

UPPER AND LOWER NEURON LESIONS

Objectives:

- ❖ Appreciate **what is meant** by upper and lower motor neurons.
- **Explain manifestations** of upper and lower motor neurone lesions.
- ❖ Know effects of lesion in pyramidal tracts at various levels.
- **❖** Know **effects of lesion** in the <u>internal capsule</u>.

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Color index: Important - Further explanation - Doctors Notes - Numbers.

^{*}Please check out this link before viewing the file to know if there are any additions or changes.

Upper and Lower motor neurones.

"Performance of normal voluntary movement, the integrity of two sets of neurons is important"

Upper motor neurones:

- Neurons originating in the cerebral cortex and the brainstem, and Synapse <u>directly</u> and <u>indirectly</u> with the <u>anterior horn</u> cells or with the motor neurones of the <u>cranial</u> nerves (to activate them).
- Grouped into: pyramidal and extrapyramidal systems.

To initiate a voluntary contraction of a skeletal muscle, a LMN must be innervated by an UMN. The cell bodies of UMN are found in the **brainstem** and **cerebral cortex**, and their axons descend into the spinal cord in a tract to reach and synapse on LMN.

of UMN are found in the heir axons descend into the pse on LMN. Spinal cord Brainstem Corticospinal tract Lower motor neuron (cranial nerve) Lower motor neuron (spinal nerve)

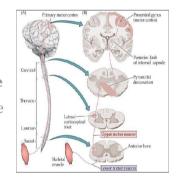
Motor cortex

Lower motor neurones:

They are:

- **Motor cranial nuclei** and their axons, motor fibers of the cranial nerves (3rd, 4th, 5th, 6th, 7th, 8th, 9th, 10th, 11th, 12th)
- **the anterior horn motor neurons** and their axons in the spinal cord. The motor nerves to skeletal muscles.

The LMN are found in the **ventral (anterior) horn** of the spinal cord and in **cranial nerve nuclei in the brainstem**. Axons of LMN of spinal nerves exit in a ventral root, then join the spinal nerve to course in one of its branches to reach a synapse directly at a NMJ ¹ in skeletal muscle. Axons of LMN in the brainstem exit in a cranial nerve. LMN are activated by UMN.



Upper motor neuron



(UMN & LMN) (Duration:5:38)



(<u>UMNL & LMNL</u>)(Duration:7:56)

Manifestations of upper and lower motor neuron lesions.

	UMNL	LMNL
Causes:	Cerebrovascular stroke due to	Lesion of the lower motor neurons as

¹ Neuromuscular junction

	 hemorrhage, thrombosis or embolism in the <u>posterior</u> limb of internal capsule → damage of both pyramidal and extrapyramidal fibers. Spinal lesion. 	 in (poliomyelitis). Damage of the motor nerves e.g. (DM² or alcoholism). Spinal root lesions or peripheral nerve lesion. (e.g. nerve injury by trauma or compressive lesion) 		
	<u>Characteristics:</u>			
1- Paralysis :	 On the opposite side of the body (contralateral. Widespread affecting half of the face, upper & lower limbs. Poor recovery. 	 On the same side of the lesion. Localized to muscles supplied by the affected segment only. Recovery may occur. 		
2-Muscle tone:	 Hypertonia of the spastic type in the paralyzed muscle. "Klasp knife type":resistance to passive movement then sudden release. What causes that? Loss of inhibitory effect of the cortical extrapyramidal area & increase facilitatory impulses of the (γ) motor neurons → facilitation of the stretch reflex. 	 Hypotonia or atonia: Flaccid paralysis (loss of tone in paralyzed muscles) What causes that?: Interruption of stretch reflex. 		
3-Deep reflex:	 Exaggerated deep reflexes of the affected side: (e.g. knee & ankle jerks). Clonus is present. What causes that? Release of stretch reflex from cerebral inhibition. 	Absent deep reflexes_in muscles supplied by the affected segment or motor nerves. • Clonus is absent.		
4-Superficial reflex:	Lost on the affected side. - Abdominal & cremasteric reflexes are absent. - The plantar reflex → +ve Babinski's sign. What causes that? Loss of supraspinal facilitation.	Lost on the affected segments only.		
5- Muscle wasting:	 Not significant. Cause: Paralyzed muscles are still innervated and can contract reflexly. 	 Marked (Disuse atrophy). Cause: Muscles cannot contract neither reflexly nor voluntary. 		

