Physiology Team

LECTURE 29

Upper & Lower Motor Neuron Lesions

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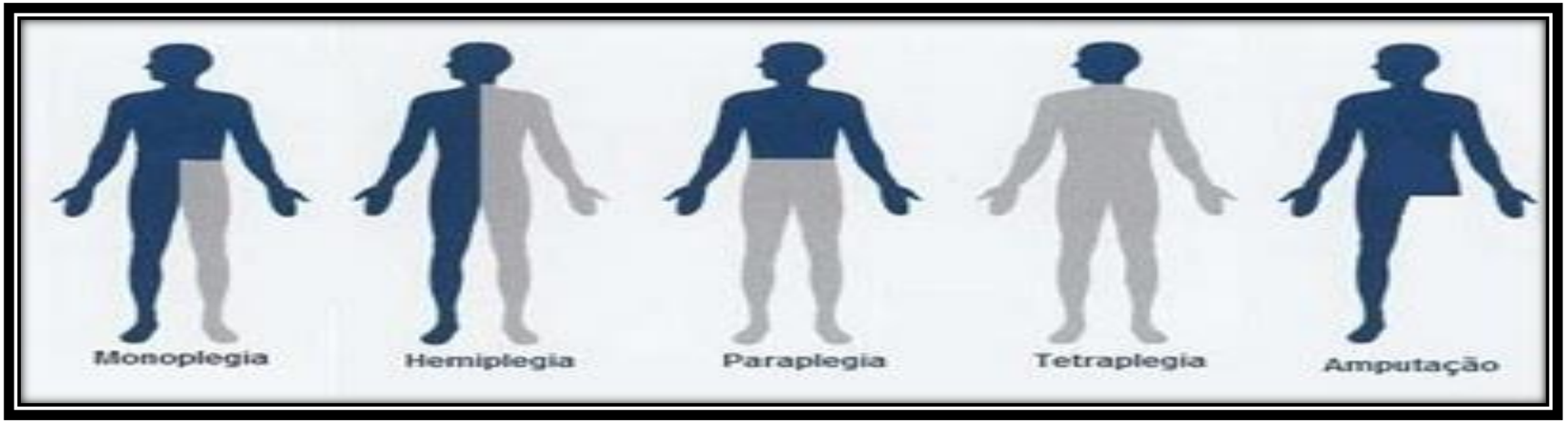
OBJECTIVES

At the end of this lecture, student should be able to describe:

- 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.



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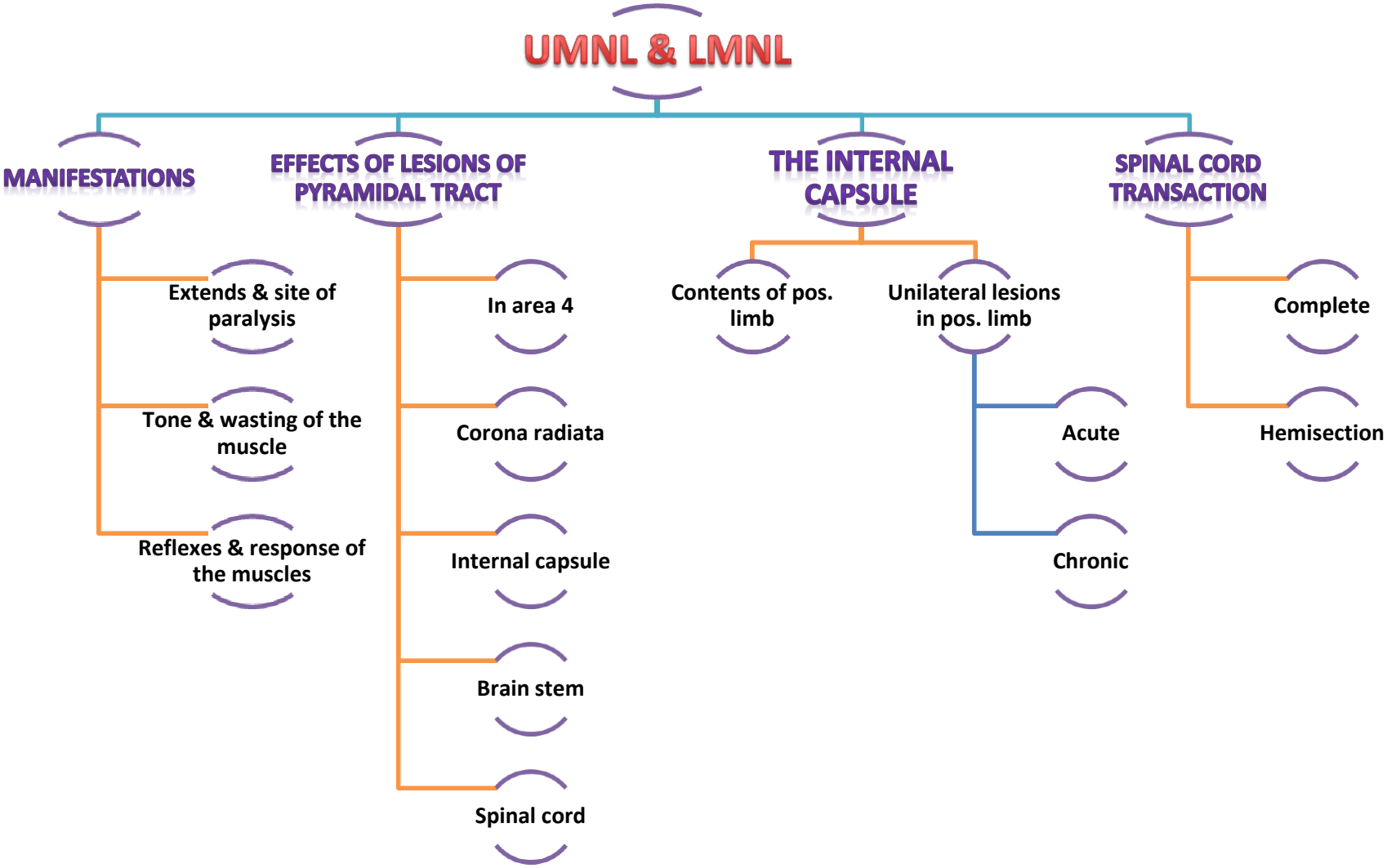
MIND MAP



Slides | Important | Doctor's Notes | Explanation | Boy's Slides

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UMN & LMN

The performance of a voluntary act needs the integrity of 2 sets of motor neurons; upper and lower motor neurons.

