UPPER & LOWER MOTOR NEURON

lesion

By

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Ph.DX

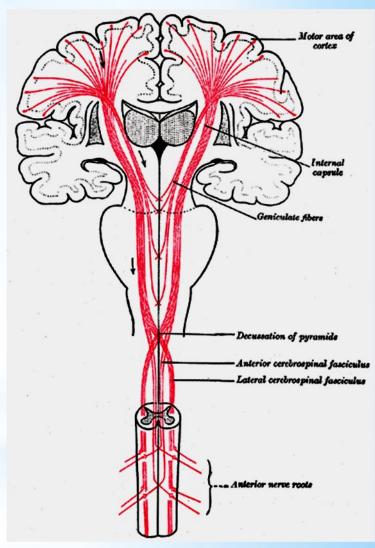
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Objectives

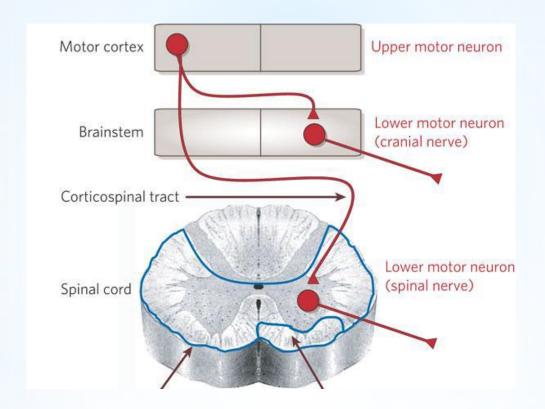
- Appreciate what is meant by upper and lower motor neurons
- •Explain manifestations of upper and lower motor neurons lesions
- •Know effects of lesion in pyramidal tracts at various levels
- •Know effects of lesion in the internal capsule
- •Explain the manifestations of complete spinal cord transection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

<u>Cerebral Dominance: The Dominant</u>
Hemisphere

- *hemisphere giving out the pyramidal tract usually has more <u>influence than the other hemisphere and is called the "Dominant Hemisphere ".</u>
- * About 90 % of people are right-handed, and consequently their Dominant Hemisphere is on their left cerebral hemisphere.
- *The remaining 10 % of people have their right cerebral hemisphere as the Dominant Hemisphere, and therefore they are left-handed & their speech center in the right hemisphere.



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Upper and Lower motor neurones

* Causes of UMNL & LMNL

Upper Motor Neuron Lesion, UMNL

Can be due to

- (1) Cerebral stroke by haemorrhage, thrombosis or embolism
- (2) Spinal cord transection or hemisection
- (Brown- Sequard syndrome)

Lower Motor Neuron
Lesion,LMNL

Can result from

- (1) Anterior horn cell lesions (e.g., poliomyelitis, motor neuron disease)
- (2) Spinal root lesions or peripheral nerve lesion
- (e.g. nerve injury by trauma or compressive lesion)

	<u>UMNL</u>	<u>LMNL</u>
1-extent of paralysis	widwspread	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 <u>minor</u> wasting may occur due to(disuse atrophy)	Marked muscle wasting (atrophy)
8-Clonus	Clonus present (rhythmic oscillation on tendon stretch)	No clonus
9-Fasciculations (seen) Fibrillation potentials by EMG .	No fasciculations No fibrillation potential	Fasciculations may be seen . & Fibrillation by EMG

* 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:

* 1- In area 4:

*This leads to restricted paralysis_e.g. <u>contralateral monoplegia</u> (paralysis of one limb because area 4 is widespread so it is rarely damaged completely.

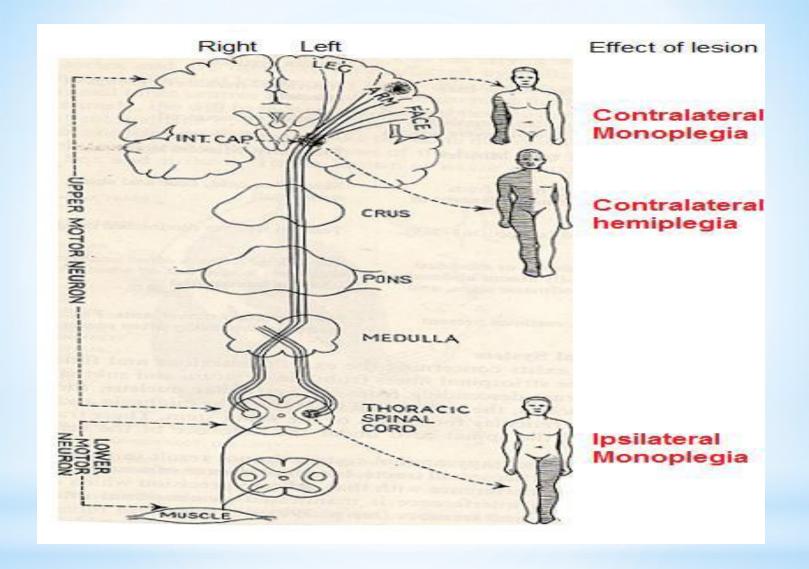
*2- In the corona radiata:

*This leads to <u>contralateral monoplegia or hemiplegia</u>, 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 (crossed hemiplegia UMNL)

- + ipsilateral paralysis of the cranial nerves of the LMNL type (due to damage of their nuclei in the brain stem), the nerves affected differ as follows:
- 1-If the lesion was in the midbrain, the 3rd & 4th are affected.
- 2- If the lesion was in the <u>pons</u>, the 5th, 6th, 7th, and 8th cranial nerves are affected.
- 3- If the lesion was in the <u>medulla</u>, 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:

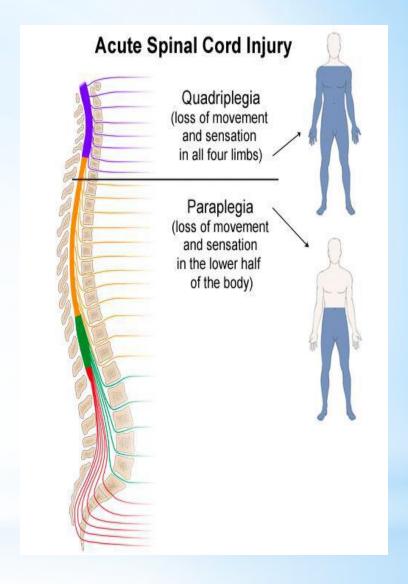
*In the upper cervical region, are **fatal** due to interruption of the respiratory pathway.

A-Bilatral lesions

- In the lower cervical region, they lead to **quadriplegia**.
- In the midthoracic region lead to paraplegia.

B-Unilatral lesions

- In the <u>cervical region</u>, they lead to <u>ipsilateral hemiplegia</u>.
- In the <u>midthoraic lesion</u> they lead to <u>ipsilateral monoplegia</u> in the corresponding <u>lower limb</u>.
- 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



*The 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

The anterior limb:

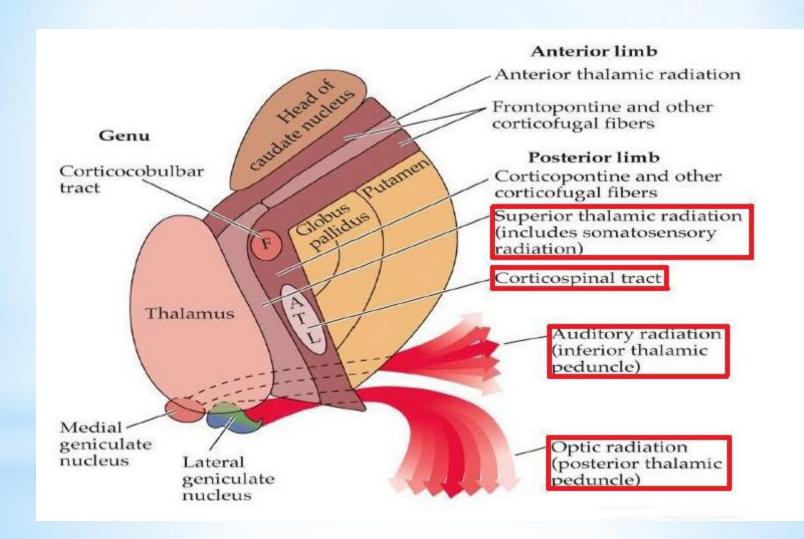
Contains descending fibers from the cerebral cortex <u>to</u> red nucleus, pons to cerebellum, thalamus, 3, 4, and 6 cranial nerves.

