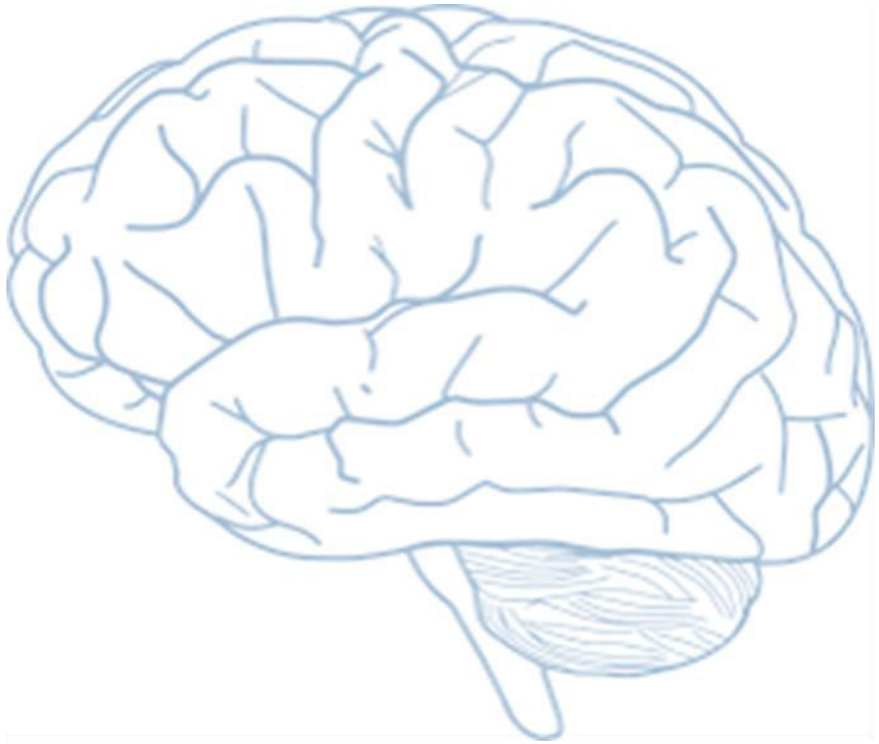
MEDICINE
KING SAUD UNIVERSITY

تمت المراجعة
شوق الأحمري & عبدالعزيز العنقري



Physiology Team 436

CNS PHYSIOLOGY

- Text
- Important
- Formulas
- Numbers
- Doctor notes
- Notes and explanation

Lecture No.24

"You Will Never Have This Day Again, So Make It Count ".

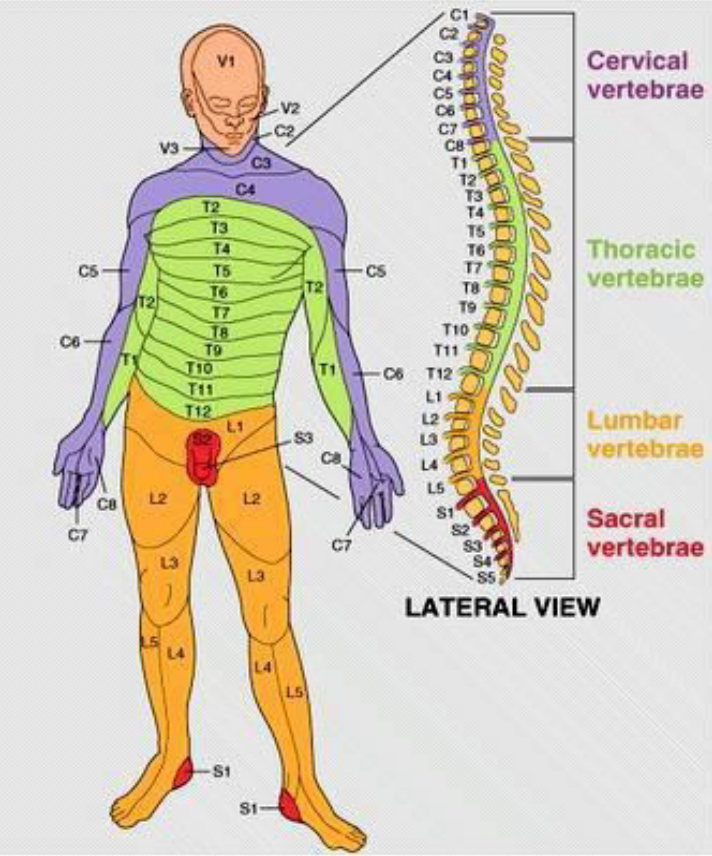
UPPER & LOWER MOTOR NEURON LESION

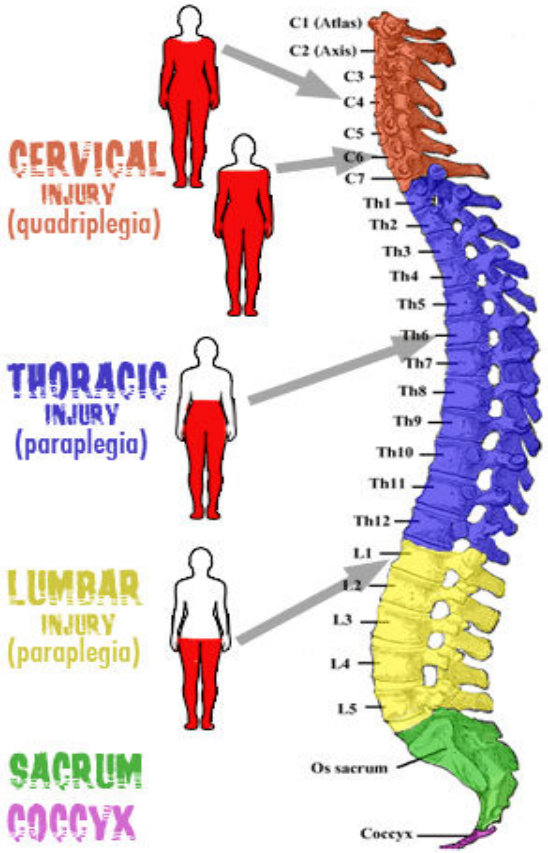
Objectives:

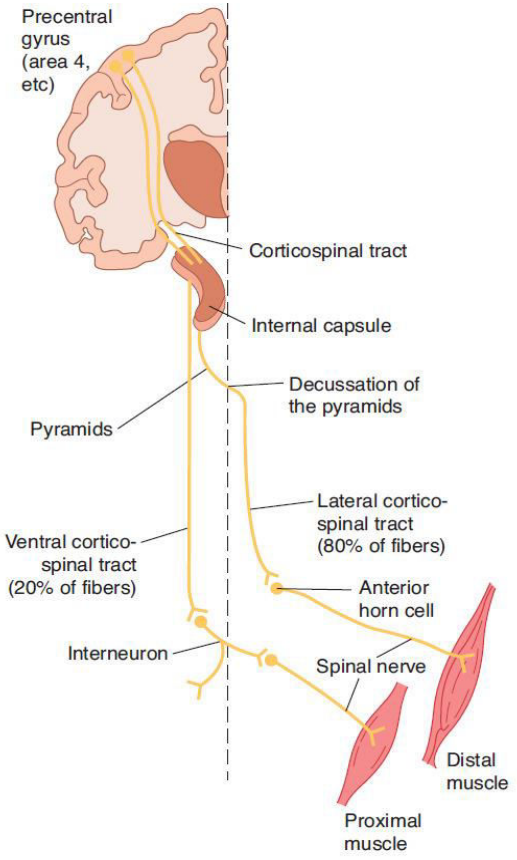
1. Appreciate what is meant by upper and lower motor neurons and their functional anatomy
2. Explain and differentiate between features of lesions of the upper and lower motor neurons
3. Describe effects of lesions in pyramidal tracts
4. Describe effects of lesions in the internal capsule
5. Explain the manifestations of complete spinal cord transection and hemisection
6. Explain features of Brown Sequard Syndrome
7. Correlate the site of lesion with pattern of loss of sensations
8. Describe facial, bulbar and pseudobulbar palsy

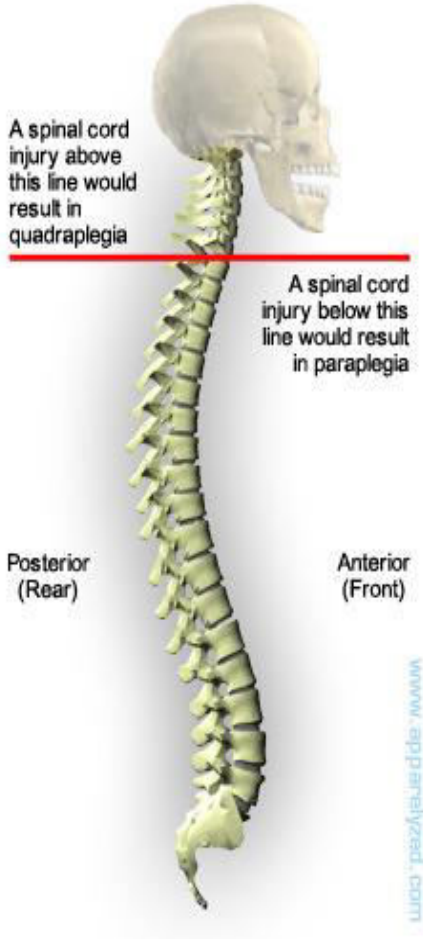
introduction

- 31 segments: embryological development → growth of cord lags behind → mature spinal cord ends at L1.
- Upper cervical cord lesions produce quadriplegia and weakness of the diaphragm.
- Lesions at C4-C5 produce quadriplegia.









ONLY IN MALES' SLIDES

Comaprison between upper & lower motor neuron lesions

Two sets of neurons are important

Upper motor neurones

- Paralysis affect movements.
- Wasting not pronounced.
- Spasticity Muscles
- hypertonic (Clasp Knife).
- Tendon reflexes increased.
- Superficial reflexes diminished.
- Babinski's sign +ve.
- NCV- normal
- No denervation potentials in EMG

Characteristic of upper motor neurone lesions:

- no wasting.
- Loss of skilled finger/toe movements
- increased tone of claspknife Type.
- weakness most evident
- in anti-gravity muscles.
- increased reflexes and clonus.
- • extensor plantar responses.