Upper Motor Neuron

- These consist of the motor cells of the cerebral cortex and their axons, which relay at the motor nuclei of the brain stem and spinal cord **& anterior horn cells of spinal cord,** chiefly of the opposite side.
- **These descending tracts include:**

➢ **The corticonuclear tract:**
- Arises from area 8 of the frontal cortex.
- Terminates in the motor nuclei of the III, IV, VI CN.

➢ **The cortico-bulbar tract:**
- Arises from the motor area.
- Terminates in the motor nuclei of V, VII, IX, X, XI, and XII CN.

➢ **The corticospinal tract (pyramidal tract):**
- Arises from the motor area.
- Terminates at the anterior horn cells of the spinal cord.

Lower Motor Neuron

- Neurons in the motor nuclei of all the above mentioned cranial nerves and their axons to skeletal muscles of the eyes and the head.
- Anterior horn cells of the spinal cord and their axons to skeletal muscles of the body.

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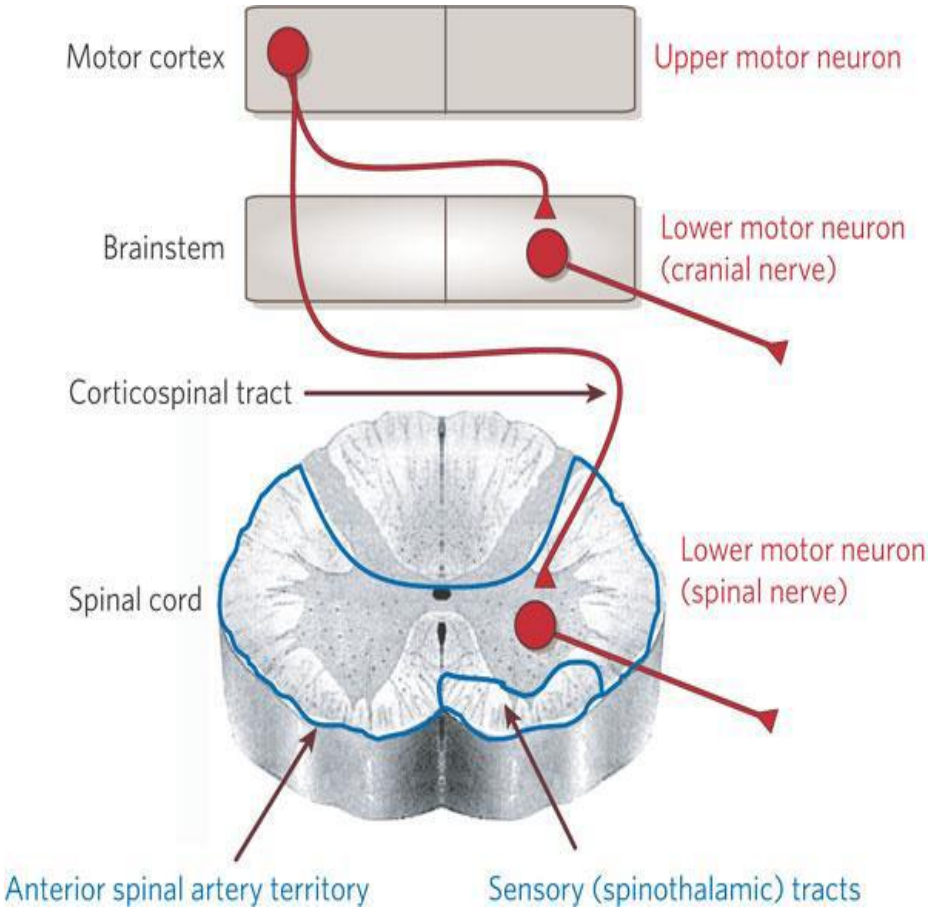
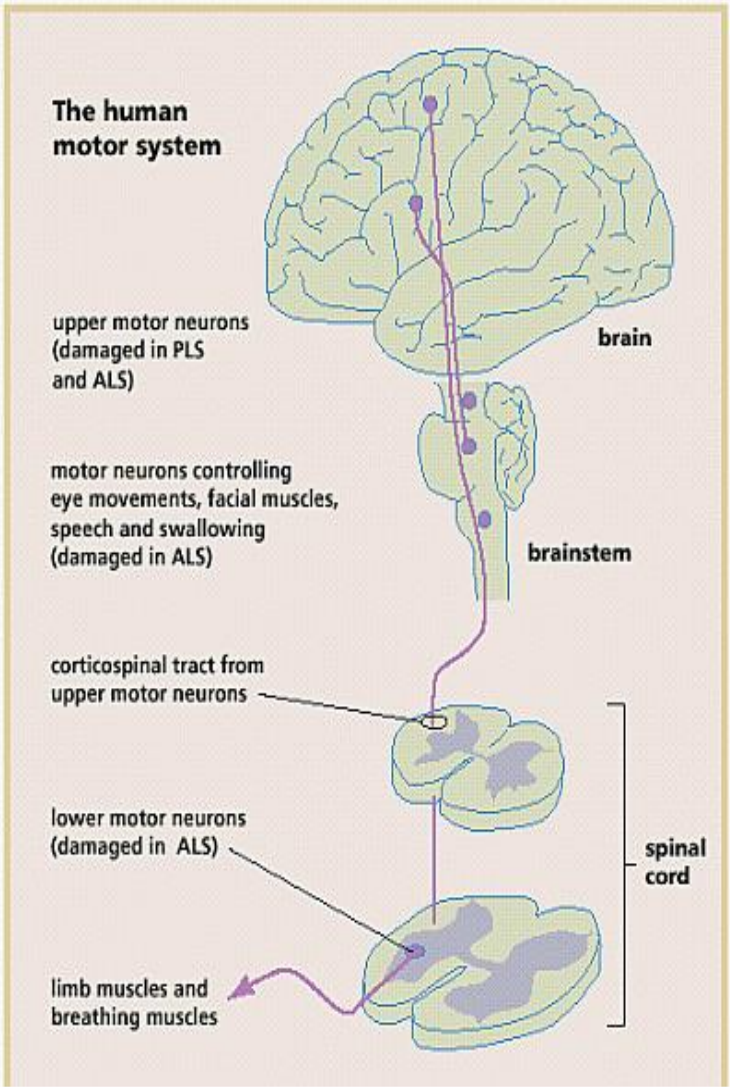
Upper Motor Neuron





