Upper and lower motor neuron lesions

* When there is a lesion in the Upper or Lower Motor Neuron → paralysis of the muscles

BUT in each type it has its own characteristics

LMNL: Damage to the "Anterior Horn Cell" or its axon.

	UMNL	LMNL
Extent of Paralysis	**Widespread** (because even when the lesion affect even a small area, the responses are in many fibers)	**Localized** (only few fibers respond)
Site of Paralysis	**Opposite Side** (Because fibers decussated)	**Same Side**
Tone of Muscles	**Hypertonia (spastic paralysis) & Hyper-reflexia** (↑muscle tone) (because, if there's a lesion in the pyramidal tract, the extrapyramidal will be taken with it – inhibitory)	**Hypotonia (Flaccid Paralysis)** (because the "Reflex arc" itself is damaged)
Reflexes	**Absent**	
***Deep**	***Exaggerated with <u>Clonus</u>** (rhythmic contraction of muscles – sudden maintained reflex)	**Absent**
***Superficial**	***+ve Babinski's sign** (when applied, if normal → plantar flexion of foot if UMNL → dorsiflexion of foot & big toe)	**Absent** (because the "Reflex arc" itself is damaged)
Wasting (happens due to -disuse of muscles -denervation of muscles)	**Very Slight** (muscles with paralysis will waste overtime)	**Marked** (wherever affected, there will be wasting because they are denervated)
Response of Muscles (to electric stimulation)	**Normal Contraction** **Normal Excitability** (because low neurons are not affected)	**Weak Contraction** **Decreased Excitability** (because the lower neurons are affected)

<u>If Tone Increase:</u> There are two types of ↑ tone according to tract affected:

1- corticospinal (pyramidal) → spasticity (clasp knife).

2- extrapyramidal → rigidity

(sometimes there's tremor + rigidity, what we call **cog-wheel rigidity**. d/t ↑ gamma & loss of inhibitor signals)

Effect of lesions of the Pyramidal Tracts 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:

1- In Area 4:

this leads to **restricted paralysis** in the opposite side.
(To one limb only because it is very rare to affect the whole area 4)

2- In the Corona Radiata:

this leads to **contralateral monoplegia** (one limb) or **hemiplegia** (upper and lower limb), depending on the extent of the lesion.
(the fibers are radiating and separated at the corona radiate)

3- In the Internal Capsule:

this often leads to **contralateral hemiplegia** because almost all fibers are injured.
(the fibers are gathered at the internal capsule)

4- In the Brain Stem:

this leads to **contralateral hemiplegia** + **ipsilateral paralysis of the cranial nerves of the LMNL type**
(due to damage of their nuclei in the brain stem).
Cranial nerves nuclei are NEVER included in UMNL

This condition is called **crossed hemiplegia**, and the nerves affected differ as follows:
* If the lesion was in the **Midbrain → the 3rd & 4th CNs** are affected.

* If the lesion was in the **Pons → the 5th, 6th, 7th, and 8th CNs** are affected.

* If the lesion was in the **Medulla → the 9th, 10th, 11th & 12th CNs** are affected.

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

5- In the Spinal Cord:

- **Bilateral lesions**

* In the **upper cervical region** → are **fatal** due to interruption of the respiratory pathway.

* In the **lower cervical region** → lead to **quadriplegia**. (all limbs)

* In the **mid-thoracic region** → lead to **paraplegia.** (both lower limbs)

} **Do not affect respiratory pathway**

❖ **Unilateral lesions**

* In the **cervical region** → lead to **ipsilateral hemiplegia**.
* In the **midthoraic region** → they lead to **ipsilateral monoplegia** in the corresponding lower limb.

* **In both conditions** → there is also **ipsilateral paralysis** of the LMNL type of the muscles at **the level of the lesion** due to damage of the spinal motor neurons.

The Internal Capsule

Effects of a unilateral lesion in the posterior limb of internal capsule

Such lesion commonly called **Cerebral Stroke.** It is usually caused by thrombosis or hemorrhage of
lenticulo-striate artery
(a branch of the middle cerebral artery, which supply the **posterior limb** or the internal capsule)
Patients pass into an acute then chronic stage.

Acute stage:

This lasts a few days up to 2-3 weeks. It is a stage of acute UMNL, showing the following manifestations
in the opposite side:

- ❖ Flaccid paralysis including the upper and lower limbs, the lower parts of the face and half of the tongue.
- ❖ Hemi-anesthesia (loss of all sensations in half side of body).
- ❖ Hypotonia and areflexia (absence of a reflex).
- ❖ Loss of the superficial reflexes.
- ❖ May be +ve Babinski's sign.

N.B: The manifestations of this stage are similar to those of LMNL.

However, they can be differentiated from the LMNL by the following:
a. The extent of paralysis is much more widespread than in LMNL.
b. There is associated **hemi-anesthesia**.
c. There may be +**ve Babinski's sign**
d. Absence of **muscle atrophy.**

Chronic (permanent or spastic) stage:

The main manifestations of this stage include the following:

- **Contralateral hemiplegia** of the UMNL type,
  which is characterized by
  Hypertonia - Muscle Spasticity of Clasp Knife Type - Exaggerated Tendon Jerks & Clonus
  Loss of Superficial Reflexes - Appare Nt +Ve Babinski's Sign.
- Permanent loss of **fine sensations** in the opposite side, but the **crude sensations** recovers gradually.
- **Contralateral homonymous hemianopia**.
  (A type of partial blindness resulting in a loss of vision in the same visual field of both eyes).
- Diminished hearing power in both ears (by about 50 %), because of damage of auditory radiation.

