Physiology 435 team

UPPER AND LOWER NEURON LESIONS

Done by:

- **Summary:** Ola Alnuhayer

SUMMARY:

	Upper motor neurons	Lower motor neurons
They are	Neurons originating in cerebral cortex and the brainstem (pyramidal/extrapyramidal) or their axons "descending motor tracts"	Motor cranial nuclei and their axons ($3^{rd}/4^{th}/5^{th}/6^{th}/7^{th}/9^{th}/10^{th}/11^{th}/12^{th}$) In spinal cord they are AHCs and their axons
Lesion causes	• Cerebral stroke • Spinal lesion • Trauma / tumor	• Anterior horn cell lesions (poliomyelitis) • Spinal root lesions or peripheral nerve lesion (trauma/compressive lesion/DM/alcoholism)
Paralysis	❖ On the contralateral side of the body ❖ Widespread "face/upper & lower limbs" ❖ Poor recovery	❖ On the same side of the body ❖ Localized to muscles supplied by the affected segment ❖ Recovery may occur
Muscle tone	• Hypertonia & spasticity • Klasp-knife type "resistance to passive movement then sudden release" Cause: Loss of inhibitory effect of the cortical extra pyramidal area → ↑ on facilitatory impulses on gamma motor neurons "facilitation of stretch reflex"	• Hypotonia or atonia "flaccid paralysis" Loss of tone on paralyzed muscles Cause: Interruption of stretch reflex
Deep reflexes	• Hyper-reflexia on the affected side • Clonus is present Cause: release of stretch reflex from cerebral inhibition	• Hypo-reflexia / Areflexia in muscles supplied by the affected segments or motor nerves
Superficial reflexes	• Lost on the affected side "loss of supra-spinal facilitation" • Abdominal/ceremastric reflexes: absent • Planter reflex → +ve Babinski sign	• Lost on the affected segment only
Muscle wasting	Not significant "paralyzed muscles are still innervated and can contract reflexly" Spasticity saves muscle from wasting	Marked disuse atrophy "muscle cannot contract neither reflexly nor voluntary"
Fasciculation	Absent	Present

extra-pyramidal tracts	
origin	From area (6) + area (4) → descends to corpus striatum → Globus pallidus From the globus pallidus fibers pass to: Reticular formation - Vestibular nuclei - Red nucleus - Tectum of midbrain. From these nuclei the extrapyramidal tracts arise
functions	Regulation of body posture, involving involuntary movements of large muscle groups of trunk & limbs
note	Complex and overlapping function exist between Pyramidal and extra pyramidal systems Example: while doing fine work like needle work (Pyramidal system) one has to subconsciously assume a particular posture of arms (extra pyramidal system) that enables to do your work

UMNL "could be in different parts of the motor systems"	
In area 4	In the corona radiata
This leads to restricted paralysis. Example:. contralateral monoplegia (paralysis of one limb because area 4 is widespread so it is rarely damaged completely)	This leads to contralateral monoplegia or hemiplegia "depends on the extent of the lesion"
In the internal capsule "Most common site of UMNL. The arteries that supplies this area are lenticulo-striate arteries"	
Motor loss "hemiplegia"	Sensory loss "somatic/vision/hearing"
1. Contralateral paralysis (loss of voluntary movements) of the distal muscles of the limbs, lower facial muscles & muscles of the tongue. 2. Contralateral paresis (weakness) of the axial muscles and upper facial muscles. Axial muscles are supplied by descending motor tracts + corticobulbospinal tract"bilateral". Whereas muscles of the upper face are bilaterally innervated by corticobulbospinal tract 3. Spasticity (increased muscle tone) of the skeletal muscle due to increased supraspinal facilitation to motor neurons. A lesion at the level of internal capsule interrupts the descending inhibitory cortical fibers, which feeds the inhibitory reticulospinal tract leaving the facilitatory vestibulospinal and reticulospinal to act. This spasticity is of the clasp-knife type	1. Contralateral hemianaesthesia loss of all sensations on the opposite side of the body 2. Contralateral homonymous hemianopia loss of vision in the two opposite halves of the field of vision "lesion in the optic radiation" 3. Bilateral diminution of hearing acuity. No complete loss of hearing as both ears are bilaterally represented in both cortices "lesion in the auditory radiation"

LMNL		
structural effects	In muscles → atrophy & increase Ach receptors	In nerve → degeneration / regeneration
functional effects	1. Flaccid paralysis	2. Reaction of degeneration
	• Paralysis of denervated muscles with loss of all types of movements "voluntary, postural & reflex". The extent of paralysis is usually limited to a small group of muscle • All reflexes are lost including stretch reflex resulting in loss of muscle tone and tendon jerk (flaccidity).	-
		3. Denervation hypersensitivity
		Denervated muscle becomes supersensitive to acetylcholine. This is due to increase in the number of Ach receptors which cover the entire surface of muscle cell membran
	4. Fasciculation	5. Fibrillation
	• Appears few days or weeks after denervation • Disappear when the motor nerve completely degenerates or successful re-innervation of the muscles occurs.	
	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	As degeneration of the injured axon continues, the axon terminals are now separate from the main axon and hence, from each other. Injury potentials are still generated along the terminals leading to asynchronous contraction of the individual muscle fibers attached to terminals. Invisible to the observer and detected only by electromyogram (EMG).