Lower Motor Neuron

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Upper Motor Neuron Lesions

	Manifestations of Upper Motor Neuron Lesions
Extent of paralysis	Wide spread because the pyramidal fibers form a **compact bundle** which occupies a small area **& supply large number of muscles .**
Site of paralysis	Opposite to the lesion e.g. hemorrhage into the **right** internal capsule causes hemiplegia or paralysis of the muscles of the **left half** of the face and of the **left** upper and lower limbs.
Tone of muscles	**Hypertonia*** and **hyperreflexia** (1) due to block of the **extrapyramidal inhibitory** discharge on the gamma efferent and hence the **excitatory reticular formation** becomes unopposed. So, spasticity is a release phenomenon from the normal inhibitory discharge.
Reflexes	**Superficial reflexes:** are **absent** on the affected side as they receive their facilitatory influence from the pyramidal tracts.
	Deep reflexes: are **exaggerated** with appearance of **clonus** (2) **. Babinski's sign**(3) with its center in S1& 2 is +
Wasting of the muscles	**Very slight** due to exaggerated tone (spasticity). The slight wasting results from the lack of voluntary movements **but reflex arc is intact.**
Response of muscles	To electrical stimulation is **normal**, **with normal excitability**.

- **No fasciculations (involuntary muscle contraction and relaxation visible under the skin)**
- **No fibrillation potential in EMG (rapid, irregular, and unsynchronized contraction of muscle fiber**

***Hypertonia = spasticity = increase muscle Tone (clasp – knife spasticity)**

(1) exaggerated tendon jerk

(2) clonus = sudden & sustained dorsiflexion of the foot >> repetitive planter flexion (soul of foot).

(3) Babinski's sign = dorsiflexion of the toe .. The normal planter response when we do scratching of the lateral side if the soul of foot by rough object (key for example) >> that causes planter flexion.

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Clasp-Knife spasticity

Clonus

Babinski Sign

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Lower Motor Neuron Lesions

	Manifestations of Lower Motor Neuron Lesions
Extent of paralysis	**localized** depending on the site of the lesion. **(limited # of muscles e.g. if the lesion occur at the level of T1 .. The muscles supplied by that segment will be paralyzed).**
Site of paralysis	On the **same side** of the lesion e.g. damage of the AHCs on the right side of spinal cord causes paralysis of the muscles supplied by these AHCs on the right side.
Tone of muscles	**Hypotonia** because the stretch reflex arc is cut.
Reflexes	Both superficial and deep reflexes are **absent** in the affected segments **e.g. if the lesion occur at C5-C6 absence of the biceps reflex**
Wasting of the muscles	Very **marked** due to absence of reflex tone as well as lack of voluntary movements.
Response of muscles	to electrical stimulation is **abnormal.** The response is weak contraction with decreased excitability, then **no response** when it is transformed into **fibrous tissues.**

- ❑ **Fasciculation's may occur**
- ❑ **Fibrillation potentials present**

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Boys' slides

Examples of Conditions in Which there is UMNL or LMNL

Upper Motor Neuron Lesion (UMNL)

Can result from:

(1) **Haemorrhage, thrombosis or embolism in the internal capsule.**

(2) **Spinal cord transection or hemisection.**

Lower Motor Neuron Lesion(LMNL)

Can result from:

(1) **Spinal root lesions or peripheral nerve lesion.**

(e.g. nerve injury by trauma or compressive lesion

(2) **Anterior horn cell** lesions.

(e.g. poliomyelitis, motor neuron disease

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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***

A- In Area 4

This leads to restricted paralysis in the opposite side e.g. monoplegia (paralysis of one limb because area 4 is widespread so it is rarely damaged completely.

B-In the corona radiata

This leads to **contralateral monoplegia or hemiplegia**, depending on the extent of the lesion.

C- In the internal capsule

This often leads to **contralateral hemiplegia** because almost all fibers are injured.

D- 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). This condition is called crossed hemiplegia, and the nerves affected differ as follows:

If the lesion was in the:

Midbrain >> 3rd & 4th are affected.

Pons >> 5th, 6th, 7th & 8th are affected.

Medulla >> 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.