***Complete Spinal Cord transaction (UMNL) ***

This results usually from accidents.
Immediate and ever-lasting loss of sensations and voluntary movements.

- 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:

I- Stage of spinal shock (weeks to months)

All cord functions are depressed. The manifestation shown are

- Paralysis of all muscles below the lesion (**quadriplegia** or **paraplegia**) due to cut of UMN
- Complete loss of all sensation **below** the level of transaction.
- **Loss of cord reflexes**
  as the stretch reflex,(because the centers of reflexes are in the spinal cord)
  hence the paralysed muscles are **flaccid** and the deep reflexes are absent.
  The other reflexes are also absent such as the withdrawal flexor reflex.
- **ABP drops** due to sympathetic activity block.
  However, the pressure returns to normal within a few days.
- **Loss of control** of micturition and defecation reflexes leading to **retention** with overflow with dribbling of urine by a full bladder. This returns back after the first few weeks.
  (resembles a cup, when it is filled it overflows spontaneously)

- Loss of erection. (because it is a type of reflexes)

Cause of spinal shock

It is due to **sudden withdrawal of <u>supraspinal facilitation</u> on the spinal alpha motor neurons**; mainly the continual tonic discharge transmitted along excitatory reticulospinal, - vestibulospinal - corticospinal tracts.

Duration of the spinal shock

The duration of spinal shock differs in different animals according to the **degree of development** of the cerebral cortex. It is only a few minutes in rats. **In humans** the duration lasts 2-6 weeks.

Complications of spinal shock

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 against bed (upper back, gluteal region and heels) (decubitus ulcers or bed sores – تقرحات الفراش) which heal poorly due to protein depletion.
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. (↑movement of muscles → ↑metabolism)

Management of spinal shock

This arm at rapid recovery of spinal reflex activity which can be achieved by the following:
1- Giving antibiotics to prevent infection.
2- Giving **stimulants** to the spinal centers (drugs).
3- Bladder catheterization to prevent urine stasis and rectal enema to evacuate the rectum.
4- Prevention of bed sores by **cleaning the skin with antiseptics** and frequent changing the patient's position in bed.
5- Adequate nutrition.

II- Stage (period) of recovery of reflex activity:

Spinal recovery (after 2-3 weeks) occurs as follows:

- The **flexor withdrawal reflex** and **Babinski's sign** are usually the first responses to appear followed by the **extensor reflexes as the knee jerk**. (flexor then extensor !!)
- The **static stretch reflex** (muscle tone) recovers resulting in spastic paralysis.
  (i.e. recovery of spinal cord centers)
  It appears **first in flexor muscles** causing paraplegia in flexion.
  Then a few months later, the **extensor muscle tone** predominates resulting in paraplegia in extension.
- The body temperature **rises towards normal level** as a result of **recovery of muscle tone**.
- **The spinal sympathetic VC** regain their activity.
  Hence, the ABP rises and the limbs become **warm** and **with a healthy skin**, with good color helping in healing up of the ulcers.
- **Micturition and defecation** become **automatic as in children with residual urine** due to **weakness of the reflex**. (do not micturate consciously as adults)
- **Erection can occur** by direct stimulation and **ejaculation follows**.
- Touch of the patient with a relatively noxious stimulus (pain) applied to the skin produces a mass reflex.
  (especially when pinching the skin→ micturition & defecation).

III- Stage of failure of reflex activity:

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

**Urinary tract infections and bed sores infection result in failure of reflex activity and the patient dies from renal failure.

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

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

2- Loss of the defecation and micturition reflexes resulting in constipation and urine retention with overflow.

3- Hypotension due to depression of the spinal VC centers.

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

Mass Reflexes:
A package of reflexes occurring when you stimulate any one of them. This last for short time then disappear..

Where do we call it an UMNL or LMNL of spinal cord???
Below lesion level → its UMNL.
At level of lesion → its LMNL
because it also damages the anterior horn cells of the lesion-ed site while the ones of the lower levels are not damaged. Instead, the pyramidal tract is the one damaged (UMNL).

Complete: all the segment damaged → 
Hemi-section: only one half of the segment damaged → 

Hemi-section of the spinal Cord

Brown Sequard syndrome

This is **unilateral transverse lesion in SC** that interrupts the continuity of both ascending & descending tracts at only one half ((e.g. due to tumor or trauma)).

Manifestations

Above the level of lesion

Cutaneous hyper-aesthesia ((i.e. increased sensibility to **pain, touch & temp**)).
It occurs in ipsilateral dermatome due to **irritation of the dorsal nerve roots by the neighboring lesion**.

At the level of lesion and at the same side

1- **Loss of all sensations** in area innervated by afferent nerves that enter damaged segments.
2- Paralysis of muscles supplied by efferent nerves that arise from damaged **segments (LMNL).**
3- **Loss of all reflexes** (superficial& deep) mediated by damaged segments.

Below the level of lesion

On the same side

1- Paralysis of voluntary muscles (UMNL).

2- Dorsal column sensations are lost.

3- Touch is impaired (but NOT lost) because the dorsal column is transected (only half of it is affected).

Yet, **crude touch sensation still persists** because of its transmission by the opposite intact ventral spinothalamic tract (because it is impaired contralaterally).

On the opposite side

Loss of pain & temperature sensations due to cut of lateral spinothalamic tract coming from intact side (loss of crude touch).
((no motor loss – no fine touch loss))

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