بسم الله الرحمن الرحيم

السلام عليكم ورحمة الله وبركاته

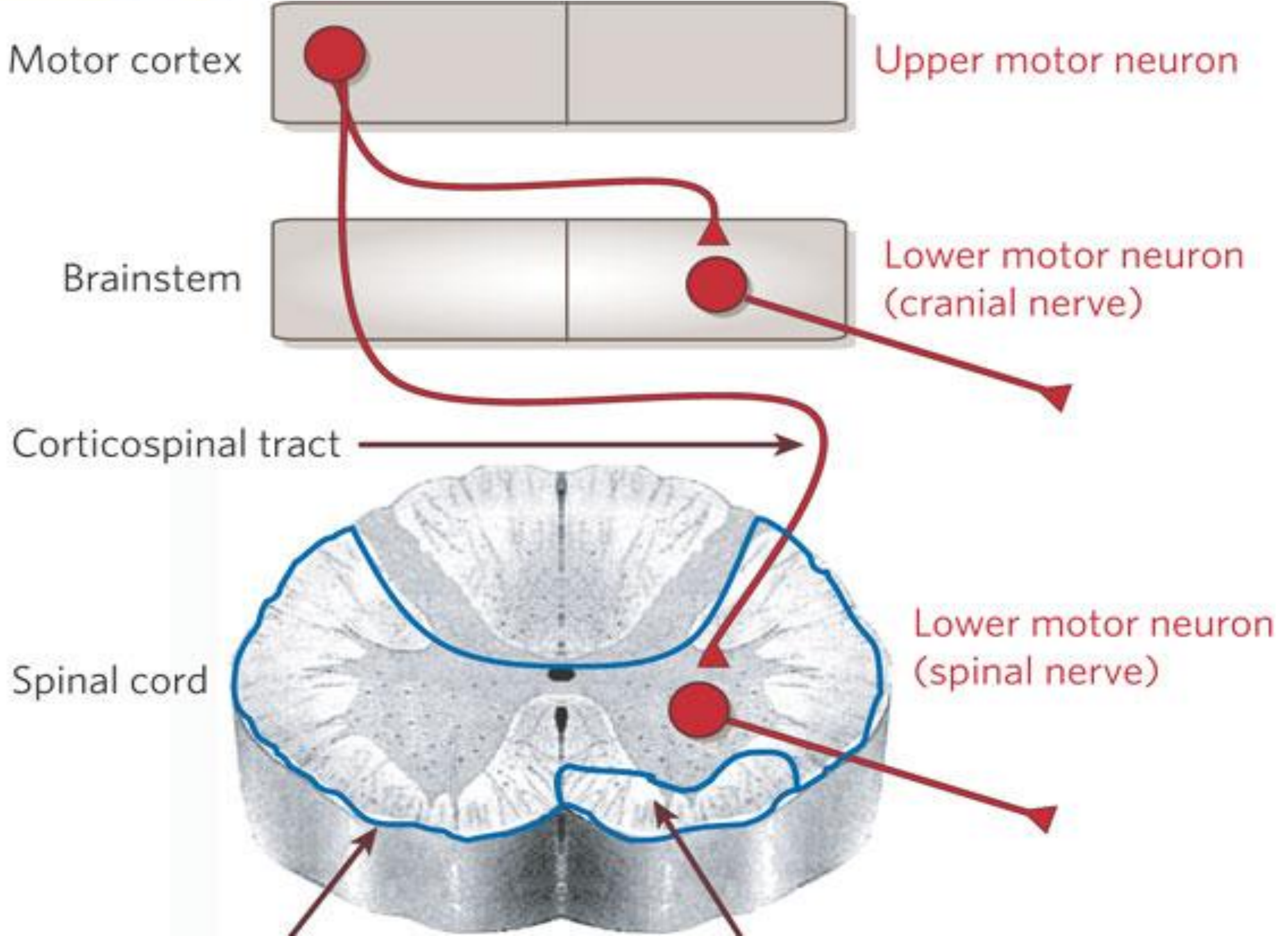
MOTOR LESIONS

Dr. Hayam Gad

