

20 Motor lesions

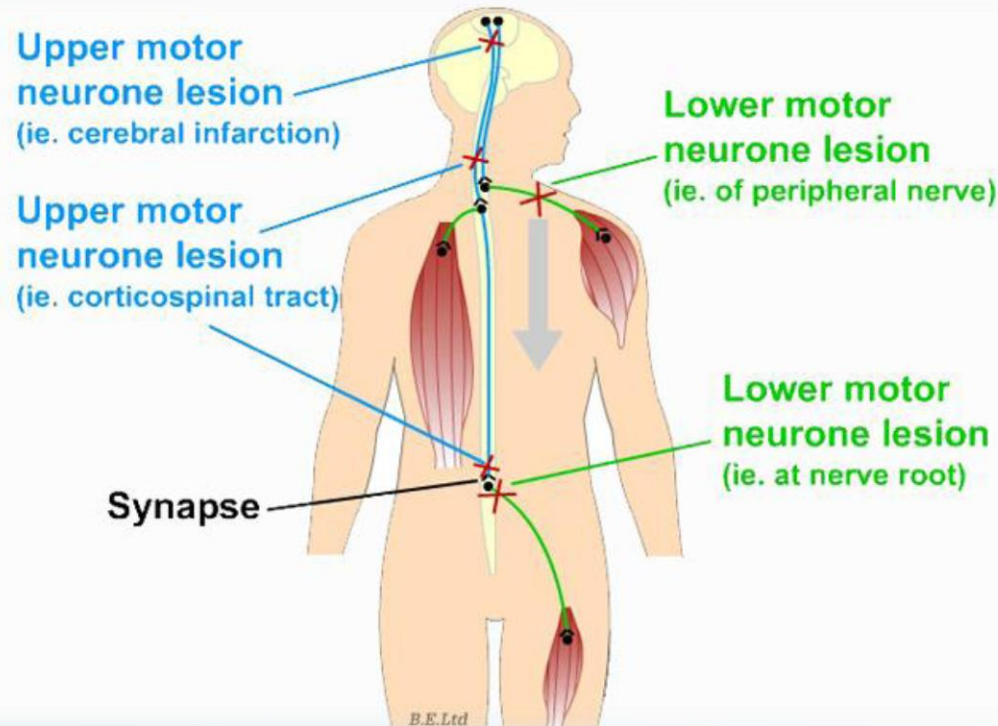
CNS



Sources :
Females slides

Objectives:

- ✓ Appreciate what is meant by upper and lower motor neurons
- ✓ Explain manifestations of upper and lower motor neurons lesions
- ✓ Know effects of lesion in pyramidal tracts at various levels
- ✓ Know effects of lesion in the internal capsule
- ✓ Explain the manifestations of complete spinal cord transection and hemisection.



	UMNL	LMNL
Extent of paralysis	Widespread	Localized
Site of paralysis	Opposite to the lesion	On the same side of the lesion
Tone of the muscles	Hypertonia and Hyperreflexia	Hypotonia
Superficial reflexes	Absent	Absent
Deep reflexes	Exaggerated with appearance of Clonus. Babinski's sign is positive.	Absent
Wasting of the muscles	Very slight due to lack of voluntary movement.	Very marked absence of reflex tone & lack of voluntary movement.
Response of muscles to electrical stimulation	Normal, with normal excitability.	Weak contraction with weak excitability, then no response when it's transferred into fibrous tissue.

Effect of pyramidal tracts lesions at various levels:

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:

site of lesion	In area 4:	Corona radiata	Internal capsule	Brain stem
manifestations	restricted paralysis in the opposite side Monoplegia	Contralateral monoplegia or Hemiplegia	Contralateral hemiplegia	contralateral hemiplegia + ipsilateral paralysis of the cranial nerves ((crossed hemiplegia))

Notes:

- If the lesion was in the midbrain, the **3rd** & **4th** cranial nerves are affected.
- If the lesion was in the pons, the **5th**, **6th**, **7th**, and **8th** cranial nerves are affected.
- If the lesion was in the medulla, the **9th**, **10th**, **11th** & **12th** cranial nerves are affected.