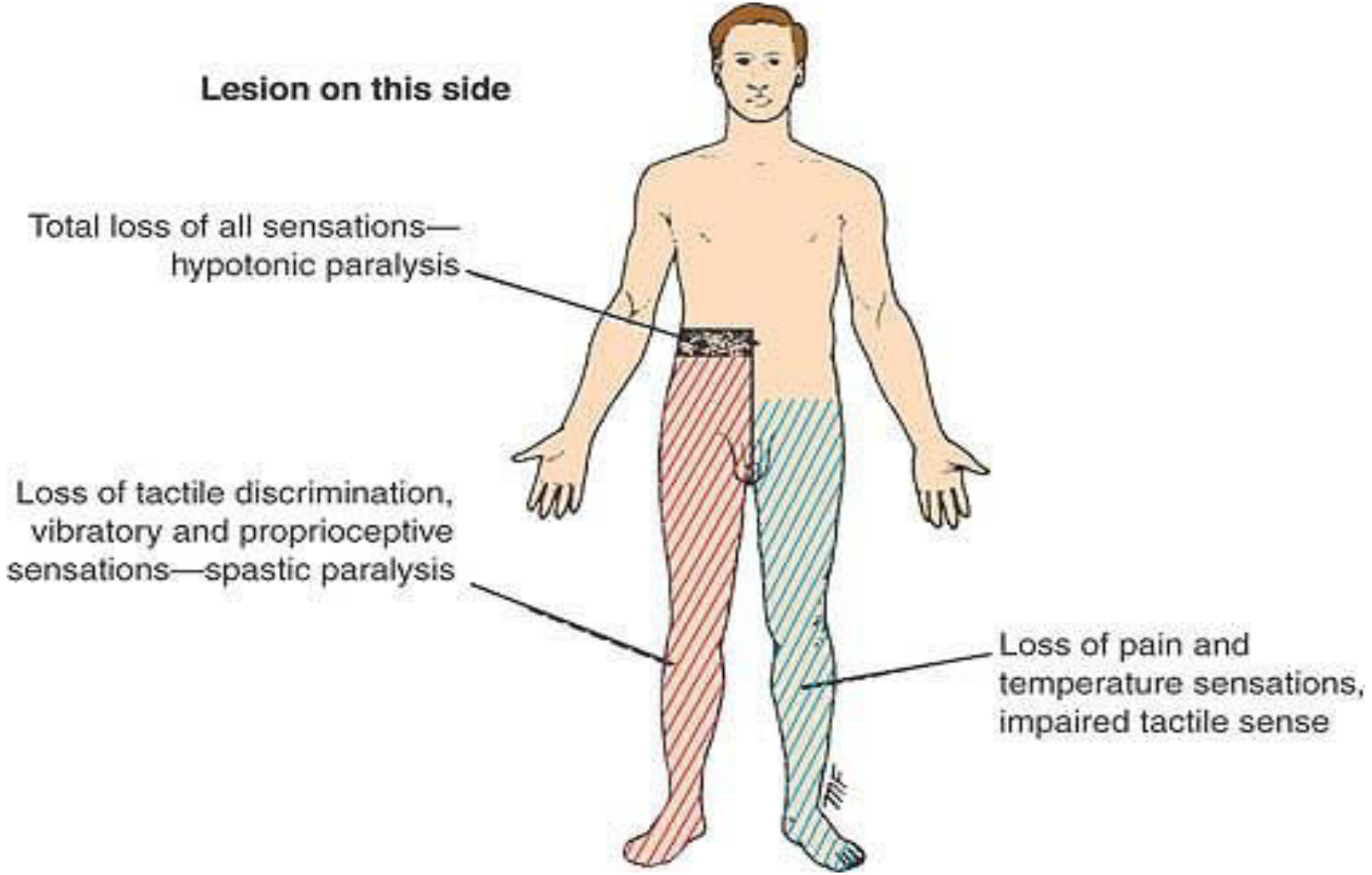
Lower motor neurones

- Individual muscle or group of muscles are affected.
- Wasting pronounced.
- Flaccidity. Muscles hypotonic.
- Tendon reflexes diminished or absent.
- Ncv- abnormal
- Denervation potentials in EMG (fibrillations)
- Muscle contractures
- Trophic changes in skin and nails.

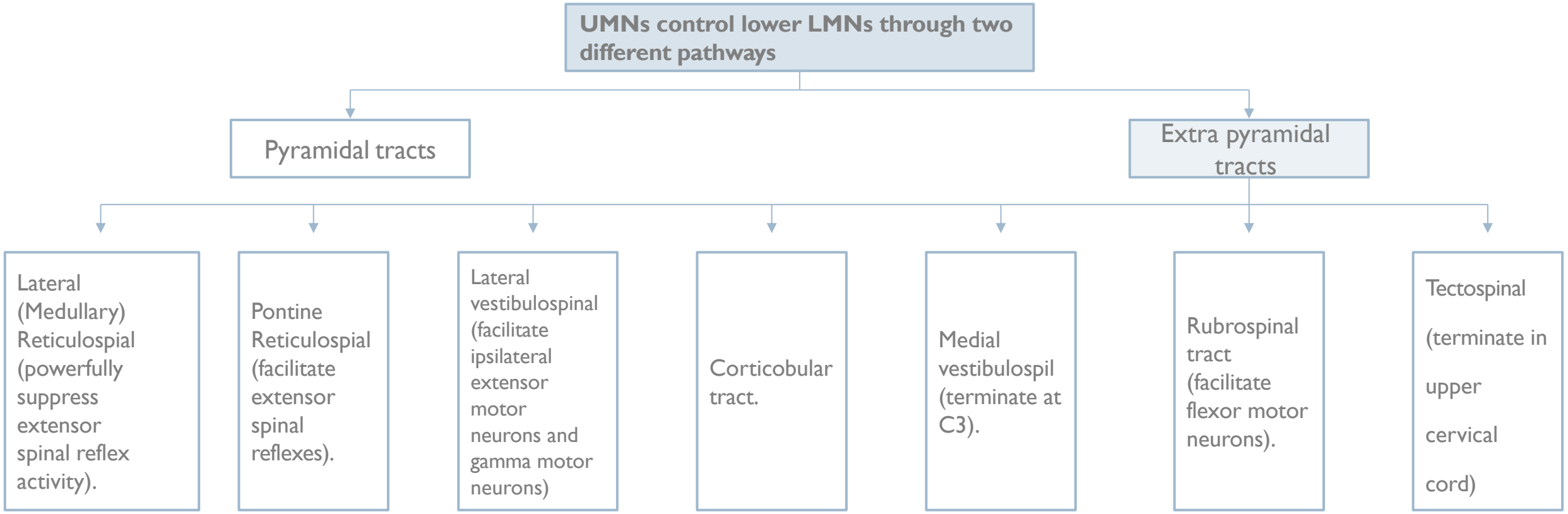
Characteristic of upper motor neurone lesions:

- Wasting.
- Fasciculation (tapping produce it)
- Decreased tone (i.E. Flaccidity)
- Weakness.
- Decreased or absent reflexes.
- Flexor or absent plantar responses.

Cont.


Lesion on this side
Total loss of all sensations—hypotonic paralysis
Loss of tactile discrimination, vibratory and proprioceptive sensations—spastic paralysis
Loss of pain and temperature sensations, impaired tactile sense

UMNs control lower LMNs through two different pathways



Origin of extrapyramidal tracts: From area (6) and area (4) → descends to corpus striatum → Globus pallidus → from the globus pallidus fibers pass to:
1- Reticular formation 2- Vestibular nuclei 3- Red nucleus 4-Tectum of mibrain → these nuclei give the extrapyramidal tracts.