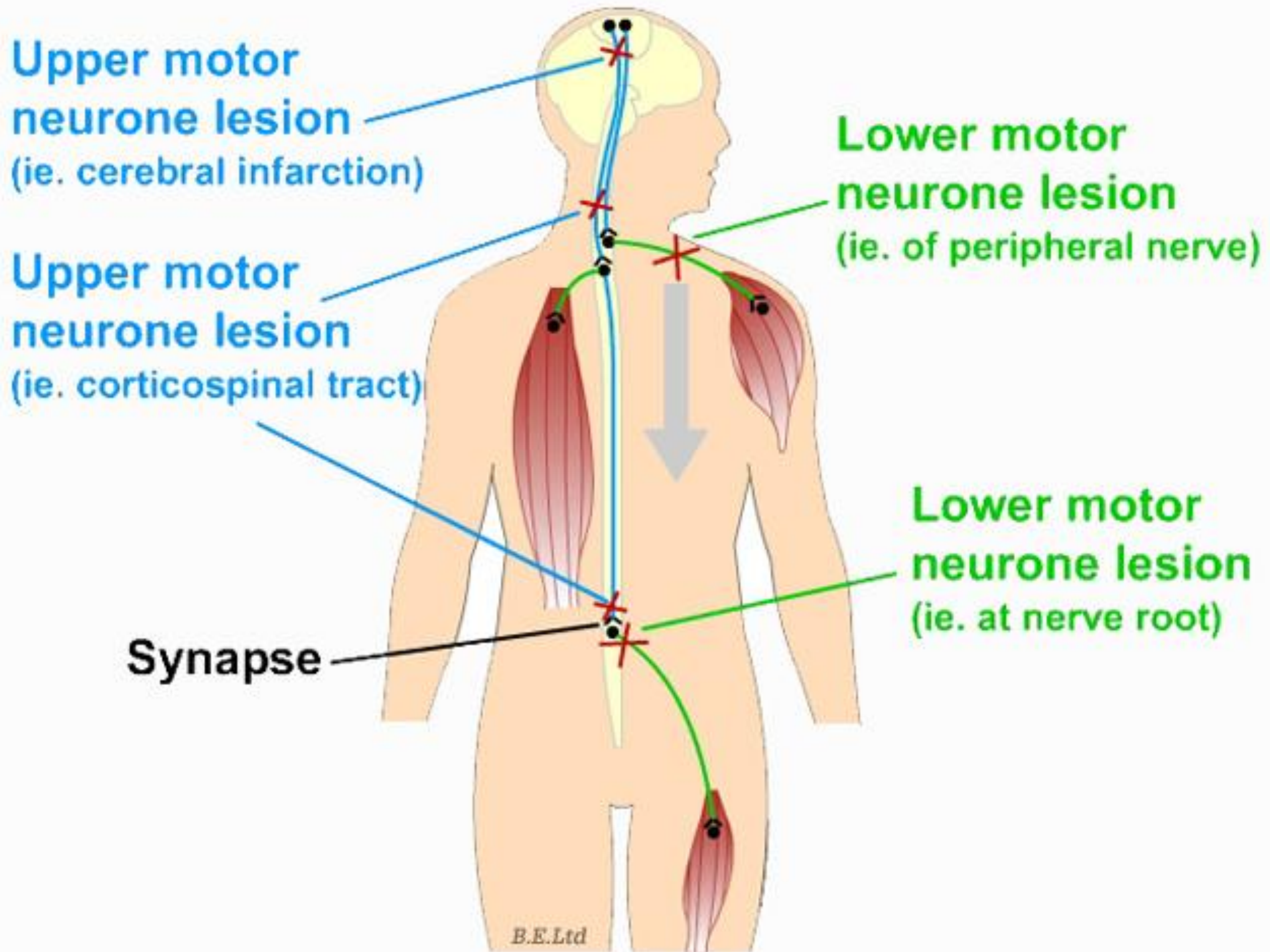
Associate Professor of Physiology

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.



