →means that it affects axial proximal muscles. They synapse with interneurons:
- excitatory interneurons → extensors.
- Inhibitory interneurons → flexors.

So MAXIMAL excitation of the extensors is made by:
1. Pontine reticulospinal tract
2. Lateral vestibulospinal tract

Lateral vestibular nucleus or Deiter's nucleus is inhibited by projections from the cerebellum. So if we remove the cerebellum from an experimental animal, we will find an increase in the activity of this pathway which means an increase in excitation → continuous contraction of the extensors.

Medial vestibulospinal tract

It starts from MEDIAL VESTIBULAR NUCLEUS and found in the junction between pons and medulla. Medial vestibulospinal tract doesn't descend alone; it descends as a component of an important bundle of fibers. This bundle is called medial longitudinal fasciculus (MLF) that contains descending and ascending fibers. MLF links the vestibular nuclei with the cerebellum and ocular nerves (III, IV, and VI).

Vestibular system (vestibular apparatus in middle ear) is stimulated when you move your head to the right or left. It coordinates the movement of the eyes with the movement of the head.

Medial vestibulospinal tract has an important role in VESTIBULOOCULAR REFLEX (when you move your head to the right, your eyes will move to the left thus preserving the image).

- **MULTIPILE SCLEROSIS**

  it's a demyelinating disease. If we lose the myelin sheath, the nerve will be damaged and it won't be able to conduct the impulse.
  If a middle age patient is complaining from nystagmus and double vision, so his eyes don't move together (MLF is damaged) → MS.
  Early diagnosis of MS is very important → delay progression of MS.

MLF links the vestibular nuclei (that is stimulated by head movement) with the cerebellum and nuclei of III, IV, and VI. => Movement of the eye opposes the movement of the head => focusing on certain point (vestibuloocular reflex)

* TECTOSPINAL TRACT

Tectum → posterior part of midbrain. It has 2 superior colliculi and 2 inferior colliculi.
- Superior colliculi → vision
- inferior colliculi → hearing

Tectospinal tract originates from the superior colliculus and descends to alpha and gamma in the neck that control neck movement towards light.

In fact, tectospinal tract is **cortico**tectospinal.

ALL PYRAMIDAL AND EXTRAPYRAMIDAL PATHWAYS begin in the CORTEX and descends together in the internal capsule:
rubrospinsal → corticorubrospinal
reticulospinal → corticoreticulospinal
** any lesion of the internal capsule (mainly vascular lesion) → both motor pathways will be affected. Internal capsule is a critical area and contains descending motor fibers *(pyramidal and extrapyramidal)* and ascending sensory fibers*(well talk about them later)*.
** Most stroke cases and CVA (cerebrovascular accident) → affects the internal capsule. So, hemiplegic patient has capsular hemiplegia mostly (in the internal capsule).

*** there's another classification of the MOTOR pathways according to their site of termination in the spinal cord into :*(and this alternative classification is only beneficial with the signs of the cerebellum injuries)*
1- MEDIAL MOTOR SYSTEM→medial alpha and gamma → axial & proximal muscles.
2- LATERAL MOTOR SYSTEM→lateral alpha and gamma →distal mainly.

<u>Lateral Motor system</u> includes:
1. lateral corticospinal tract
2. rubrospinal tract
→ distal muscles mainly (and proximal).
3. part of corticobulbar that affects the lower face. (facial VII)
4. part of corticobulbar that affects the tongue. (hypoglossal XII)

Remember: HH! → Hemiplegia and hemiface>>>
A lesion in the internal capsule → Hemiplegia → the lower face is affected → hemiface.

Medial motor system: affects alpha and gamma medially that affects the axial and proximal muscles responsible for posture

1- Ventral corticospinal tract.
2- Extrapyramidal pathway in general.
3- Part of the corticobulbar tract.

UMN LESIONS:

- Any injury in the pyramidal or extra pyramidal tracts before they synapse at the alpha and gamma *(cortex, internal capsule, midbrain, pons, etc...)* is called : Upper motorneuron lesions *(UMN LESIONs)*

In order to understand the signs of UMN lesions, we need to be familiar with some of these mechanisms or reflexes:

Stretch reflex to start with, also called myotatic reflex, deep tendon reflex, and jerk and the outcome of this reflex is the **tone,** which is the **partial contraction of the muscle.** In addition to the contraction you know that we need to relax the antagonizing muscle, when I activate the quadriceps I also need to relax the hamstrings, and how is that accomplished? Through interneurons as you remember.

Components:

a sensory nerve coming out from the receptor and it's called: **afferent** and passes along the dorsal root of spinal nerve, which have a central process that enters and synapse directly with alpha, this single synapse is called **monosynaptic reflex** and its exclusively the only monosynaptic reflex in our body and its importance is being a very rapid pathway of transmission in comparison with a polysynaptic reflex *(imagine a road with many traffic lights)*

Now the signal has reached alpha, and a motor fiber gets out from an alpha toward a muscle where the action happens.

Furthermore, the doctor mentions that a **passive stretch** in the muscle leads to its partial contraction, how is that possible and we used to take that the muscle either gets flexed or extended? According to the dr that's complete rubbish, he explains the idea where each muscle is formed of many motor units, when you stimulate a few of these motor units, you reach the **partial contraction** i.e. The **MUSCLE TONE**, and the stretch reflex function is to maintain the muscle tone!

Regarding the muscle tone: This muscle tone is neurogenic this means that if I interrupt any part of this functional loop I lose the tone! For ex: **poliomyelitis** which destroys the alpha, interrupting the reflex and causing the muscle to lose its contraction and tone.

Dx: you examine the intact side, by any random movement of the part where you find some resistance as a result of the tone and you find none of that on the injured side.

Importance of muscle tone: maintaining posture / the tone of the muscles in your lower limb directs the venous return toward your heart **(muscle pump)**

Passive stretch: any muscle length is a little bit shorter than the length between origin and insertion… sooo it's partially contracted and that means? MUSCLE TONE.

In conclusion, A receptor is a modified skeletal muscle fibers , which is called **muscle spindle** or intrafusal *(which is stimulated or excited by ? gamma)* that's surrounded by a capsule and connected with the surrounding fibers or the tendon of the muscle and affected by it, its peripheral fibers contain sarcomeres and are capable of contracting while the central parts aren't.

So if the muscle spindle gets stimulated it starts sending signals to alpha, and alpha in its turn starts to act on muscle fibers (extrafusal) causing contraction.

Now we have several ways of stimulating the muscle spindle:

1- Passive stretch
2- By gamma: it stimulates the peripheral parts of the muscle spindle >> contraction occurs and it causes shortening in both ends that will lead to stretch on the central part , this will stimulate the spindle and sends signals through 1A & B to alpha where the nerve gets out from alpha and works on contracting that stretched muscle.

Note: * **alpha MN** acts on the extrafusal muscle fibers (99% of muscle fibers)

* **gamma MN** acts on the intrafusal muscle fibers (1% of muscle fibers)