The genu

Contains corticobulbar tract

The posterior limb contains:

- -The descending pyramidal & extrapyramidal fibers in the anterior 2/3.
- -The somatosensory radiation that ascends behind the pyramidal fibers from thalamic nuclei to cortical sensory areas.
- -The optic radiation that ascends behind the somatosensory radiation from the lateral geniculate body to visual areas in the occipital lobe.
- -The auditory radiation that ascend most posteriorly from the medial geniculate body to auditory areas in the temporal lobe.



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

Such lesion commonly called <u>cerebral stroke</u> 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.

1--- Acute stage

- lasts a few days up to 2-3 weeks. Acute <u>UMNL</u> manifestations <u>in the opposite side</u>:
- •Flaccid paralysis including the upper and lower limbs, the lower parts of the face and half of the tongue.
- •Hemianaethesia (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 <u>more widespread</u> than in LMNL.
- b. There is associated <u>hemianaethesia</u>.
- c. There may be +ve Babinski's sign
- d. Absence of muscle atrophy.

* 2-Chronic(permenant or spastic)stage

The main manifestations of this stage include the following:

- •Contralateral hemiplgia of the <u>UMNL type</u>.
- •N.B: Partial recovery occurs after a variable period by the effect of the ipsilateral corticospinal tract, extrapyramidal tracts, so, the patient can stand and even walk, but the fine skilled movements are permanently lost.

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- -Permanent loss of fine sensations in the opposite side, but the crude sensations recover gradually.
- Contralateral homonymous hemianopia (loss of vision in the two corresponding haves of the visual fields opposite to side of lesion due to injury to optic radiation (injury of left optic radiation causes blindness of the right halves of visual field) interruption of signals from the temporal part of left retina and nasal part of right retina=loss of impulses from both left haves of both retinae = loss of both right sides of visual field)
- •Diminished hearing power in both ears (by about 50 %), because of damage of auditory radiation.

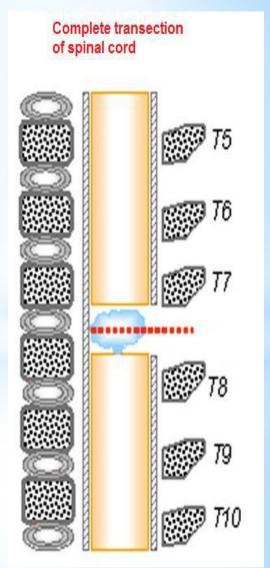
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4-Complete transection of spinal cord:e.g. following tumor or trauma

•1- if the transection is in the upper cervical region

immediate death follows, due to paralysis of all respiratory muscles;

- •2- in the lower cervical <u>region below the 5th</u> <u>cervical segment</u> diaphragmatic respiration is still possible, but the patient suffers complete paralysis of all four limbs <u>(quadriplegia)</u>.
- 3-transection lower down in the thoracic region allows normal respiration but the patient ends upwith paralysis of both lower limbs (paraplegia)--



* Stages :-

- A/ Spinal shock (2-6 weeks)
- B/ Recovery of reflex activity
- C/ Paraplegia in extension

a/ spinal shock

- This stage varies in duration but usually lasts a maximum of 2-6 weeks, after which some reflex activity recovers.

<u>Cause:-•</u>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, yestibulospinal and corticospinal tracts.

the manifestation 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.
- (3) <u>loss of tendon reflexes and superficial reflexes (abdominal</u>, plantar & withdrawal reflexes) below the level of the lesion.

- (5) the loss of muscle tone (flaccidity) and absence of any muscle activity (muscle pump) lead to decreased venous return \Box causing the lower limbs to become cold and blue in cold weather
- (6) The wall of the urinary bladder becomes paralysed and urine is retained until the pressure in the bladder overcomes the resistance offered by the tone of the sphincters and dribbling occurs. This is known as (<u>retention</u> <u>with overflow</u>).
- (7)Loss of vasomotor tone occurs, due to interruption of fibres that connect the vasomotor centres in the medulla oblongata with the lateral horn cells of the spinal cord, which project sympathetic vasoconstrictor impulses to blood vessels. vasodilatation causes a fall in blood pressure;

 Managementof this stage//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

* B/ Stage of return of reflex activity

- As the spinal shock ends, spinal reflex activity appears again this <u>partial</u> recovery may be due to:-
- increase in degree of excitability of the spinal cord neurons below the level of the section ,

due to:_

- 1-disinhibition of motoneurons as a result of absence of inhibitory impulses from higher motor centres
- -sprouting of fibres from remaining neurons
- -denervation supersensitivity to excitatory neurotransmitters.
- Features of the stage of recovery of reflex activity
- (1) Gradual rise of arterial blood pressure due to return of spinal vasomotor activity in the lateral horn cells. But, since vasomotor control from themedulla is absent, the blood pressure is not stable
- vasoconstrictor tone in arterioles and venules improve the circulation through the limbs.