Upper and Lower motor neurones



Upper and Lower Motor Neurone Lesions

	UMNL	LMNL
Extent of paralysis:	Wide spread	localized
Site of paralysis:	Opposite to the lesion	On the same side of the lesion
Tone of the muscles:	Hypertonia and hyperreflexia	Hyotonia
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 movements	Very marked d.t absence of reflex tone & lack of voluntary movements
Response of muscles to electrical stimulation	Normal, with normal excitability.	Weak contraction with decreased excitability, then no response when it is transformed into fibrous tissues

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- <u>In area 4:</u>

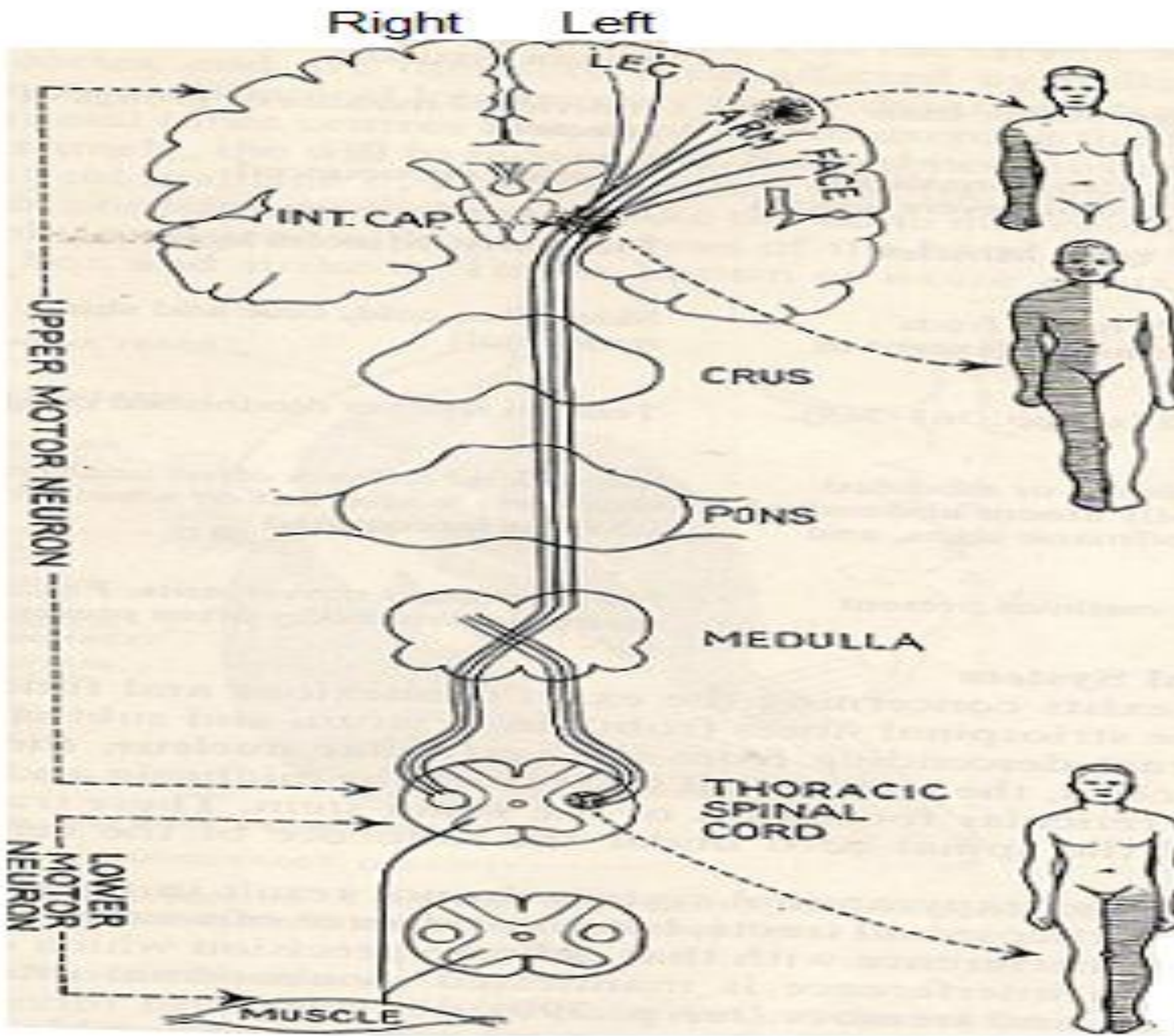
This leads to restricted paralysis in the opposite side e.g. monoplegia (paralysis of one limb because area 4 is widespread so it is rarely damaged completely.

