

Motor lesions

Objectives

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

Upper and lower motor neuron lesion

	<i>UMNL</i>	<i>LMNL</i>
<i>Extent of paralysis</i>	<i>Widespread</i>	<i>Localized</i>
<i>Site of paralysis</i>	<i>Opposite side</i>	<i>Same side</i>
<i>Tone of muscles</i>	<i>Hypertonia (spastic paralysis) & hyperreflexia</i>	<i>Hypotonia (flaccid paralysis)</i>
<i>Reflexes</i>	<i>Absent</i>	<i>Absent</i>
<i>* Superficial</i>	<i>Exaggerated with clonus.</i>	<i>Absent</i>
<i>* Deep</i>	<i>+ve Babinski's sign</i>	
<i>Wasting</i>	<i>Very slight</i>	<i>Marked</i>
<i>Response of muscles</i>	<i>Normal contraction</i>	<i>Weak contraction</i>
	<i>Normal excitability</i>	<i>Decreased excitability</i>

Effect of lesions of the pyramidal tracts at various levels

Lesions of pyramidal tract cause paralysis of the UMN 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.

2- In the corona radiata: this leads to contralateral monoplegia or hemiplegia, depending on the extent of the lesion.

3- In the internal capsule: this often leads to contralateral hemiplegia because almost all fibers are injured.

4- In the brain stem: this leads to contralateral hemiplegia + ipsilateral paralysis of the cranial nerves of the LMN type (due to damage of their nuclei in the brain stem). This condition is called crossed hemiplegia, and the nerves affected differ as follows:

- * If the lesion was in the midbrain, the 3rd & 4th 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.

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.
- * In the midthoracic region, lead to paraplegia.

● *Unilateral lesions:*

- * In the cervical region, lead to ipsilateral hemiplegia.
- * In the midthoracic lesion they lead to ipsilateral monoplegia in the corresponding lower limb.
- * In both conditions, there is also ipsilateral paralysis of the LMN 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 is usually caused by thrombosis or hemorrhage of lenticulo-striate artery

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.
- Hemianaesthesia (loss of all sensations).
- Hypotonia and areflexia.
- 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 hemianaesthesia.
 - 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 and clonus, loss of superficial reflexes, apparent +ve Babinski's sign.
- Permanent loss of fine sensations in the opposite side, but the crude sensations recover gradually.
- Contralateral homonymous hemianopia .
- Diminished hearing power in both areas (by about 50 %), because of damage of auditory radiation.

Complete spinal cord transection

This results usually from accidents.

Immediate and ever-lasting loss of sensations and voluntary movements.

- Transection 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 in man)

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 transection.
- Loss of cord reflexes as the stretch reflex, 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.
- Loss of erection.
- Cause of spinal shock
- It is due to sudden withdrawal of supraspinal facilitation on the spinal alpha motor neurons, namely; the continual tonic discharge transmitted along the excitatory reticulospinal, vestibulospinal and 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) 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.

Management of spinal shock

This aim 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.
- 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 of recovery of reflex activity:

Spinal recovery 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.
- * The static stretch reflex (muscle tone) recovers resulting in spastic paralysis. 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 healing up of the ulcers.
- * Micturition and defecation become automatic as in children with residual urine due to weakness of the reflex.
- * Erection can occur by direct stimulation and ejaculation follows.

* Touch of the patient with a relatively noxious stimulus applied to the skin produces a mass reflex.

III- Stage of failure of reflex activity:

This is a terminal (premortal) 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, then mass reflex, withdrawal reflex and 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.

Hemisection 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 hyperaesthesia 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. Yet, crude touch sensation still persists because of its 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.