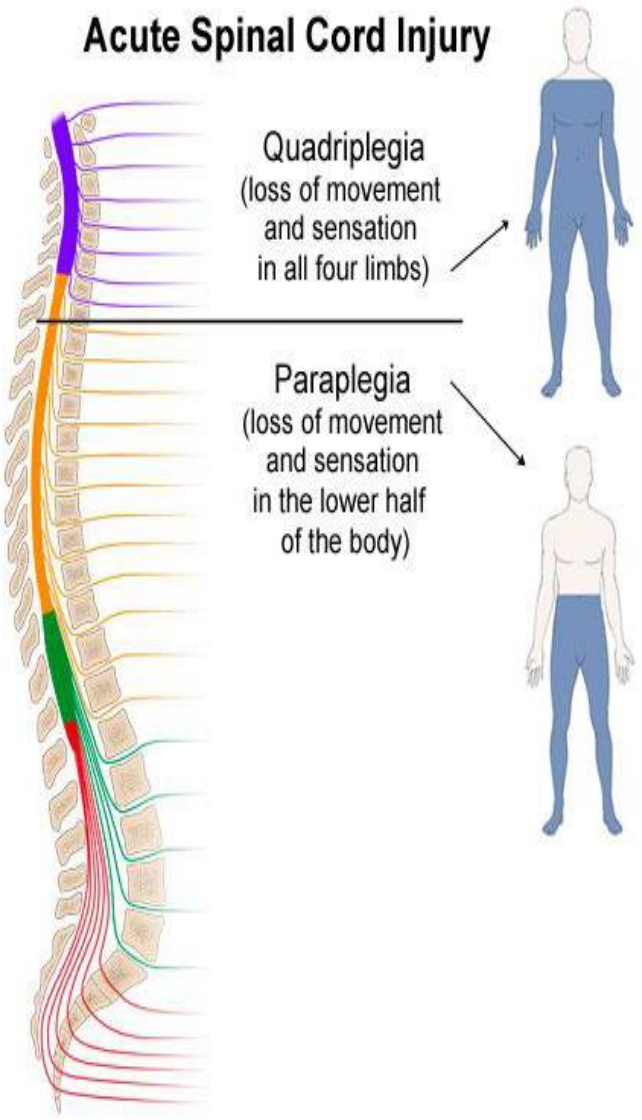
E- In the Spinal Cord

A) Bilateral lesions:

- In the upper cervical region, are **fatal** due to interruption of the respiratory pathway.
- In the lower cervical region, they lead to **quadriplegia**.
- In the midthoracic region lead to **paraplegia**.

B) Unilateral lesions:

- In the cervical region, they lead to ipsilateral hemiplegia.
- In the midthoracic lesion they lead to ipsilateral monoplegia in the corresponding lower limb.
- 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.



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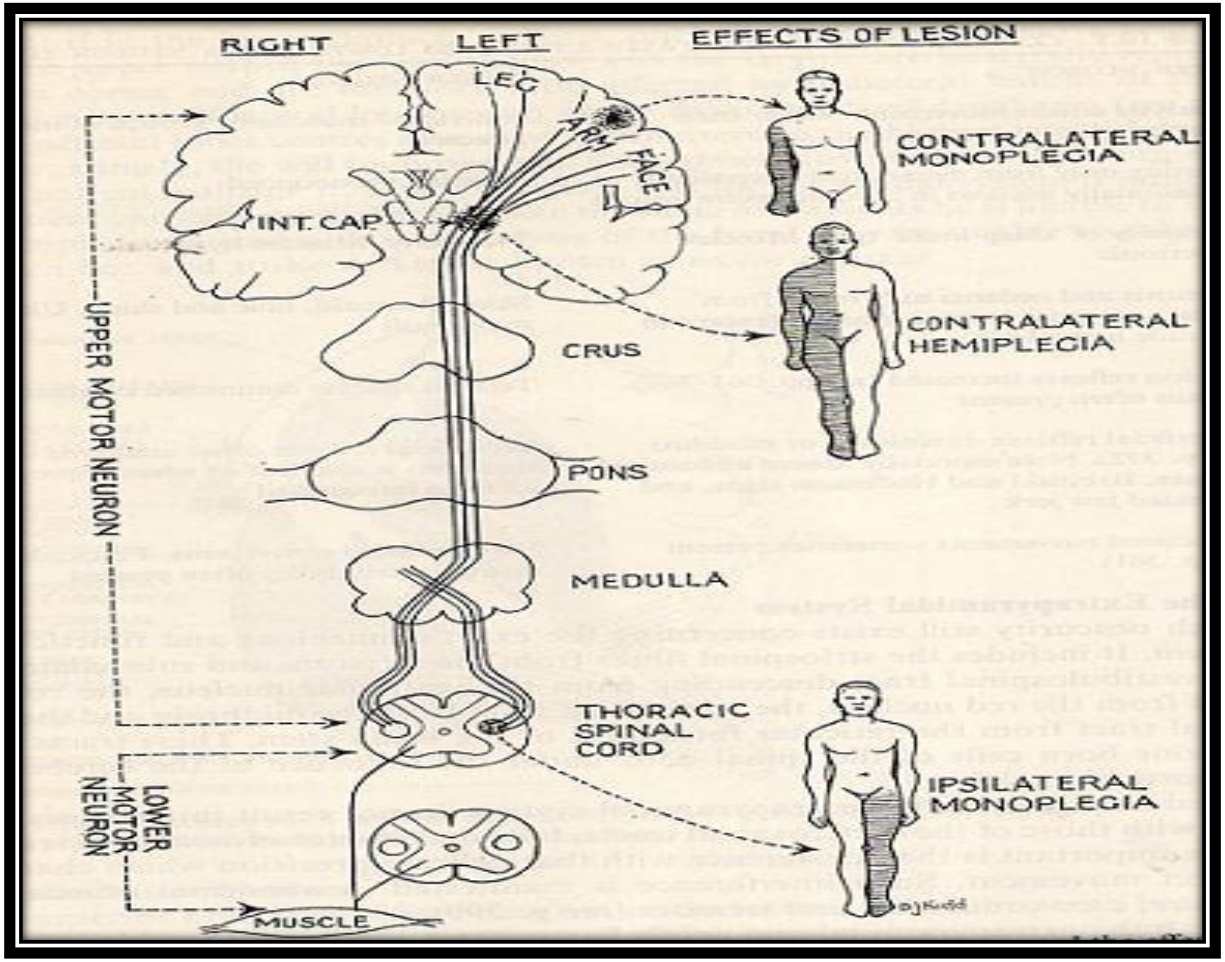
The effect of a lesion in different parts of the motor system



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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 to red nucleus, pons to cerebellum, thalamus, 3, 4, and 6 cranial nerves.