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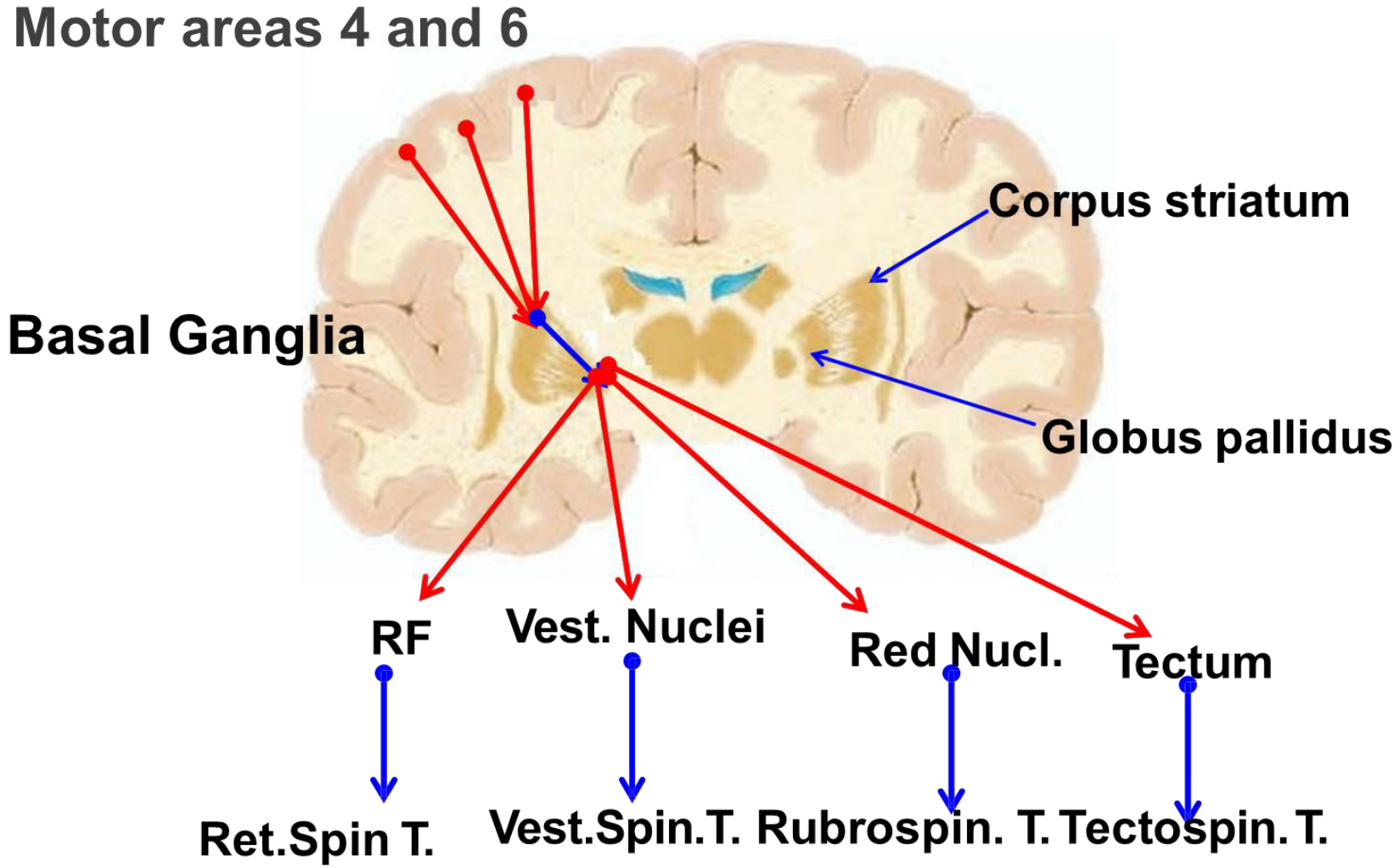
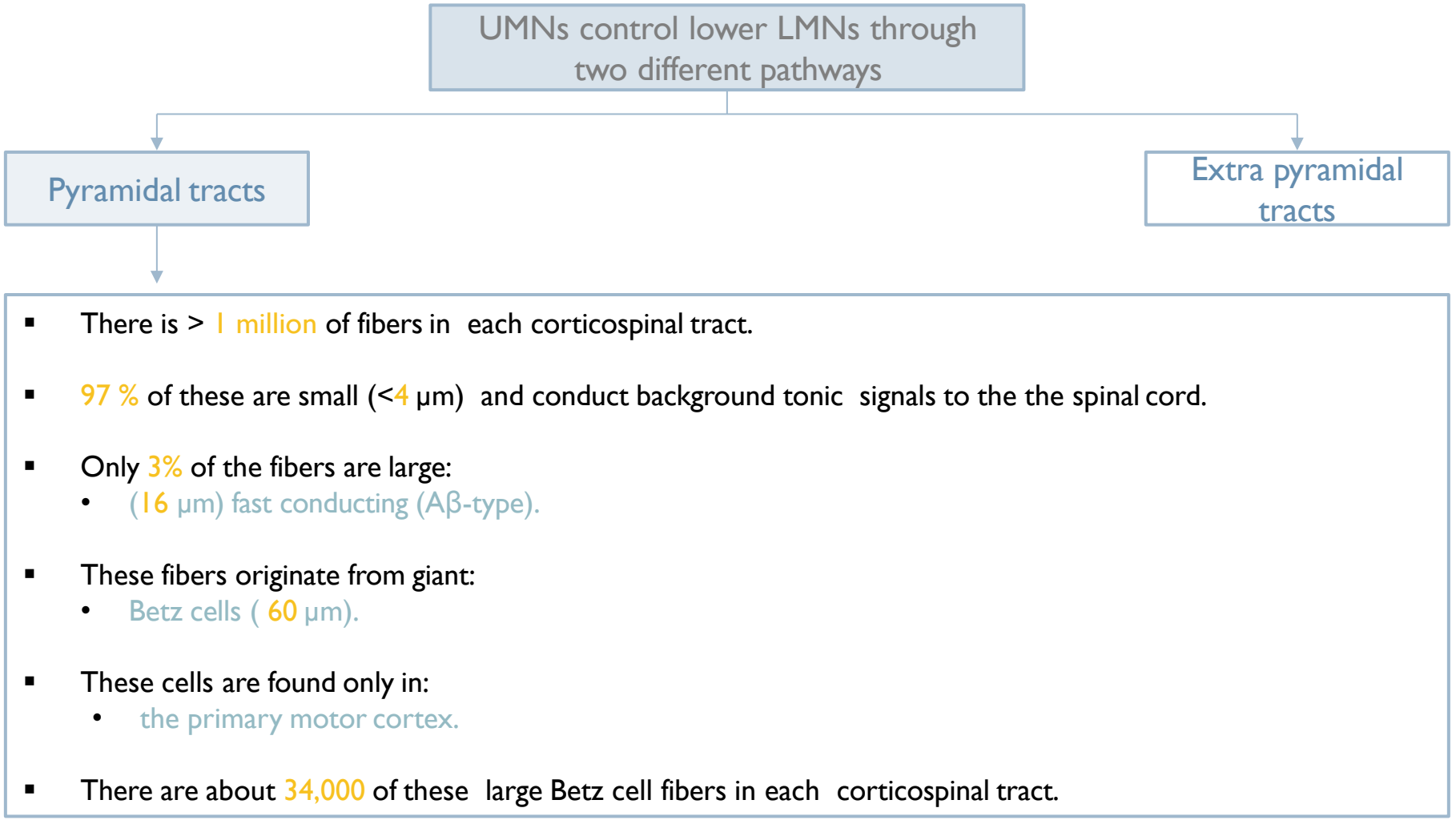
Extra pyramidal tracts



UMNs control lower LMNs through two different pathways



What are upper & lower motor neuron lesions?

Lesions

UMNL

Is a lesion of the descending neural pathway above the level of lower motor neurons.

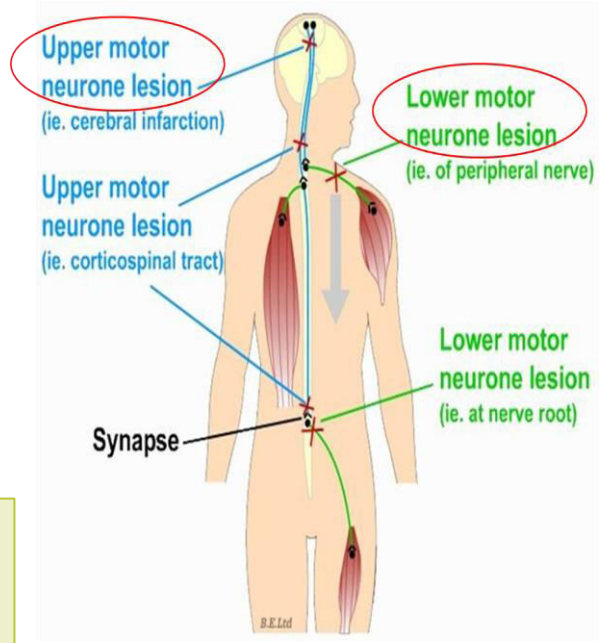
Causes:

1. Trauma
2. Tumour
3. Vascular disorders as thrombosis or hemorrhage

Most common site is the internal capsule.

- cause weakness of the
- lower part of the face on the opposite side. Frontalis is
- spared: normal furrowing of the brow is preserved; eye
- closure and blinking are largely unaffected. Lower motor
- neurone (LMN) lesions.

ONLY IN MALES' SLIDES



LMNL

It is damage of the lower motor neurons (the spinal AHCS and the cranial motor nuclei or their axons) resulting in skeletal ms paralysis

Causes:

1. Trauma
2. Neuropathy

- causes weakness (ipsilateral) of all facial
- expression muscles. The angle of the mouth falls; unilateral
- dribbling develops. Frowning (frontalis) and eye closure are
- weak. Corneal exposure and ulceration occur if the eye does
- not close during sleep.