2- <u>In the corona radiata:</u>

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

3- <u>In the internal capsule:</u>

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



Effect of lesion

Contralateral Monoplegia

Contralateral hemiplegia

Ipsilateral Monoplegia

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

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

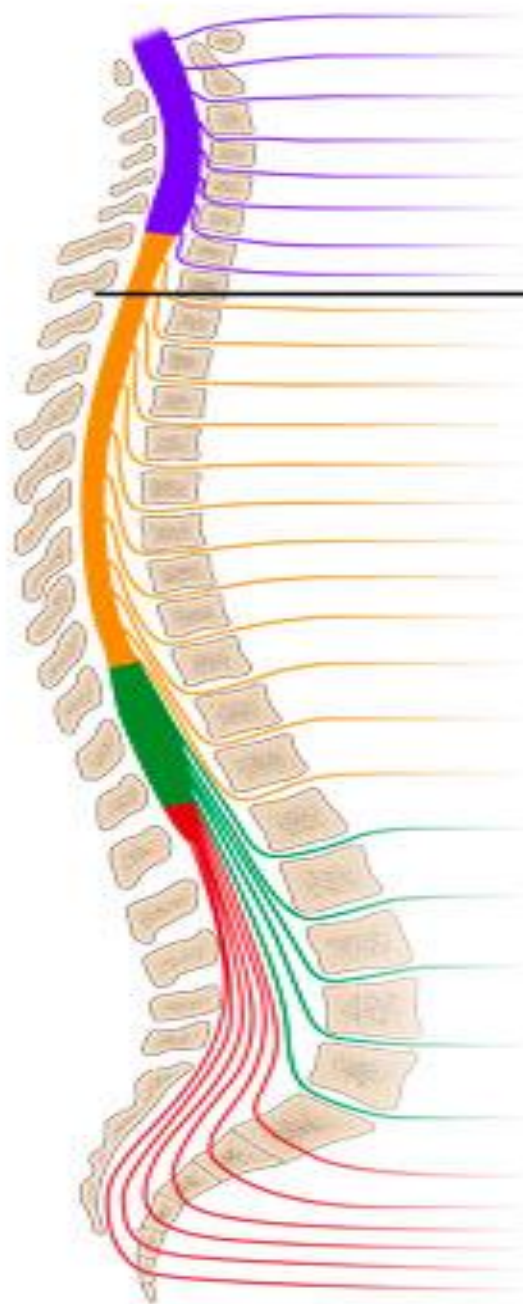
- If the lesion was in the midbrain, the 3rd & 4th are affected.
- If the lesion was in the pons, the 5th, 6th, 7th, and 8th cranial nerves are affected.
- If the lesion was in the medulla, the 9th, 10th, 11th & 12th cranial nerves are affected.
- Bilateral lesion in the brain stem is rare and leads to quadriplegia and bilateral paralysis of the cranial nerves.

5- In the spinal cord:

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**.