² diabetic mellitus.

	Spasticity saves muscles from wasting.	
6- Fasciculatio ns ³ :	• Absent.	 Present. Visible spontaneous contractions of bundles of fibers in the affected muscles.

Summary (From the slides)

	UMNL	LMNL
1-extent of paralysis	widespread	localized
2-site of paralysis	Opposite side to lesion	Same side of lesion
3-Tone of muscles	Spasticity (hypertonia) " clasp-knife spasticity	Hypotonia " flaccid paralysis
4- Deep reflexes	Brisk (exaggerated) tendon jerks	Diminished or absent
5- Superficial reflexes	Absent	Absent
6-Planter reflex	Extensor plantar reflex , Babinski sign (dorsiflexion of the big toe and fanning out of the other toes) , or just an upgoing toe .	Absent.
7-muscle waisting	No marked muscle wasting , but minor wasting may occur due to(disuse atrophy)	Marked muscle wasting (atrophy)
8-Clonus	Clonus present (rhythmic oscillation on tendon	No clonus
9-Fasciculatio ns (seen) . - Fibrillation potentials by EMG	No fasciculations No fibrillation potential	Fasciculations may be seen . & Fibrillation by EMG

Motor loss (Hemiplegia):

- Exaggerated tendon jerk & clonus : Due to increased supraspinal facilitation.
- Positive "Babinski's sign"



³ a small, local, involuntary muscle contraction and relaxation which may be visible under the skin. Deeper areas can be detected by EMG testing, though they can happen in any skeletal muscle in the body. <u>video</u>

Upper motor neuron lesion:

- **Definition**: It is the damage of upper motor neuron in the higher center or the descending motor tract.
- Causes: Trauma Tumor Vascular disorders as thrombosis or hemorrhage.

Site: Most common site of UMNL is the internal capsule.

The effect of a lesion in different parts of the motor system:.

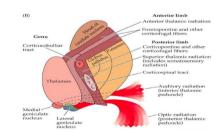
Lesions of pyramidal tract cause paralysis of the UMNL type below the level of the lesion. However, the side affected and the extent of paralysis vary according to the site of the lesion:

-In area 4:	In the corona radiata:	In the internal capsule:
This leads to restricted paralysis e.g. contralateral monoplegia (paralysis of one limb because area 4 is widespread so it is rarely damaged completely)	This leads to contralateral monoplegia or hemiplegia, depending on the extent of the lesion.	This often leads to contralateral hemiplegia because almost all fibers are injured

Internal Capsule

The internal capsule is the only subcortical pathway through which nerve fibers ascend to and descend from the cerebral cortex.

- -Consist of: (important to understand the lesions).
 - The anterior limb: contain descending fibers from the cerebral cortex to (red nucleus), pons (to cerebellum),thalamus, III, IV and VI cranial nerves.
 - The genu: contain corticobulbar tract.
 - The posterior limb:contain Pyramidal and Extrapyramidal fibers, somatosensory radiation, optic radiation and auditory radiation.



Damage of posterior limb of internal capsule:

Such lesion commonly called cerebral stroke is usually caused by thrombosis or hemorrhage of **lenticulo-striate artery** (a branch of the middle cerebral artery). Such lesion commonly called

cerebral stroke is usually caused by thrombosis or hemorrhage of **lenticulo-striate artery** (a branch of the middle cerebral artery).