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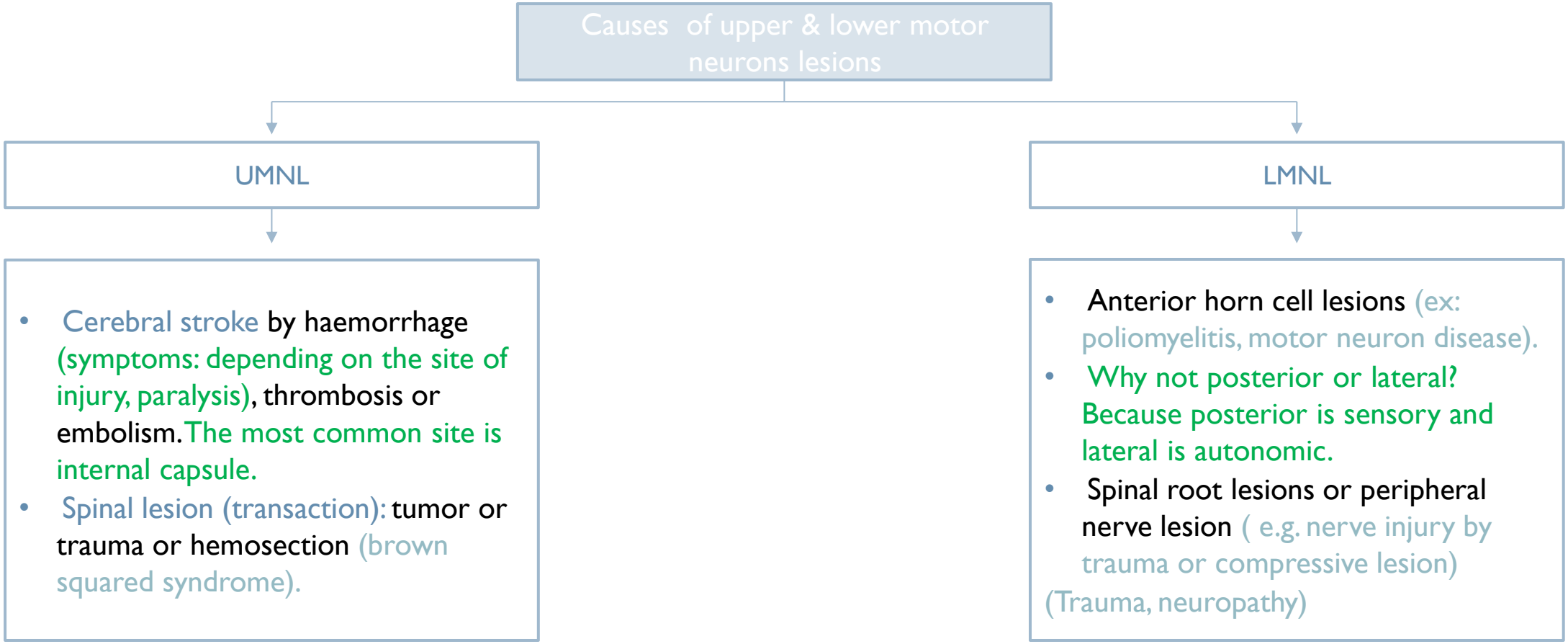
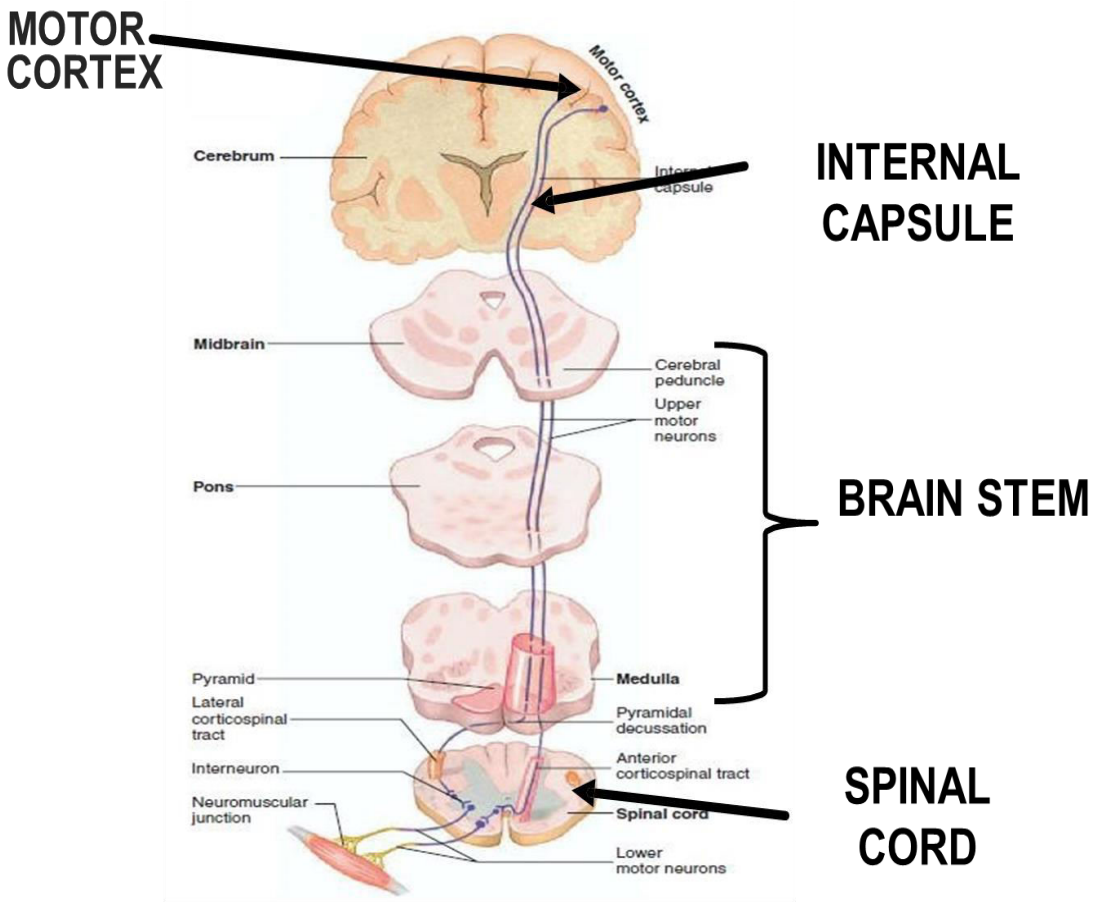
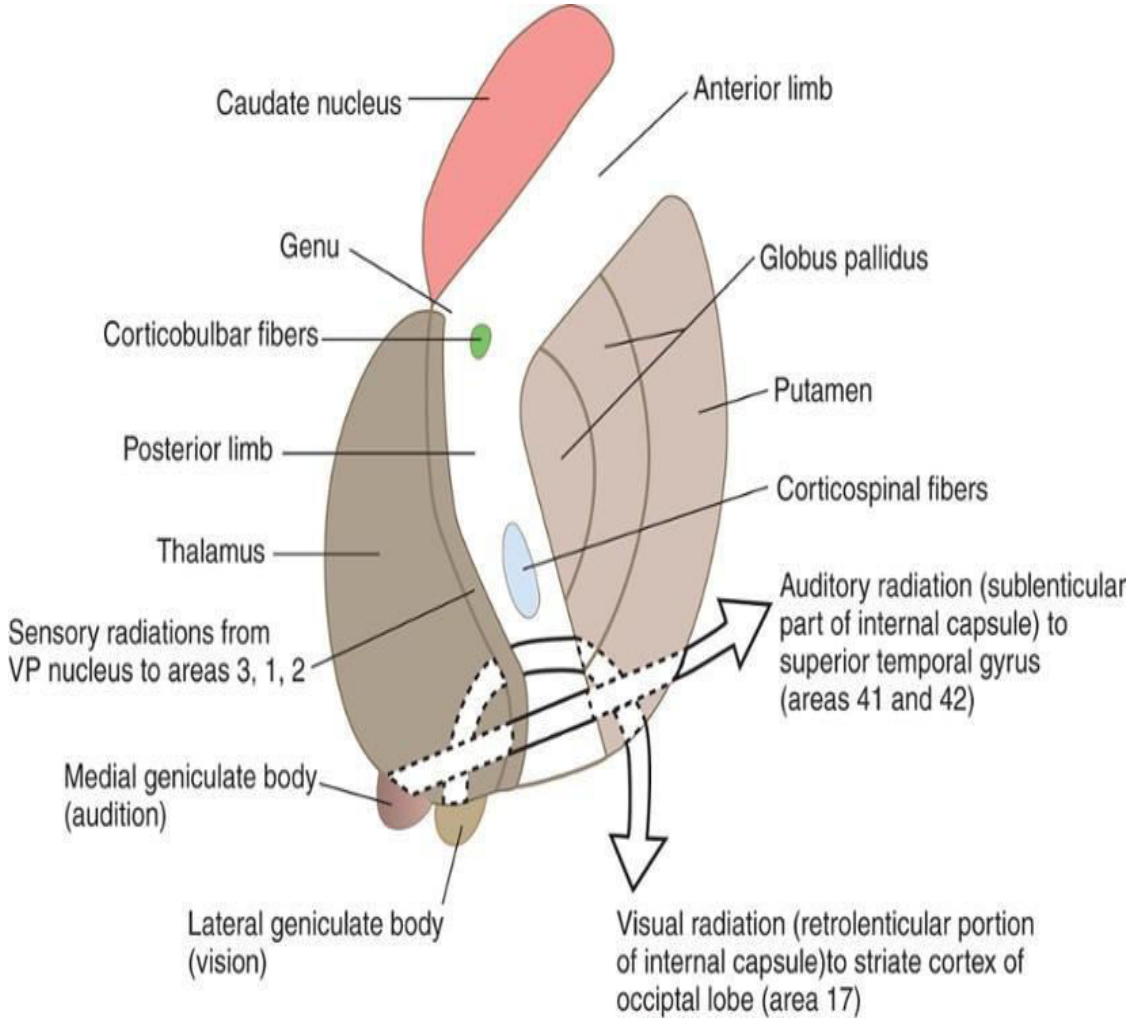
Causes of upper & lower motor neuron lesions



UMNL



Internal Capsule



Hallmarks of motor neuron lesions

As a doctor, you need to examine and diagnose the patient before he goes to the MRI or other investigations.

	UMNL	LMNL
Extent of paralysis	Widespread (affects movements)	Localized (individual muscle or group of muscles are affected)
Site of paralysis	Opposite side to lesion (because of the decussation of fibers, e.g. pyramidal decussation)	Same side of lesion
Tone of muscles	Increased tone(hypertonia) and spasticity clasp-knife spasticity	Decreased tone (hypotonia), flaccidity paralysis
Deep reflexes	Brisk (exaggerated) tendon jerks (tendon reflexes) the reflex when examining with a hammer: very strong	Diminished or absent
Superficial reflexes	Absent, diminished	Absent
Planter reflex	Extensor plantar reflex , positive babinski sign (dorsiflexion of the big toe and fanning out of the other toes), or just an upgoing toe .	Flexor or absent
Muscle waisting	No marked muscle wasting , but minor wasting may occur due to (Di-use atrophy) Weakness most evident in antigravity muscles	Marked muscle wasting (atrophy)
Clonus	Clonus present (rhythmic oscillation on tendon stretch) لما نسوي له فليكشن يصير فيه تذبذبات	No clonus
Fasciculations (seen) - fibrillation potentials by EMG	No fasciculations No fibrillation potential (no denervation potentials in EMG)	Fasciculations may be seen (tapping produce it) & Fibrillation by EMG (denervation potentials)
NCV (nerve conduction velocity)	Normal	Abnormal
Other		Muscle contractures and trophic changes in skin and nails

UMNL & LMNL

	UMNL	LMNL
Cause	Cerebrovascular strokes due to hemorrhage or thrombosis in the post limb of internal capsule ⇒ damage of both pyramidal and extrapyramidal fibers	1- Lesion of the lower motor neurons as in poliomyelitis 2. Damage of motor nerves e.g. DM or alcoholism
	Characters	
1- Paralysis	* On the opposite side of the body (contralateral) * Widespread affecting half of the face, upper &lower limbs * Poor recovery	* On the same side of the lesion * Localized to muscles supplied by the affected segment only * Recovery may occur.

Cont.

	UMNL	LMNL
2- Muscle tone	* Hypertonia of the spastic type in the paralyzed muscle * Klasp knife type: resistance to passive movement then sudden release * Cause: loss of inhibitory effect of the cortical extrapyramidal area & ↑↑ facilitatory impulses on the γ motor neurons ⇒ facilitation of stretch reflex	* Hypotonia or Atonia: Flaccid paralysis (loss of tone in paralyzed muscles) * Cause: interruption of stretch reflex
3- Deep reflexes	* Exaggerated deep reflexes on the affected side: (e.g. knee & ankle jerks). * Clonus is present. * Cause: release of stretch reflex from cerebral inhibition	* Absent deep reflexes in muscles supplied by the affected segments or motor nerves

Cont.