The genu:

Contains corticobulbar tract.

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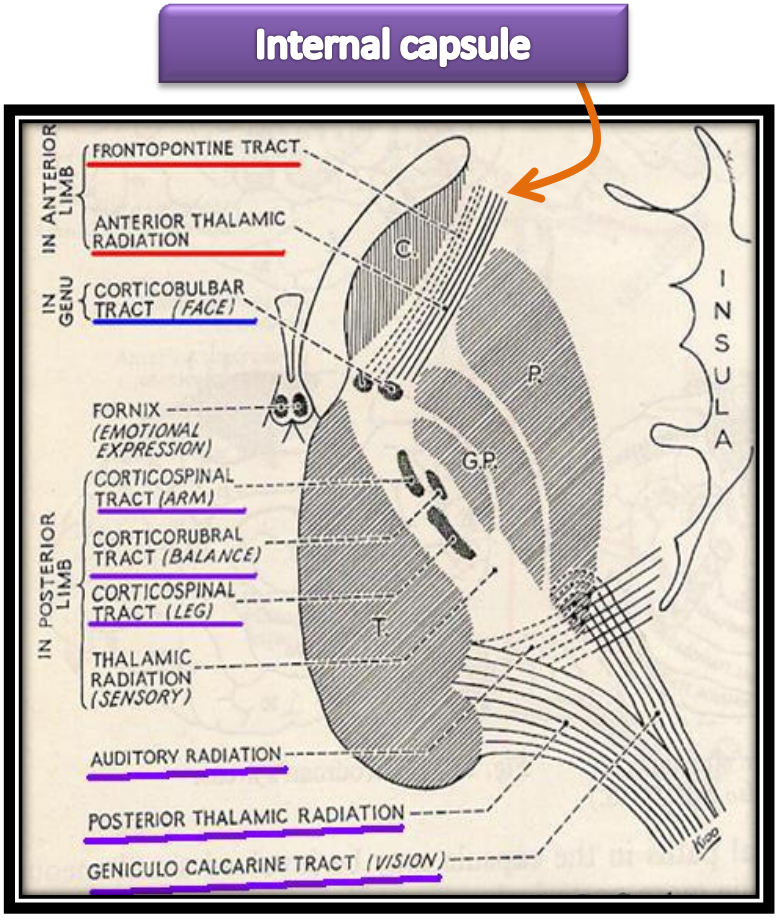
The internal capsule

The posterior limb :

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.



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Effects of unilateral lesion in the posterior limb of internal capsule

Such lesion commonly called **cerebral stroke** is usually caused by **thrombosis** or **hemorrhage** (1) of **lenticulo-striate artery** (a branch of the middle cerebral artery).

* Patients pass into an **acute** then **chronic stage.**

A) Acute stage:

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

- **Flaccid paralysis** including the upper and lower limbs, the lower parts of the face and half of the tongue. (excitatory reticulospinal tract still not take the upper hand)
- **Hemianaethesia** (loss of all sensations).
- **Hypotonia and areflexia.**
- **Loss of the superficial reflexes** (because it receives facilitation from the higher center)
- **May be +ve Babinski's sign.** (may appear or not)

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 hemianaethesia.
c. There may be +ve Babinski's sign
d. Absence of muscle atrophy.

(1) Cause motor & sensory manifestations due to damage of posterior limb of internal capsule.

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Effects of unilateral lesion in the posterior limb of internal capsule

B) Chronic (permanent or spastic) stage:

The main manifestations of this stage include the following:

- **Contralateral hemiplegia of the UMNL type.** (spastic paralysis)

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.

- **Permanent loss of fine sensations in the opposite side, but the crude sensations recover gradually.**
- **Contralateral homonymous hemianopia (loss of vision in the opposite haves of the 2 visual fields due to interruption of signals from the temporal part of ipsilateral retina and nasal part of contralateral retina.**

Damage to the optic radiation which crossed to the opposite side. There will be loss of vision on the opposite ½ of the two visual field >> damage to the temporal part of the ipsilateral retina {e.g: right side of the internal capsule} .. So, temporal fibers of the right side & nasal fibers of the left side (because the nasal fibers cross)

- **Diminished hearing power in both areas (by about 50 %), because of damage of auditory radiation.**

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Boys' slides

Paraplegia

- Due to complete spinal cord transection (E.G. Following tumor , trauma (E.G . bullet injury , fractures spine , etc)
- The higher the level of the section, the more serious are the consequences.
- If the transection is in the upper cervical region → immediate death follows, due to paralysis of all respiratory muscles
- In the lower cervical region below the 5th cervical segment → diaphragmatic respiration is still possible, but the patient suffers complete paralysis of all four limbs (quadriplegia).
- Transection lower down in the thoracic region allows normal respiration but the patient ends up with paralysis of both lower limbs (paraplegia).