5 **Motor Loss Sensory Loss** (loss of somatic sensations, vision and hearing) (Hemiplegia) 1- Contralateral Hemianesthesia: 1- Contralateral paralysis: Loss of only voluntary movements of the **DISTAL** Loss of all sensations on the **opposite side** of the muscles of the limbs, LOWER FACIAL muscles and body. muscles of the TONGUE (he cannot speak). • Remember: - Distal muscles of the limbs supplied by the lateral corticospinal tract which decussate in the brain -UMNL cause paralysis of the lower facial muscles because it only receive pyramidal fibres from opposite cerebral cortex. 2- Contralateral Paresis⁴ 2- Contralateral Homonymous hemianopia: (weakness, the muscles retain some movement) of the AXIAL muscles and UPPER FACIAL muscles. Loss of vision in the **two opposite halves** of the field of vision. WHY paresis not paralysis? Because the Axial muscles supplied by descending motor tracts other than CBS⁵ and whereas muscles of the upper face are ipsilaterally innervated by CBS. Muscles of the upper face receive pyramidal fibers from both cerebral hemispheres. **3-Bilateral Diminution**⁶ of hearing acuity. 3-Spasticity (increase muscles tone) of the skeletal muscles due to increased **Supraspinal Facilitation**⁷ to Y-motor **No complete loss** of hearing because both ears are bilaterally represented in both cortices. neurons. HOW? A lesion at the level of internal capsule **interrupts** the descending inhibitory cortical fibers which feeds the inhibitory reticulospinal tract leaving the

Clasp-knife type Spasticity

⁵ corticobulbospinal tract

reticulospinal active.

هذا النوع من السكاكين في البداية يفتح بصعوبة وفي النهاية

facilitatory vestibulospinal and

يصبح سهل وبسرعة مثل الليمب المتأثر ، واضح بالفيديو! (Duration: 0:11)

⁴ شلل جزئي

Lower motor neuron lesion:

- It is damage of the **lower motor neurons** (the spinal anterior horn cells and the cranial motor nuclei or their axons) resulting in **skeletal muscles paralysis**.
- Causes: Trauma Neuropathy.
- Effect Of Lower Motor neuron Lesion:
 - Structural Changes:
 - ★ In The Nerve (degeneration and regeneration).
 - ★ In The Muscle (atrophy and increase Ach receptors).
 - **★** Functional Changes:

	Functional Effect of Lower Motor neuron Lesion	
1- Flaccid Paralysis:	 - Paralysis of denervated muscles with loss of all types of movements (voluntary, postural and reflex). - All Reflexes are lost including stretch reflex resulting in loss of muscle tone and tendon jerk (Flaccidity). - The Extent of paralysis is usually limited to a small group of muscles. 	
2-Fasciculations ⁸ and Fibrillations ⁹ :	- Appears: few days or weeks after denervation Disappear when: the motor nerve completely degenerates or successful Re-innervation of the muscles occurs.	
A) Fasciculation	- Synchronous visible contraction of the motor unit (all muscle fibers) supplied by the injured axon. -Result from: spontaneous generation of action potential (injury potentials) in distal segment of the injured axon. Potential from the muscle itself	
B)Fibrillation	-As Degeneration of the injured axon continues, the axon terminals are now separate from the main axon and hence, from each otherInjury Potentials are still generated along the terminals leading to asynchronous contraction of the individual muscle fibers attached to terminals.	

	-Invisible to the observer and detect only by electromyogram (EMG), in fibrillation you will see giant wave.	
3- Denervation supersensitivity	-Denervated muscles becomes Supersensitive to Acetylc l -This is due to increase in the number of Ach receptors which cover the entire surface of muscle cell membrane.	holine.

4- Reactivation of Degeneration



موجود بالسلايدات وحلو مره نراجع جزئية الميد :Extrapyramidal Tracts

Origin:

From motor area (4) and premotor area(6) \rightarrow descend to (2)corpus striatum \rightarrow (3)Globus pallidus \rightarrow globus pallidus fibers pass to:

- Reticular Formation.
- Vestibular Nuclei.
- Red Nucleus.
- Tectum of midbrain.

→From these nuclei the extrapyramidal tracts arise.

Functions of Extrapyramidal tract (Multineuronal system):

Regulation of body posture, Involving involuntary movements of the large muscle groups of trunk and limbs.

★ Important: Complex and overlapping function exit between pyramidal and extrapyramidal systems for example while doing fine work like Needlework (pyramidal system) one has to subconsciously assume a particular posture of arms (extrapyramidal system) that enables to do your work.

-امثلة على حالات تشتغل فيها ال pyramidal وال Extrapyramidal مع بعض: writing.

★ References:

- 435 girls slides and notes.
- Kaplan Medical USMLE Step 1: Anatomy: Lecture Notes 2016.
- Wikipedia.
- Guyton and hall physiology textbook 13th edition.