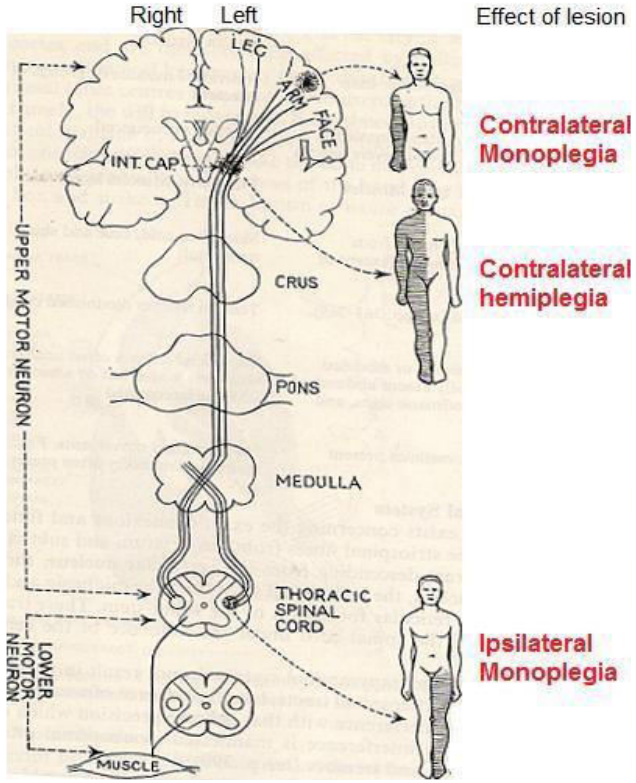
	UMNL	LMNL
4- Superficial reflex	* Lost on the affected side. * Cause: loss of supra-spinal facilitation * Abdominal & ceremasteric reflexes: absent * The planter reflex ⇒ +ve Babiniski's sign.	* Lost on the affected segments only
5- Muscle wasting	* Not significant * Cause: paralyzed muscles are still innervated and can contract reflexly. * Spasticity saves muscle from wasting.	* Marked (disuse atrophy) * Cause: muscles cannot contract neither reflexly nor voluntary
6- Fasiculations	Absent	Present Visible spontaneous contractions of bundles of fibers in the affected ms

ONLY IN FEMALES' SLIDES

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.

The side affected and the extent of paralysis vary according to the site of the lesion	
Side affected	Extent of paralysis
Area 4	Restricted paralysis (ex: Contralateral monoplegia, paralysis of one limb Because area 4 is widespread and is rarely damaged completely)
Corona radiata	This leads to contralateral monoplegia or hemiplegia, depending on the extent of the lesion.
Internal capsule	This often leads to contralateral hemiplegia because almost all fibers are injure
Brain stem	Contralateral hemiplegia & ipsilateral paralysis of the cranial nerves as follows: • Midbrain lesion: the III and IV • Pons lesion: V, VII and VIII. • Medulla lesion: IX, X, XI and XII



Bilateral lesion in brain stem is rare and leads to quadriplegia and bilateral paralysis of the cranial nerves.

Effects of a unilateral lesion in posterior limb of internal capsule

- Such lesion is called cerebral stroke.
- It is usually caused by thrombosis or haemorrhage 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. It is characterized by acute UMNL manifestations in the opposite side:

- Paralysis including the upper and lower limbs, the lower parts of the face and half of the tongue.
- Hemianaethesia (loss of all sensations, due to damage of the thalamocortical fibers).
- Hypotonia and areflexia & loss of the superficial reflexes.
- Babinski's sign may be present.

Manifestations of this stage are similar to those of LMNL, but the extent of paralysis is much more than that of LMNL.

2- Chronic (permanent or spastic) stage

The main manifestations of this stage include:

- Contralateral hemiplegia (paralysis of the opposite half of the body) of the UMNL type (partial recovery occurs after a variable period (possible walking), 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 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
- Diminished hearing power in both ears (by about 50 %), because of damage of auditory radiation.

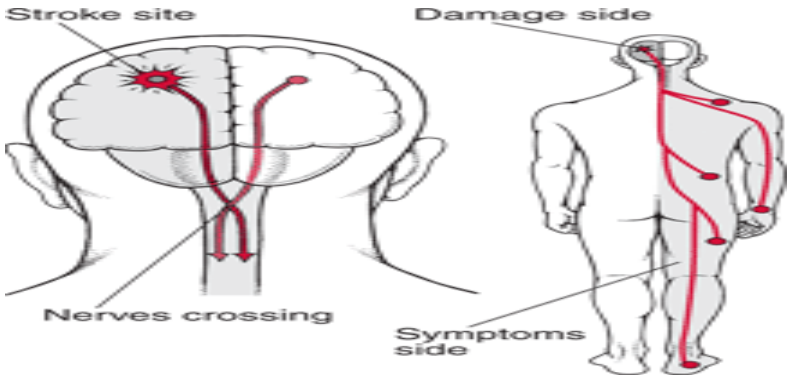
Damage of posterior limb of internal capsule (UMNL)

Damage of posterior limb of internal capsule
Most commonly the posterior limb

Motor loss
(Hemiplegia) contralateral

1- contralateral paralysis

(loss of only voluntary movements) of the distal muscles of the limbs, lower facial muscles and muscles of the tongue.



2- contralateral paresis

(weakness i.e., the muscles retains some movements) of the axial muscles and upper facial muscles.
Axial ms: are supplied by descending motor tracts other than CBS whereas ms of the upper face are ipsilaterally innervated by CBS tract.

Sensory loss
(Loss of somatic sensation, Vision and Hearing)

3- spasticity (increased ms tone)

of the skeletal ms due to increased supraspinal facilitation to γ motor neurons.
A lesion at the level of internal capsule interrupts the descending inhibitory cortical fibers which feeds the inhibitory reticulospinal tract leaving the facilitatory vestibulospinal and reticulospinal to act.
This spasticity is of the clasp-knife type

سكينة لما نفتحها تنفتح على طول، بس لما نسكرها بشكل بسيط ونترك تنفتح بسرعة بنفسها

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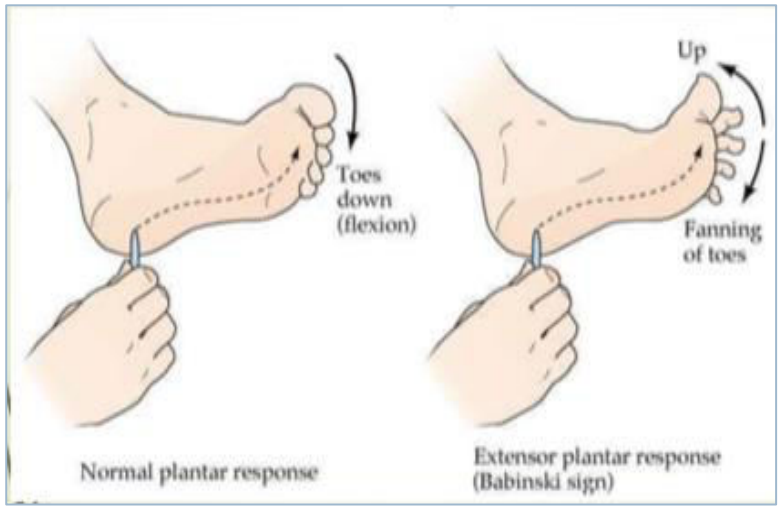
Damage of posterior limb of internal capsule

Motor loss (Hemiplegia)

4- Exaggerated tendon jerk & clonus

due to increased supraspinal facilitation.

5- Positive Babinski's sign



6- Minimal atrophy

The paralyzed ms show no or minimal atrophy as the lower motor neuron is intact and the ms contracts reflexly.

Sensory loss (Loss of somatic sensation, Vision and Hearing)

7- Normal response to electric stimulation

Normal response of the paralyzed ms to electric stimulation:

a) Faradic current produces clonic or tetanic contractions
b) Galvanic current produces contractions that occur only at closing (make) and opening (break) of the circuits.

CCC > ACC > AOC > COC

Cont.

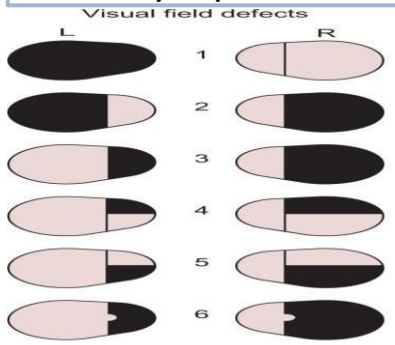
Damage of posterior limb of internal capsule

Motor loss
(Hemiplegia)

Sensory loss
(Loss of somatic sensation, Vision and Hearing)

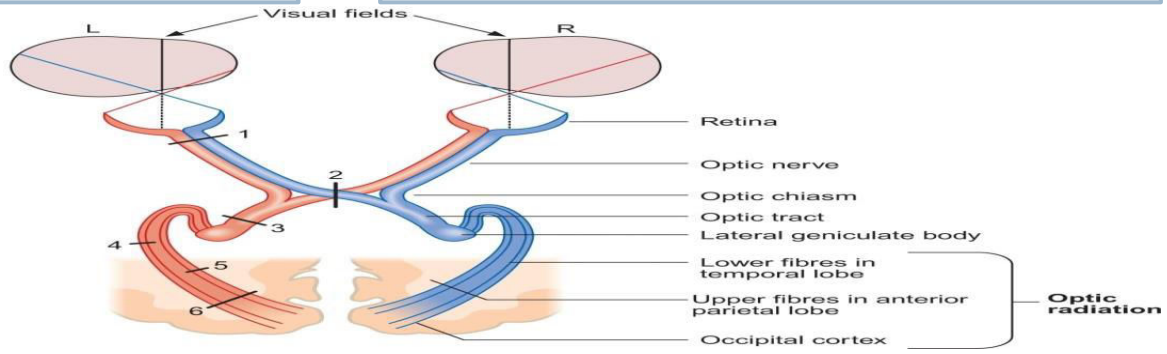
Bilateral diminution

Bilateral diminution of hearing acuity.
No complete loss of hearing as both ears are bilaterally represented in both cortices.



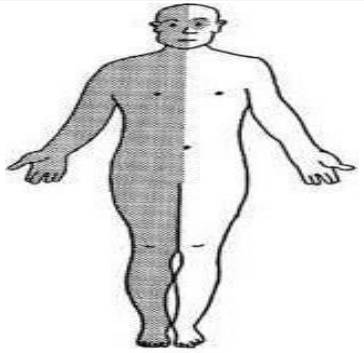
Contralateral homonymous hemianopia

Loss of vision in the two opposite halves of the field of vision.