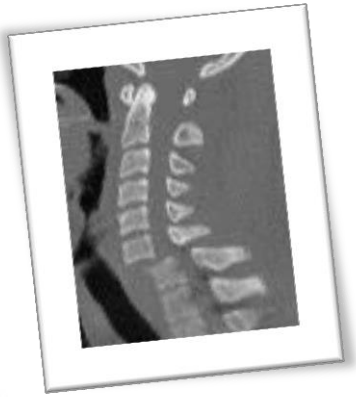
Complete Spinal cord transaction

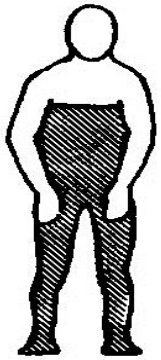
Paraplegia

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

- Transection in the upper cervical regions (above the 3rd cervical segment) >> immediate death due to respiratory arrest as in hanging.
- However, at lower levels, patients pass 3 stages: spinal shock, recovery of spinal reflex activity, then its failure and death.

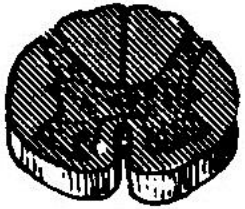
If there is a damage to the AHC (anterior horn cells) >> LMNL
If there is a damage to the corticospinal tract >> UMNL





Quadriplegia or Paraplegia according to the site of lesion

Bilateral loss of all modalities.
Bilateral leg weakness.



COMPLETE CORD LESION

Stages of Spinal cord transaction

1- Stage of Spinal Shock (weeks – months)

All cord functions (sensory & motor) are depressed

Manifestations

- **Paralysis** of all muscles below the lesion (quadriplegia or paraplegia) due to cut of UMN.
- {MOTOR}
- Complete **loss of all sensation** below the level of transection.
- {SENSATION}
- **Loss of cord reflexes** as the stretch reflex, hence the paralyzed muscles are flaccid and the deep reflexes are absent.
- (Because their center is in the S.C.)
- **ABP drops markedly** if the transaction is at the level of the first thoracic segment due to sympathetic activity block. However, the pressure returns to normal within a few days (1)

Loss of control of micturition and defecation reflexes (facilitatory pathways from the higher centers are interrupted by the transaction leading to retention with overflow with dribbling of urine by a full bladder).

NO contraction of the bladder

- **Loss of erection.**
- (Their centers in the S.C.)

(1) ABP is maintained by vasoconstrictor tone (thoracolumbar out flow) – sympathetic. Lateral horn cells give origin for vasoconstrictor fibers to the arterioles to maintain the muscle tone of the arterioles. So, there is a continuous sympathetic discharge to the smooth muscles of the arterioles to maintain blood vessels constricted .. Thus maintain BP.

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Spinal Shock

Causes 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 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.

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Cont.

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. **(hypothermia)**

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.**

Stages of Spinal cord transaction

2- Stages of recovery of reflex activity

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

Manifestations

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 centers* below the level of the transaction 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. **(reflex contraction due to stretch of the bladder wall)**
- Erection can occur by direct stimulation and ejaculation follows.

Touch of the patient with a relatively noxious stimulus applied to the skin produces a flexor withdrawal reflex and 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 as flexor spasm of both lower extremities and contraction of the anterior abdominal wall.

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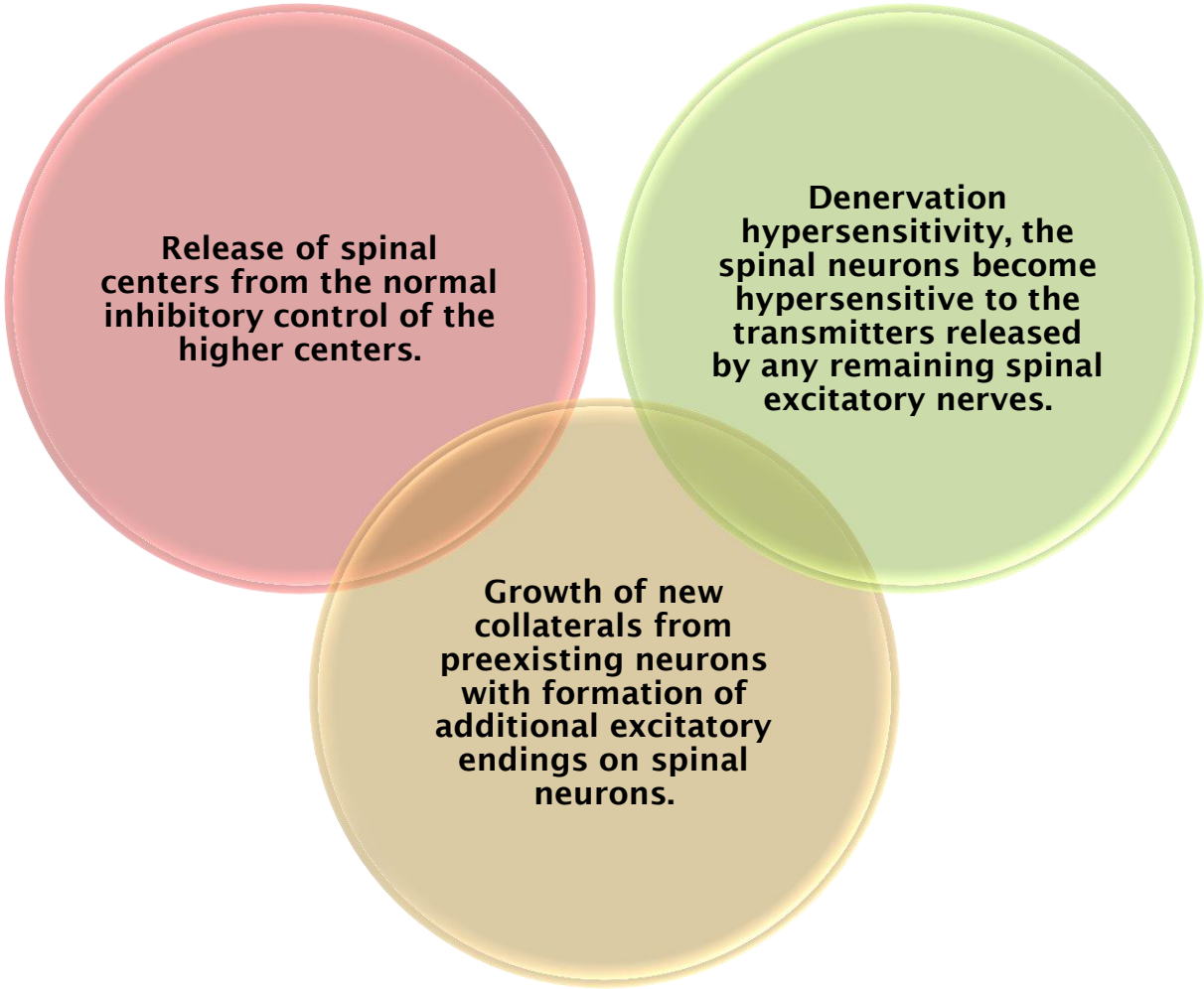
Reappearance of spinal reflexes may be due to:



Stages of Spinal cord transaction

3- Stage of failure of reflex activity

Manifestations

- 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 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	**At the level of lesion & ipsilateral**	**Below the level of lesion**
• Cutaneous hyperaesthesia i.e. increased sensibility to pain, touch & temp. occurs in ipsilateral dermatome due to irritation of the dorsal nerve roots by the neighboring lesion.	• 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 (both superficial and deep) mediated by damaged segments	***On the same side*** • Paralysis of voluntary muscles (UMNL). • Dorsal column sensations are lost. • 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

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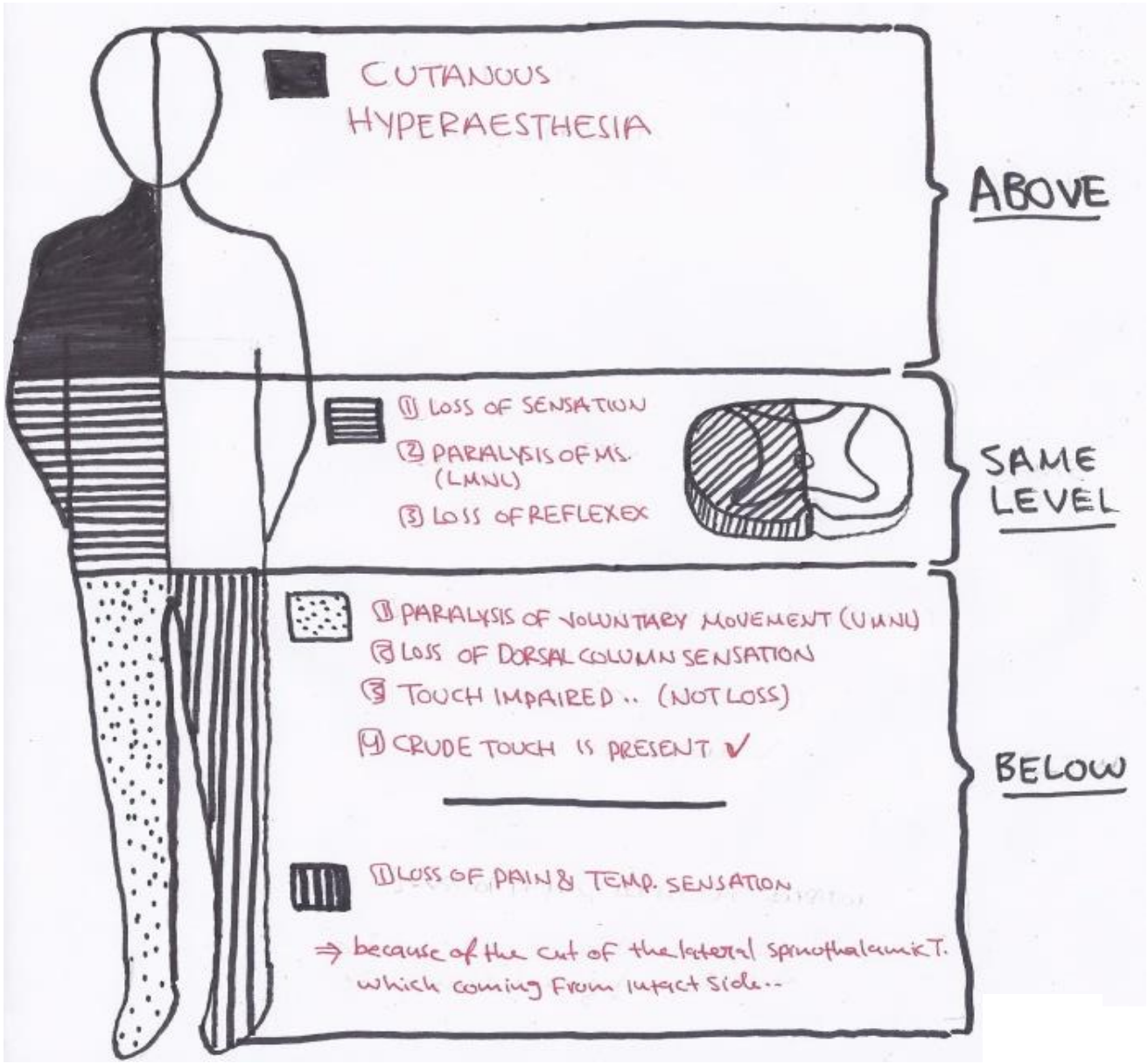
Hemisection of spinal cord
(brown sequard syndrome)