Acute Spinal Cord Injury



Quadriplegia
(loss of movement
and sensation
in all four limbs)

Paraplegia
(loss of movement
and sensation
in the lower half
of the body)

b) Unilateral lesions:

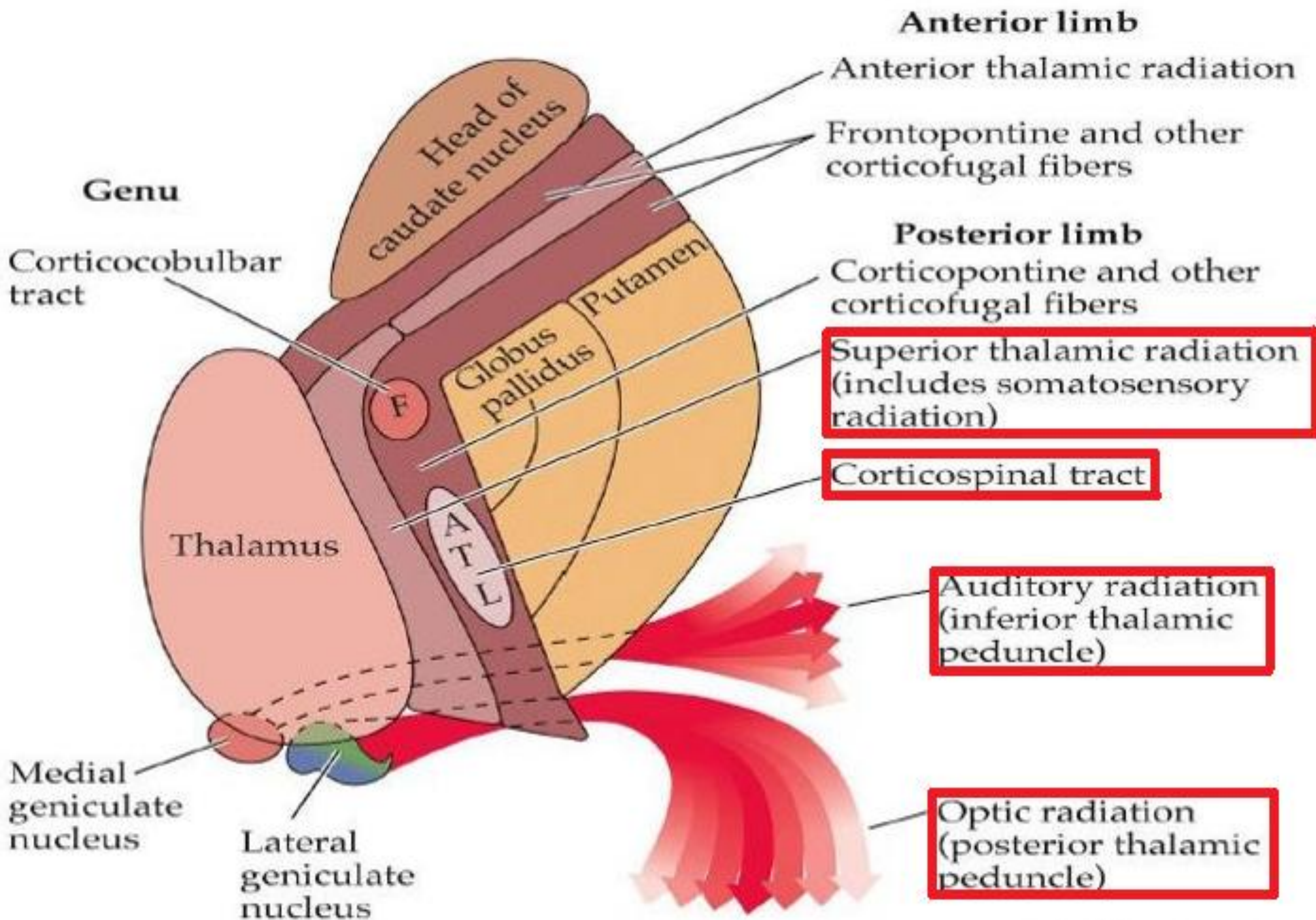
- In the cervical region, they lead to ipsilateral hemiplegia.
- In the midthoraic lesion they lead to ipsilateral monoplegia in the corresponding lower limb.
- In both conditions, there is ipsilateral paralysis (LMNL) of the muscles at the level of the lesion due to damage of the spinal motor neurons.