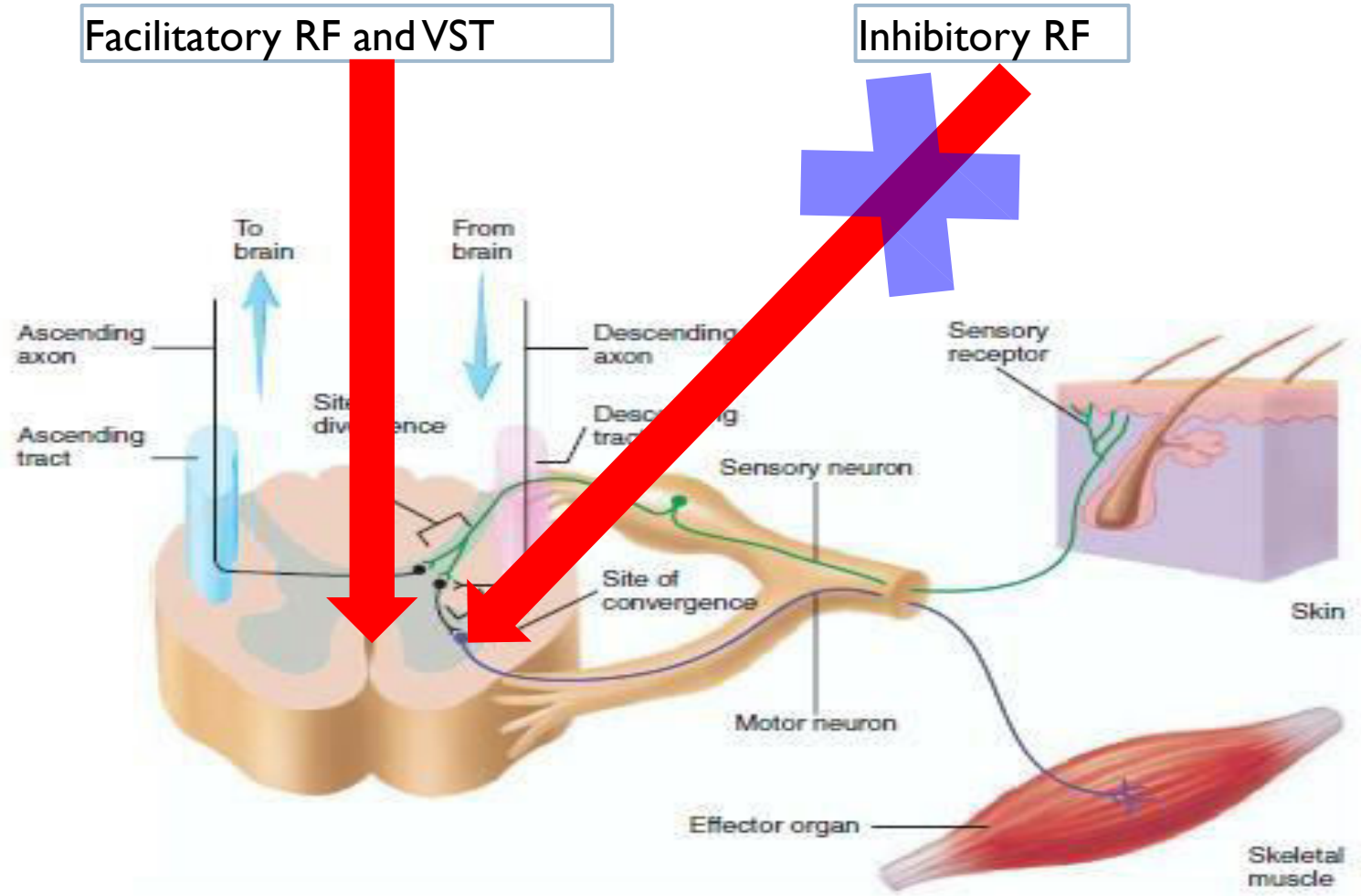
Contralateral hemianaesthesia

Loss of all sensations on the opposite side of the body.



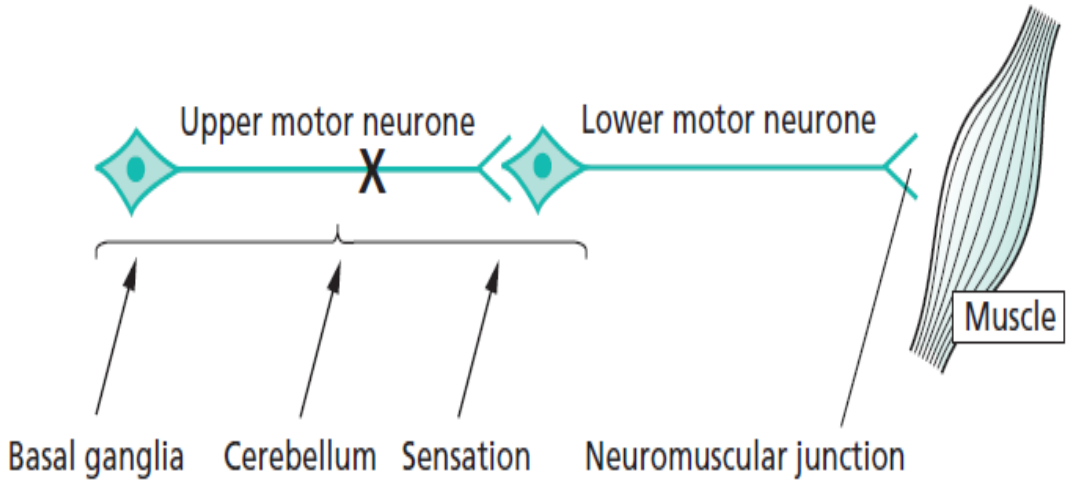
Important: Complex and overlapping function exist between Pyramidal and extra pyramidal systems for example while doing fine work like needle work (Pyramidal system) one has to subconsciously assume a particular posture of arms(extra pyramidal system) that enables to do your work

Spasticity

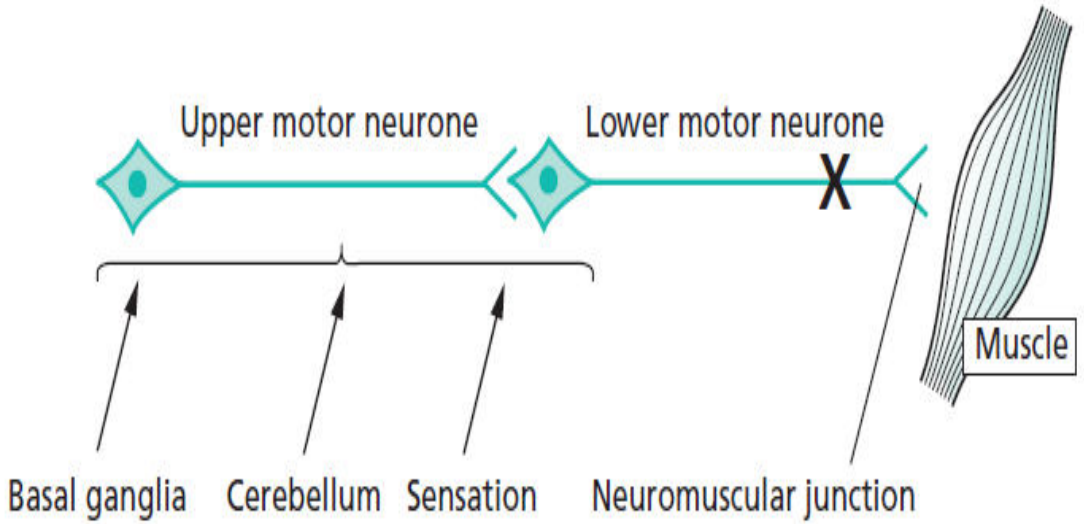

Facilitatory RF and VST
Inhibitory RF
To brain
From brain
Ascending axon
Ascending tract
Descending axon
Sensory receptor
Sensory neuron
Site of convergence
Skin
Motor neuron
Effector organ
Skeletal muscle

Remember

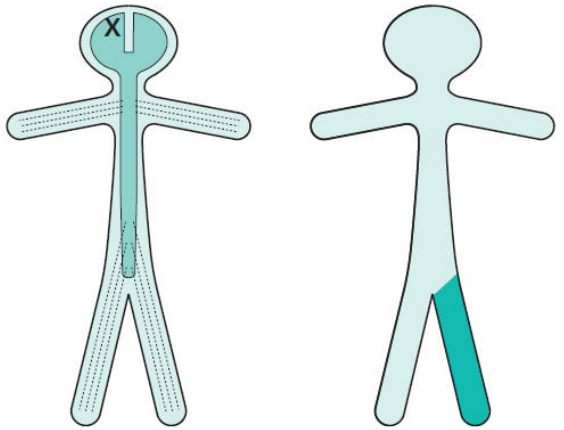
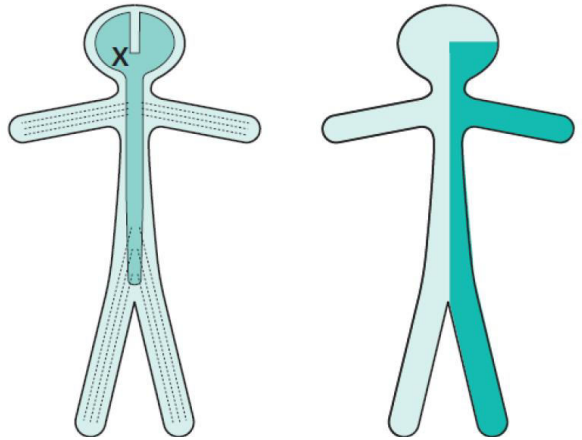
Upper motor neurone



Lower motor neurone



Comparison

Contralateral monoparesis	A lesion situated peripherally in the cerebral hemisphere, i.e. involving part of the motor homunculus only, produces weakness of part of the contralateral side of the body, e.g. the contralateral leg. If the lesion also involves the adjacent sensory homunculus in the postcentral gyrus, there may be some sensory loss in the same part of the body.	
Contralateral hemiparesis	• Lesions situated deep in the cerebral hemisphere, in the region o the internal capsule, are much more likely to produce weakness of the whole of the contralateral side of the body, face, arm and leg Because of the funnelling of fibre pathways in the region of the internal capsule, such lesions commonly produce significan contralateral sensory loss(hemianaesthesia) and visual loss (homonymous hemianopia), in addition to the hemiparesis.	