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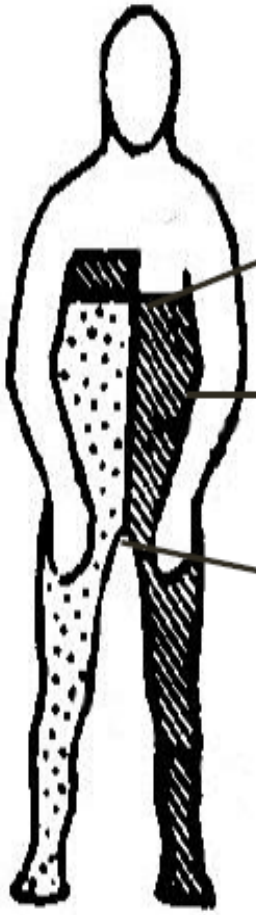
Hemisection of spinal cord
(brown sequard syndrome)

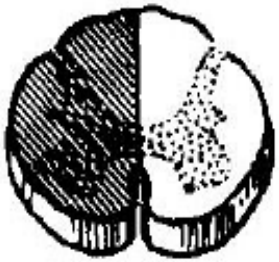


Loss of all modalities at one or several dermatome levels.

Loss of pain and temperature below a specific dermatome level.

Loss of proprioception and 'discriminatory' touch up to similar level and limb weakness.

BROWN-SEQUARD SYNDROME



(Partial cord lesion)

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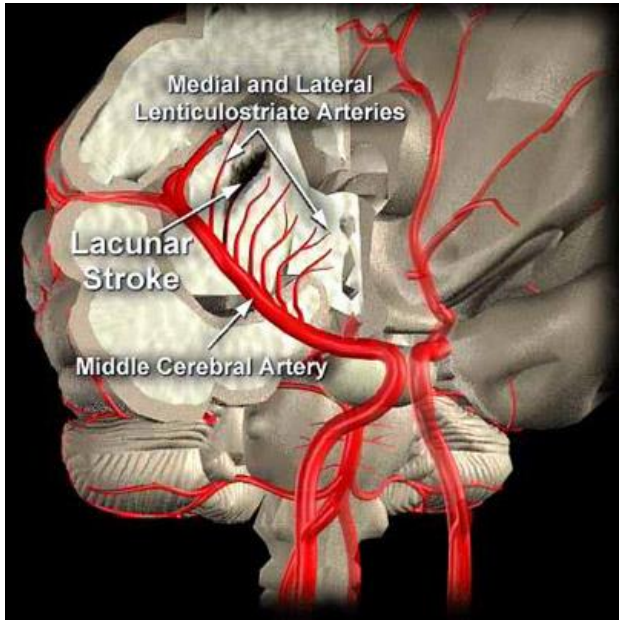
Hemiplegia

Boys' slides

Causes of Hemiplegia

- Cerebral heamorrhage, thrombosis or embolism (Difference???) → results in paralysis of the opposite half of the body .
- The commonest cause of cerebral haemorrhage is hypertension , usually associated with rupture of the lenticulo-striate branch of the middle cerebral artery in the internal capsule .



Features of Hemiplegia

(1) UMNL involving the half of the body contralateral to the site of the lesion .
(2) Hypertonia causes the limbs to acquire a specific posture → (Next slide)

Hemiplegia

Upper limb is:

- Adducted to the side of the trunk.
- Flexed at the elbow.
- The forearm is semipronated.
- With flexion of the wrist and fingers.

Lower limb is:

- Adducted
- Extended at the knee and ankle.

(3) Loss of sensation on the opposite side of the body (Hemianesthesia), due to damage of the thalamocortical fibers.
(4) Homonymous hemianopia (loss of vision in two corresponding halves of the visual fields in both eyes), may occur if the optic radiation is lesioned.

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SUMMARY

According to	Upper	Lower
Extent of paralysis	Wide spread	Localized
Site of paralysis	Opposite site	Same side
Tone of the muscles	Hypertonia and hyperreflexia	Hyotonia
Superficial reflexes	Absent	Absent
Deep reflexes	Exaggerated	Absent
Wasting of the muscles	Very slight	Very marked
Response of muscles	Normal	Abnormal

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

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SUMMARY

Site of Lesion			Effects	
Area 4			restricted paralysis in the opposite side e.g. monoplegia	
Corona Radiata			contralateral monoplegia or hemiplegia	
Internal Capsule			contralateral hemiplegia	
Brain Stem		midbrain	crossed hemiplegia: (contralateral hemiplegia + Ipsilateral paralysis of the CN)	3rd & 4th CN
		pons		5th, 6th, 7th, and 8th CN
		medulla		9th, 10th, 11th & 12th CN
Spinal Cord	bilateral	upper cervical	interruption of the respiratory pathway	
		lower cervical	quadriplegia	
		midthoracic	paraplegia	
	unilateral	cervical region	Ipsilateral hemiplegia	Ipsilateral paralysis (LMNL)
		midthoracic	Ipsilateral monoplegia	

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QUESTIONS

1- A
2- C
3- C
4- C

1- Which of the following is a sign for UMN lesion ?

A- disuse atrophy
B- denervation atrophy
C- hypotonia
D- fasciculation

2- Lesion in corona radiata can produce ?

A- contralateral quadriplegia
B- ipsilateral quadriplegia
C- contralateral monoplegia
D- ipsilateral monoplegia

3- Bilateral lesion in the spinal cord (in midthorasic) lead to ?

A- quadriplegia
B- monoplegia
C- paraplegia
D- hemiplegia

4- Which one of the following is a manifestation of brown sequard syndrome ?

A- hyperaesthesia at the level of lesion
B- Loss of all reflexes below the level of the lesion
C- contralateral Loss of pain & temperature below the level of lesion
D – lose of Dorsal column sensations above the level of lesion

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THE END

If there are any Problems or Suggestions,
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THANK YOU