***** 2) Return of spinal reflexes:

- 1- Flexor tendon reflexes & withdrawal reflexes return earlier than extensor ones.
- Babiniski sign (extensor plantar reflex) is one of the earliest signs of this stage
- Flexor spastic tone causes the lower limbs to take a position of slight flexion, a state referred to as paraplegia in flexion.
- The return of the stretch reflex (muscle tone)
- (2) Recovery of visceral reflexes: return of micturition, defecation & erection reflexes.
- However voluntary control over micturition and defecation, and the sensation of bladder and rectal fullness are permanently lost

(automatic micturition)

- (5) Mass reflex appears in this stage
- A minor painful stimulus to the skin of the lower limbs will not only cause withdrawal of that limb but will evoke many other reflexes through spread of excitation (by irradiation) to many autonomic centres. So the bladder and rectum will also empty, the skin will sweat, the blood pressure will rise.
- -Voluntary movements and sensations are permanently lost;
- -however, patients who are rehabilitated and properly managed may enter into a more advanced stage of recovery

(Since effective regeneration never occurs in the human central nervous system, patients with complete transection never recover fully.)

- (1) During this stage the tone in extensor muscles returns gradually to exceed that in the flexors. The lower limbs become spastically extended.
- -Extensor reflexes become exaggerated, as shown by tendon jerks and by the appearance of clonus.
- -The positive supportive reaction becomes well developed and the patient can stand on his feet with appropriate support.
- (2) The flexor withdrawal reflex which appeared in the earlier stage is associated during this stage with the crossed extensor reflex.

Stage of failure of reflex activity:

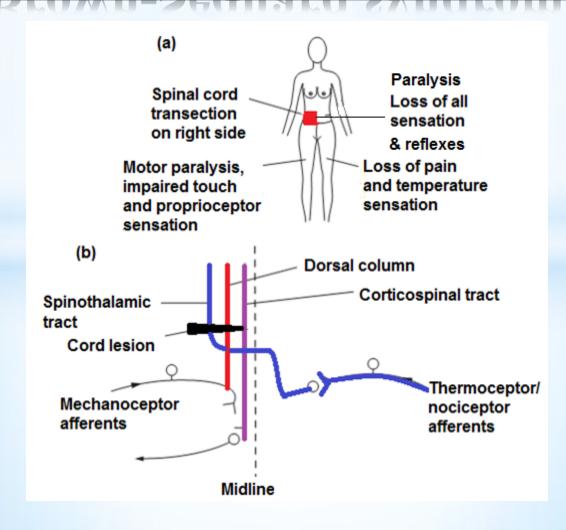
This results from bad management during the recovery stage.

- -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.

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Hemisection of the Spinal Cord (Brown-Sequard syndrome)



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- Occurs as a result of unilateral transverse lesion or hemisection of the spinal cord (e.g. due to stab injury, bullet, car accident, or tumor). It interrupts the continuity of both ascending & descending tracts at only one half
- -The manifestations of the Brown-Sequard syndrome depend on the level of the lesion. (Let us take an example of such injury involving the thoracic spinal cord)

On the same side at the level of lesion

- 1.Paralysis of the lower motor neuron type, involving only the muscle supplied by the efferent nerves from damaged segments.
- 2-Loss of all reflexes (both superficial and deep) mediated by damaged segments.
- 3. Loss of all sensations in the areas supplied by the afferent fibres that enter the spinal cord in the damaged segments

 On the same side above the level of lesion -band of Cutaneous hyperesthesia (increased sensibility to pain, touch & temp. occurs in ipsilateral dermatome due to irritation of the dorsal nerve roots by the neighboring lesion

- * B/ Ipsilaterally below the level of the lesion:
 - 1. <u>UMNL/spastic lower limb (spasticity)</u> &CLONUS
 - 2. Dorsal column sensations are lost/ Fine touch, two-point discrimination, position and vibration sense are lost, why?

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.

C/ Contralaterally below the level of the lesion: Pain and temperature sensations are lost, Why?