Bilateral lesion in the brain stem is rare and leads to **quadriplegia** and bilateral paralysis of the cranial nerves.

In the spinal cord:

bilateral lesions	Unilateral lesions
<ul style="list-style-type: none"> • In the upper cervical region, are fatal due to interruption of the <u>respiratory pathway</u>. • In the lower cervical region, they lead to quadriplegia. • In the midthoracic region lead to paraplegia 	<ul style="list-style-type: none"> • In the cervical region, they lead to ipsilateral hemiplegia. • In the midthoracic lesion they lead to ipsilateral monoplegia in the corresponding lower limb. <p style="text-align: center;">Note:</p> <p>In both conditions, there is ipsilateral paralysis (LMNL) of the muscles at the level of the lesion due to damage of the spinal motor neurons.</p>

Internal capsule:

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

- It is V-shaped, consisting of **anterior & posterior limb** and a **genu** (knee).
- It is surrounded by the putamen and globus pallidus laterally and the caudate nucleus and thalamus medially.

Anterior limb	The genu	Posterior limb
Contains descending fibers from the cerebral cortex to red nucleus, pons to cerebellum, thalamus, 3, 4, and 6 cranial nerves .	Contains corticobulbar tract .	<ul style="list-style-type: none"> • descending pyramidal & extrapyramidal fibers • somatosensory radiation • optic radiation • auditory radiation



Effects of a unilateral lesion in the 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).

Patients pass into an acute then chronic stage as a following:

<p style="text-align: center;">Acute stage</p> <p style="text-align: center;">((This lasts a few days up to 2-3 weeks. It is a stage of acute UMNL))</p>	<p style="text-align: center;">Chronic stage</p>
<p style="text-align: center;">Showing the manifestations in the <u>opposite side</u>:</p> <ul style="list-style-type: none"> - Flaccid paralysis including the upper and lower limbs, the lower parts of the face and half of the tongue. ❖ Hemianaesthesia (loss of all sensations). ❖ Hypotonia and Areflexia. ❖ Loss of the superficial reflexes. ❖ May be +ve Babinski's sign. <p>N.B: The manifestations of this stage are similar to those of LMNL. However, they can be differentiated from the LMNL by the following:</p> <ol style="list-style-type: none"> a. The extent of paralysis is much more widespread than in LMNL. b. There is associated hemianaesthesia. c. There may be +ve Babinski's sign. d. Absence of muscle atrophy. 	<ul style="list-style-type: none"> ▪ Contralateral hemiplegia of UMNL type. ▪ Permanent loss of fine sensations in the opposite side. ▪ Contralateral homonymous hemianopia. ▪ Diminished hearing power in both areas

Complete spinal cord transaction

Immediate & ever-lasting loss of sensations & voluntary movements occur due to cut of all sensory & motor tracts below transaction.

Transaction in the upper cervical regions (above the 3rd cervical segment) **results in immediate death due to respiratory arrest as in hanging.**

At lower levels, patients pass 3 stages:

- 1- Spinal shock.**
- 2- Recovery of spinal reflex activity.**
- 3- Failure and death.**

Spinal shock:

- Paralysis of all muscles below the lesion.
- Complete loss of all sensation below the level of transection.
- Loss of cord reflexes.
- ABP drops (if the transaction is at the level of the first thoracic).
- Loss of control of micturition and defecation reflexes -Loss of erection.

Cause of Spinal Shock:

It is due to sudden withdrawal of supraspinal facilitation on the spinal alpha motor neurons.

Duration of Spinal Shock:

In humans the duration lasts 2-6 weeks.