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.

- 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.



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 (a branch of the middle cerebral 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 manifestations in the opposite side:

- 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 more widespread than in LMNL.

b. There is associated hemianaethesia.

c. There may be +ve Babinski's sign

d. Absence of muscle atrophy.

Chronic (permanent or spastic) stage:

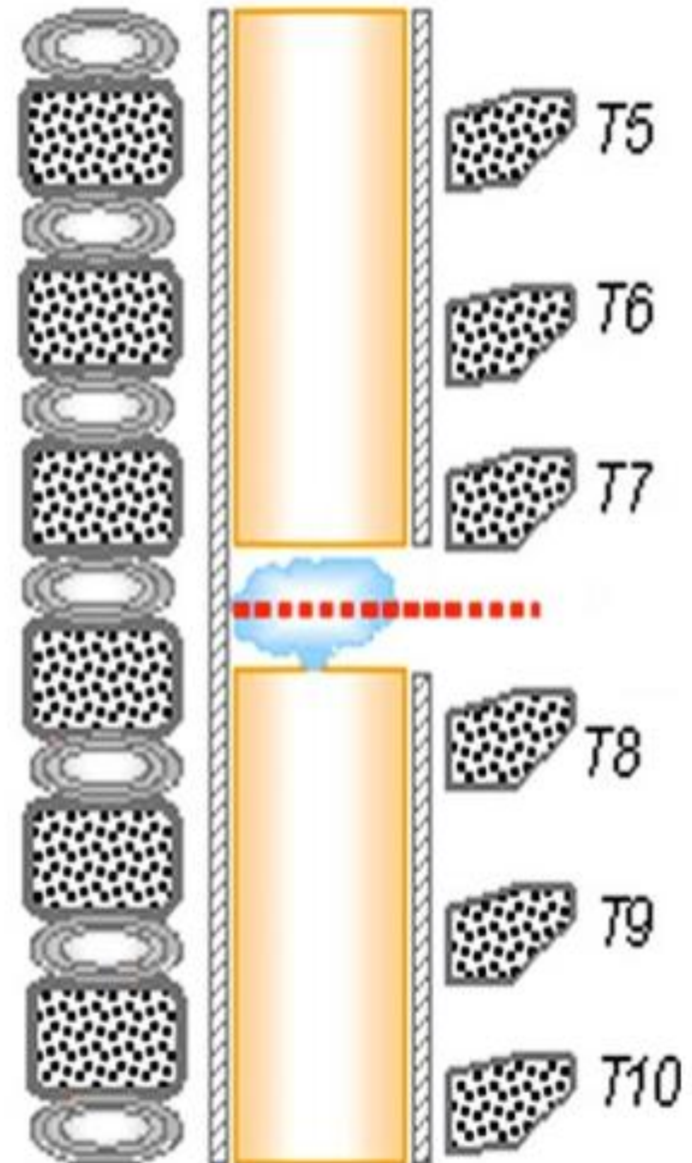
The main manifestations of this stage include the following:

- Contralateral hemiplgia of the UMNL type.
- 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.
- Diminished hearing power in both areas (by about 50 %), because of damage of auditory radiation.

Complete spinal cord transaction

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



Complete spinal cord transaction (cont.)

- Transection in the upper cervical regions (above the 3rd cervical segment) results in 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.

The following stages follow cord transaction:

I- Stage of spinal shock (weeks to months in man)

All cord functions are depressed.

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.

- **Loss of cord reflexes** as the stretch reflex, hence the paralysed muscles are flaccid and the deep reflexes are absent.
- **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.
- **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).
- **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:

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

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.
- Erection can occur by direct stimulation and ejaculation follows.

- Touch of the patient’s skin with a relatively noxious stimulus produces a flexor withdrawal reflex.
- 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, the spinal neurons become hypersensitive to the transmitters released by any remaining spinal excitatory nerves.
- Growth of new collaterals from preexisting neurons with formation of additional excitatory endings on spinal neurons.