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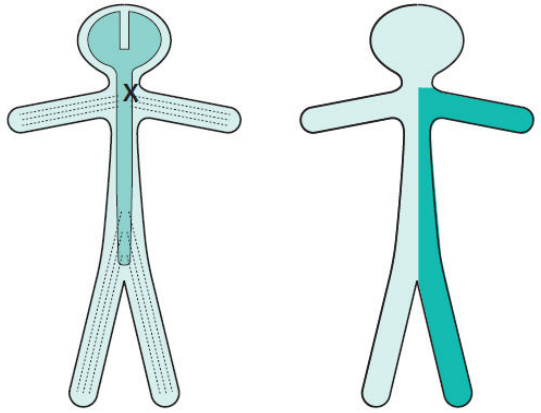
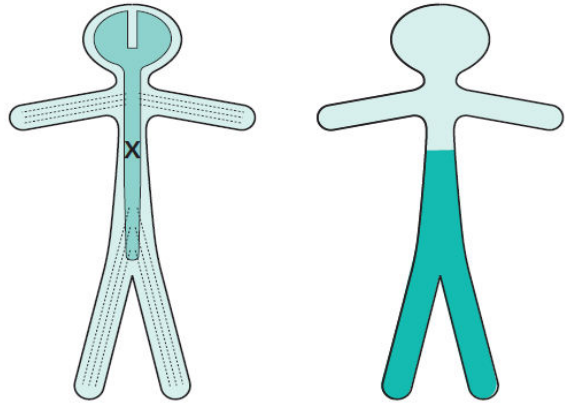
Ipsilateral hemiparesis	A unilateral high cervical cord lesion will produce a hemiparesis similar to that which is caused by a contralateral cerebral hemisphere lesion, except that the face cannot be involved in the hemiparesis, vision will be normal, and the same dissociation of sensory loss (referred to above) may be found below the level of the lesion.	
A spinal cord lesion more usually causes upper motor neurone signs in both legs, often asymmetrically since the pathology rarely affects both sides of the spinal cord equally…		
Paraparesis	Paraparesis, if the lesion is at or below the cervical portion of the spinal cord.	

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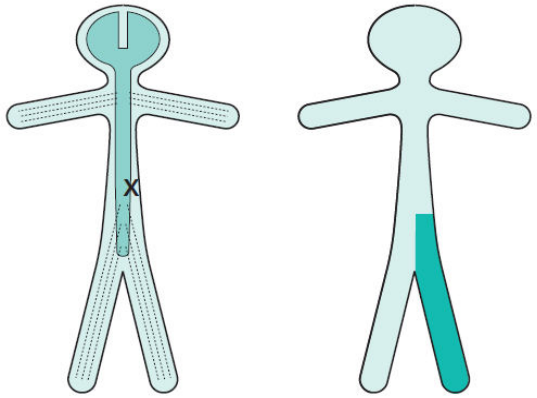
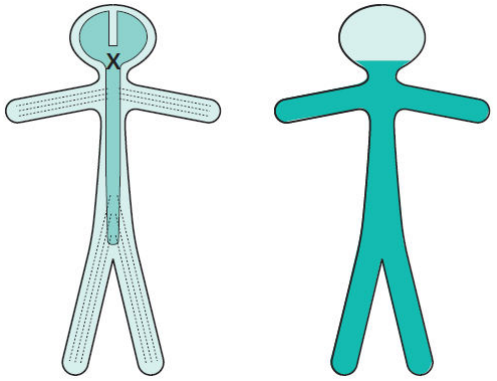
Ipsilateral monoparesis	A unilateral lesion in the spinal cord below the level of the neck produces upper motor neurone weakness in one leg. There maybe posterior column (position sense) sensory loss in the same leg, and spinothalamic (pain and temperature) sensory loss in the contralateral leg. This is known as dissociated sensory loss, and the whole picture is sometimes referred to as the Brown-Séquard syndrome.	
Tetraparesis or quadriparesis	Tetraparesis or quadriparesis, if the lesion is in the upper cervical cord or brainstem.	

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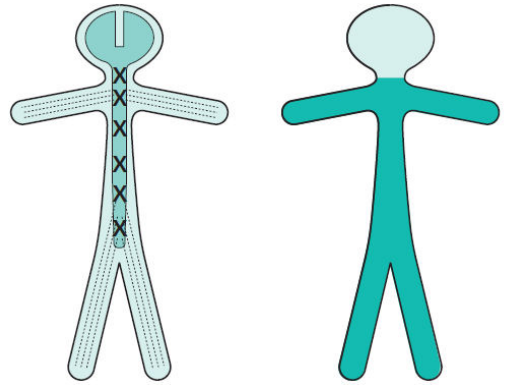
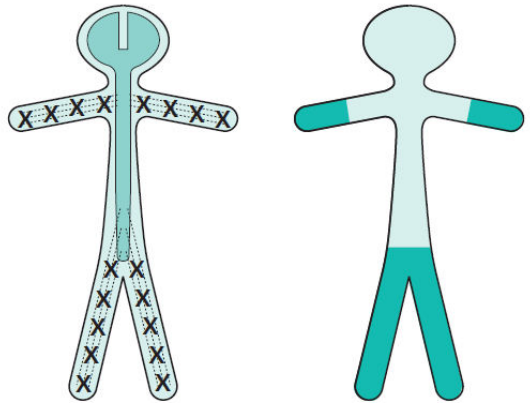
Generalized LMN weakness	Generalized LMN weakness may result from pathology affecting the LMNs throughout the spinal cord and brainstem, as in motor neuron disease or poliomyelitis. Generalized limb weakness (proximal and distal), trunk and bulbar weakness characterize this sort of LMN disorder.	
	Generalized LMN weakness may also result from widespread damage to the axons of the LMNs. This is the nature of peripheral neuropathy (also called polyneuropathy). The axons of the dorsal root sensory neurons are usually simultaneously involved. The LMN weakness and sensory loss tend to be most marked distally in the limbs.	

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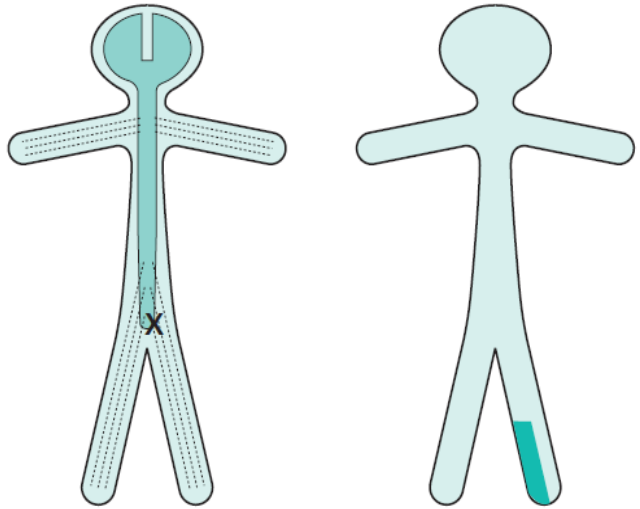
LMN weakness of one spinal root	LMN weakness may be confined to the distribution of one spinal root (above) or one individual peripheral nerve (below). In such circumstances, the LMN signs are found only in the muscles supplied by the particular nerve root or peripheral nerve in question. Almost always there is sensory impairment in the area supplied by the nerve or nerve root. Examples of such lesions are an S1 nerve root syndrome caused by a prolapsed intervertebral disc, or a common peroneal nerve palsy caused by pressure in the region of the neck of the fibula.	

Remember

Motor neuron diseases

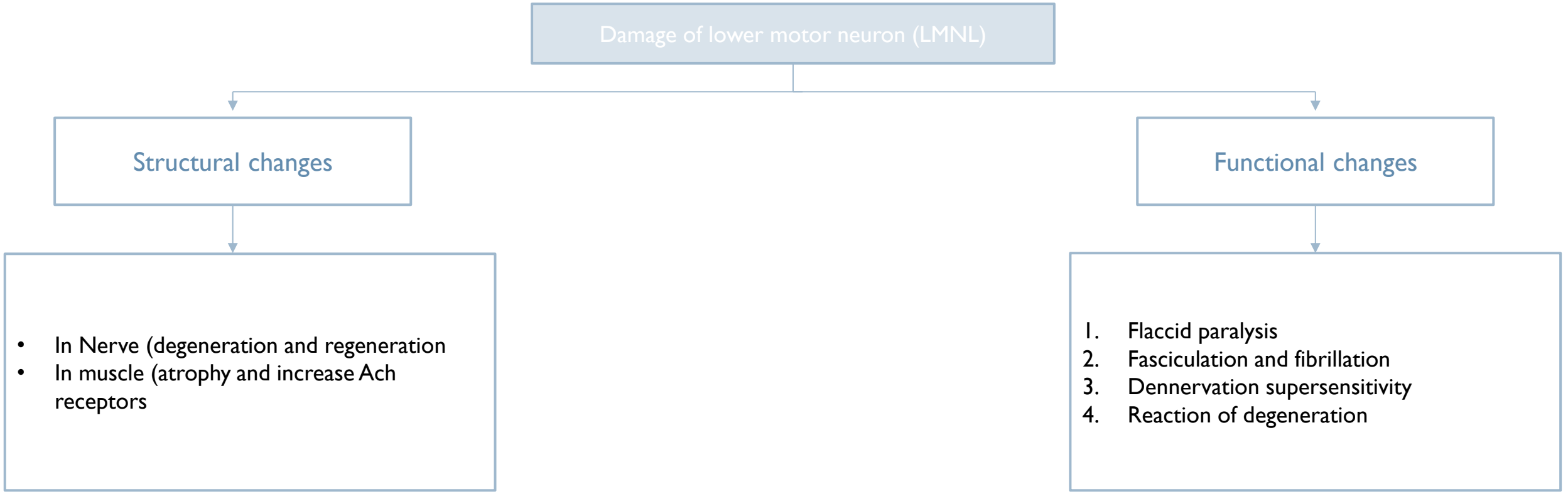
Selectively affect motor neurons, that control voluntary muscle activity

Amyotrophic lateral sclerosis	Primary lateral sclerosis	Progressive muscular atrophy	Bulbar palsy- bulbar	Pseudobulbar palsy-bulbar
UMN + LMN	UMN	LMN	LMN	UMN

Spinal cord

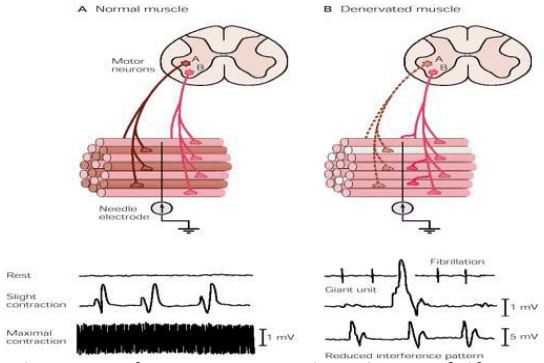
Transverse myelitis	Anterior spinal artery syndrome	Brown-Sequard syndrome
Upper sensory level for all sensations, LMN signs at the level of lesion, flaccid paralysis (spinal shock)UMN signs distally, Bladder/Bowel involved	Upper sensory level for pain/temperature, sparing of posterior columns, UMN signs distally	I/L spastic paralysis & loss of joint/position sense, C/L loss of pain/temperature sensation

Damage of lower motor neuron (LMNL)



Cont. Functional changes

- Flaccid paralysis:
  - Paralysis of denervated ms with loss of all types of movements, "voluntary, postural and reflex".
  - All reflexes are lost including stretch reflex resulting in loss of ms tone and tendon jerk (flaccidity).
  - The extent of paralysis is usually limited to a small group of ms.
- Fasiculations and fibrillations:
  - Appears few days or weeks after denervation.
  - Disappear when the motor nerve completely degenerates or successful re-innervation of the ms occurs.
- Fasiculations:
  - Synchronous visible contraction of the motor unit (all ms fibers) supplied by the injured axon.
  - Result from spontaneous generation of action potential (injury potentials) in distal segment of the injured axon.
- Fibrillations:
  - As degeneration of the injured axon continues, the axon terminals are now separate from the main axon and hence, from each other.
  - Injury potentials are still generated along the terminals leading to asynchronous contraction of the individual ms fibers attached to terminals.
  - Invisible to the observer and detected only by EMG (Electromyogram).