Complication:

- 1-**Hypotension** specially in high-level spinal cord lesion.
- 2.**Increased protein catabolism** due to lack of movement causing muscle wasting and bone dissolution.
- 3.**Ischemia of the areas compressed.**
- 4.**Urinary tract infection** due to urine stasis.
- 5.**Fall of body temp** due to reduction of the metabolic rate after loss of muscle tone. (**hypothermia**)

Management of spinal shock:

- 1.Giving **antibiotics** to prevent infection.
- 2.Giving **stimulants to the spinal centers.**
- 3.**Bladder catheterization** to prevent urine stasis.
- 4.Prevention of bed sores by **cleaning the skin.**
- 5.**Adequate nutrition.**

Stage of recovery of reflex activity:

Spinal recovery occurs as follows:

1. The **flexor withdrawal reflex** and **Babinski's sign** usually first responses to appear then followed the extensor reflexes as the knee jerk.
2. The **static stretch reflex** (muscle tone) recovers resulting in spastic paralysis.
3. The **body temperature rises towards normal** (recovery of muscle tone.)
4. The spinal sympathetic VC centers (regain their activity).
5. Micturition and defecation become automatic as in children.
6. Erection can occur by direct stimulation and ejaculation follows.
7. Touch of the patient's skin with a relatively noxious stimulus produces a flexor withdrawal reflex.
8. Impulses may radiate to autonomic centers which lead to provocation of a mass reflex i.e. sweating, pallor, micturition, defecation in addition to wide spreading flexor activity.

N.B: patients can be trained to induce urination or defecation through producing intentional mass reflex by striking the thigh's skin.

Reappearance of spinal reflexes may be due to:

- Release of spinal centers from the normal inhibitory control of the higher centers.
- Denervation hypersensitivity.
- Growth of new collaterals from **preexisting neurons** with formation of additional excitatory endings on spinal neurons.

After spinal shock, the spinal centers below the level of the lesion **recover** gradually **but paralysis and loss of sensations are permanent.**

Stage of failure of reflex activity:

This is a terminal (premortal) stage that **results from bad management during the recovery stage.**

The patients that fail to recover may die because of **renal failure** caused by **urinary tract infections and bed sores infection.**

#The spinal centers below the level of the lesion are depressed once more leading to:

1- **Loss of the muscle tone and tendon jerks, withdrawal reflex and Babinski's sign.** The **muscles become flaccid and body temperature falls.**

2- Loss of the defecation and micturition reflexes.

3- **Hypotension.**

Note:

This stage does not usually occur nowadays, because of perfect nursing and the administration of antibiotics; both lines of treatment guard against bed sores and renal infections.

Hemisection of spinal cord (Brown Sequard syndrome)

This is unilateral transverse lesion in SC.

Manifestations:-

1-Above the level of lesion:

- **Cutaneous hyperesthesia** i.e. increased sensibility to **pain, touch & temp.** occurs ipsilaterally.

2-At the level of lesion and at the same side :

- Loss of all sensations in area innervated by afferent nerves that enter damaged segments.
- Paralysis of muscles supplied by efferent nerves that arise from damaged segments (LMNL).
- Loss of all reflexes that mediated by damaged segments .

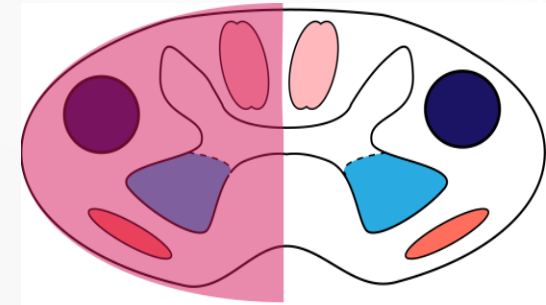
3-Below the level of lesion

#On the same side:

- **Paralysis of voluntary muscles** (UMNL).
- Dorsal column sensations are **lost.**
- Touch is impaired (but not lost) because crude touch persists due to transmission by the opposite intact ventral spinothalamic tract.

#On the opposite side:

- **Loss of pain & temperature** sensations due to cut of lateral spinothalamic tract coming from intact side.



Brown Sequard syndrome

Lateral corticospinal tract

- is affected
- **ipsilateral** UMN dysfunction below lesion

Anterior horn cells

- are affected
- **ipsilateral** LMN dysfunction at the level of the lesion

Dorsal columns

- are affected
- **ipsilateral** proprioception, light touch and vibration loss below the lesion

Spinothalamic tract

- is affected
- **contralateral** sensory loss one or two levels below the lesion

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