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.

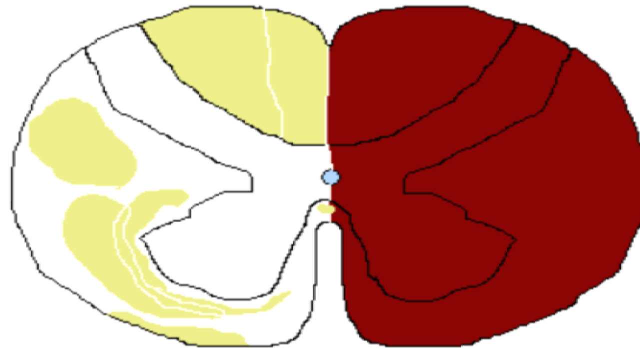
Hemisection of spinal cord
(Brown Sequard syndrome)

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.

Hemisection of the spinal cord
(causes the Brown-Séquard syndrome)

Can you predict the resulting disabilities?

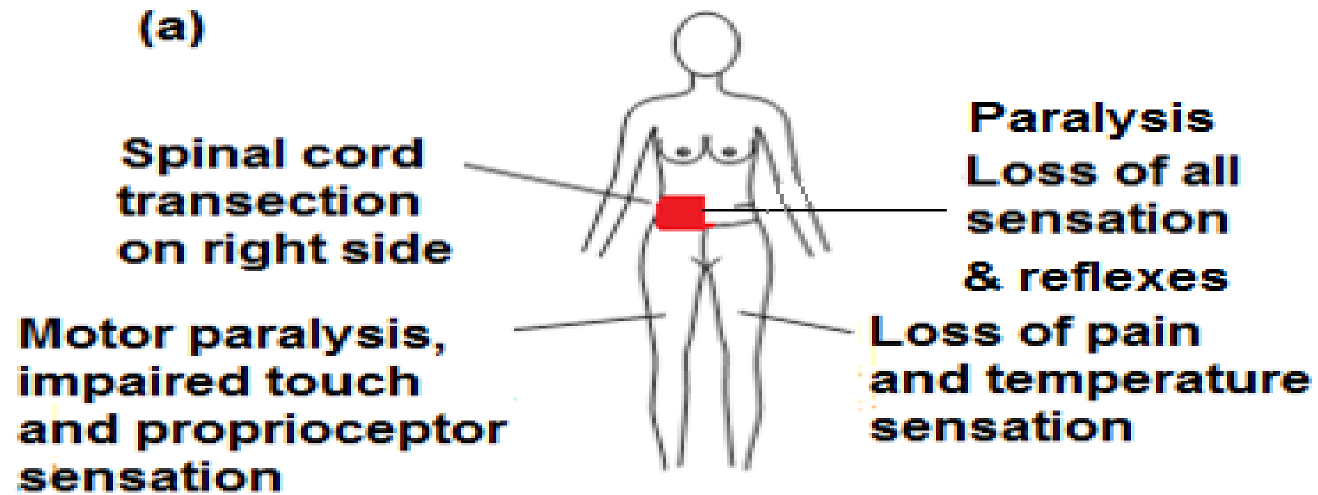
LEFT



RIGHT

Transection of half the spinal cord
(A stab wound between the laminae of vertebrae C5 and C6 could do this.)

The permanent effects of this lesion (after recovery from the initial state of "spinal shock" are attributable to severing the axons of the descending motor tracts (reticulospinal and corticospinal) and of the two major ascending sensory pathways (dorsal columns and spinothalamic tract).



(b)

Dorsal column

Corticospinal tract

Spinothalamic tract

Cord lesion

Mechanoceptor afferents

Thermoceptor/ nociceptor afferents

Midline

BROWN-SEQUARD SYNDROME

MANIFESTATIONS:

Above 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.

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 (both superficial and deep) mediated by damaged segments.

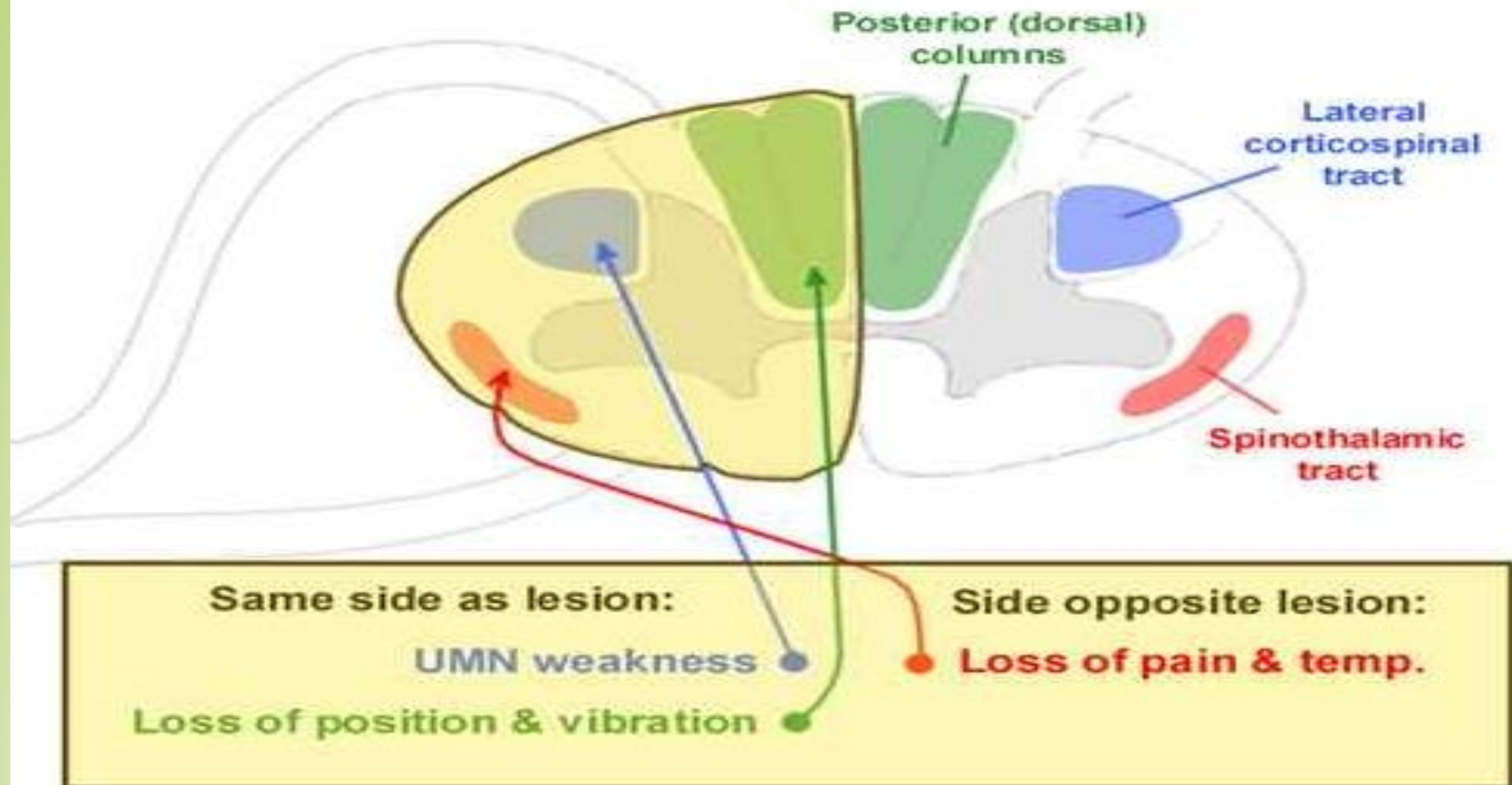
Below the level of lesion

On the same side

- Paralysis of voluntary muscles (UMNL).
- Dorsal column sensations are lost.
- Touch is impaired (but not lost) because 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.



Impairments

Thank You