- Denervation supersensitivity:
  - Denervated ms becomes supersensitive to acetylcholine.
  - This is due to increase in the number of A.Ch. receptors which cover the entire surface of ms cell membrane.

Transection of spinal cord

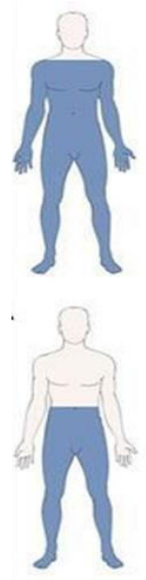
The effects of spinal cord transection are dependent on whether it is

- Complete transection
- Hemisection

- The effects of complete transection of the spinal cord (e.g. tumor or trauma) vary according to the level of transection.
- In the upper cervical region immediate death follows, due to paralysis of all respiratory muscles

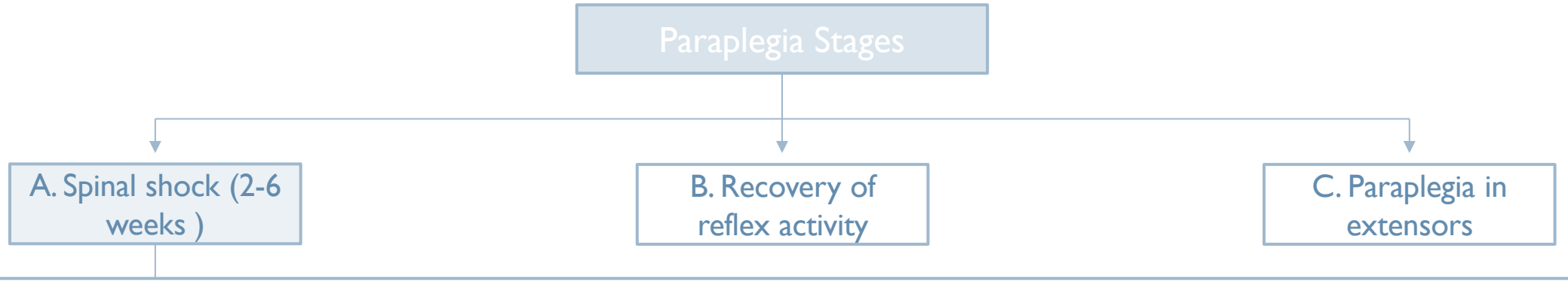
In lower cervical region below C5	Quadriplegia (paralysis in 4 limbs, A)
In the thoracic region	Paraplegia (paralysis in both Lower limbs, b)

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



- **Voluntary movements and sensations are permanently lost.**

Paraplegia Stages



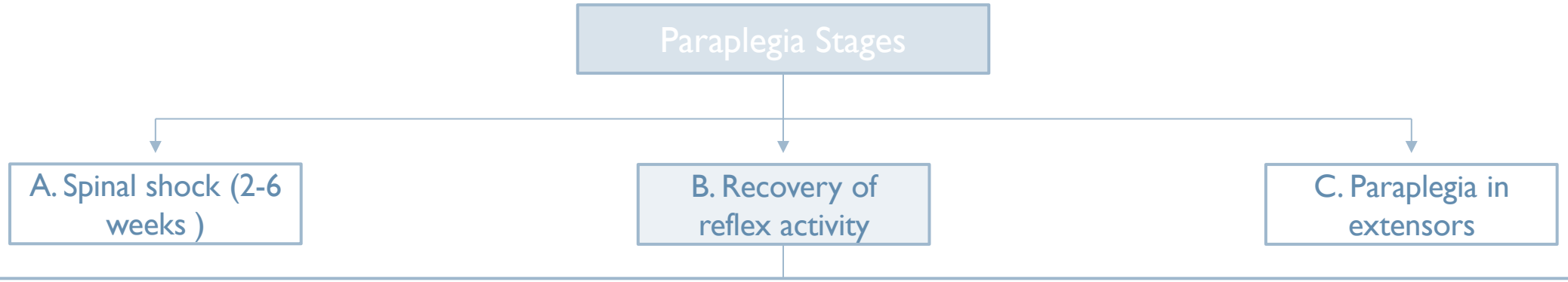
- loss of sensations accompanied by motor paralysis with initial loss but gradual recovery of reflexes Immediately following transection there is:

- Paralysis of all muscles below the lesion.
- Loss of reflexes and loss of tone (flaccidity).
- Loss of all sensations (anaesthesia) and voluntary movements below the level of the lesion due to interruption of all sensory and motor tracts.
- Loss of muscle tone (flaccidity), and vasomotor tone (vasodilation) leading to fall in blood pressure.
- Bladder urinary retention with overflow due to paralysis of the wall of the urinary bladder.

- **Cause of spinal shock**:

sudden withdrawal of supraspinal facilitation on the spinal alpha motor neurons i.e loss of continual tonic discharge transmitted along the excitatory **pontine reticulospinal**, **vestibulospinal** and **corticospinal tracts**.

Cont.



- As the spinal shock ends, spinal reflex activity returns.
- The partial recovery may be due:

- Increased excitability of the spinal cord neurons presumably to compensate for the loss of supraspinal facilitatory influences.
- Disinhibition of motor neurons as a result of absence of inhibitory impulses from higher motor centers.
- Hypersensitivity to excitatory neurotransmitters.

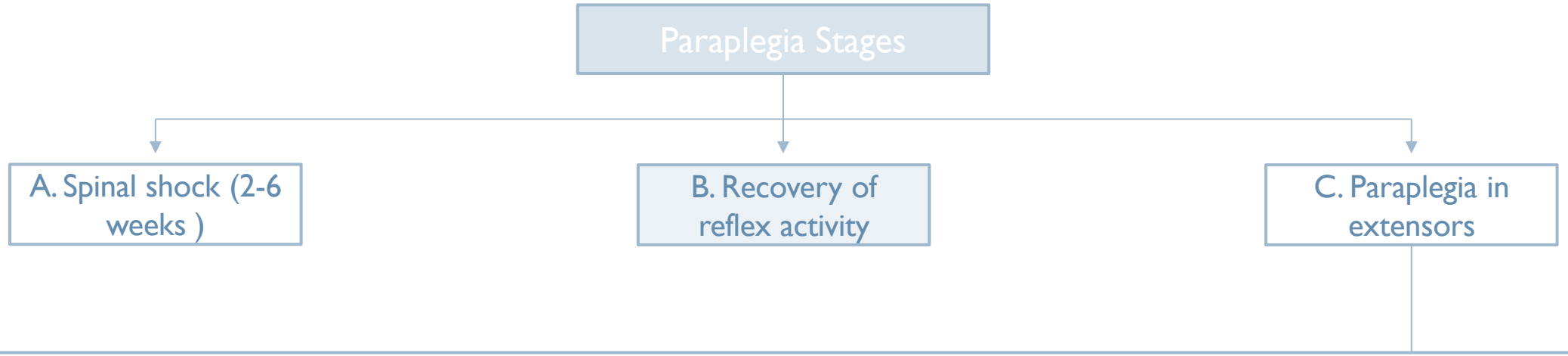
- Features of this stage:

- Gradual rise of arterial blood pressure due to return of spinal vasomotor activity in the lateral horn cells.
- Exaggerated tendon reflexes and spasticity.
- Return of visceral reflexes (micturition & defecation).

- Mass reflex: a minor painful stimulus to the skin of the lower limbs causes:

- o Withdrawal and evoke other autonomic reflexes (bladder and rectum emptying, sweating , blood pressure rise through spread of excitation (by irradiation)
- Voluntary movements and sensations are permanently lost
- Human patients with complete transection never recover fully because effective regeneration never occurs in the human`s CNS.

Cont.



- 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 returns and the patient can stand on his feet with appropriate support.
- Return of the withdrawal reflex and crossed extensor reflex.

Transection of spinal cord

The effects of spinal cord transection are dependent on whether it is

- Complete transection
- Hemisection

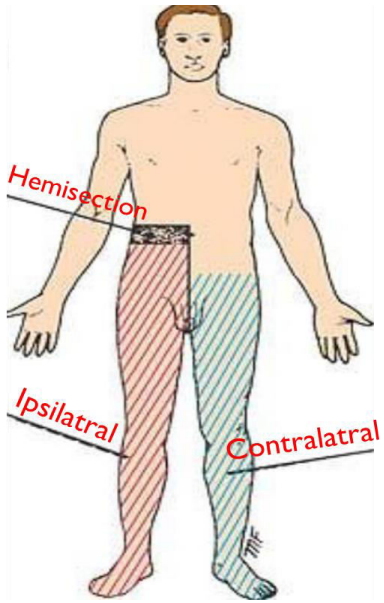
➢ Brown-Séquard syndrome: hemisection of spinal cord (hemiparaplegic syndrome). E.g stab injury, bullet, tumor, car accident.

Ipsilateral Loss:

- Fine touch, vibration, proprioception (**Dorsal Column**)
- Leg ataxia (**Dorsal Spinocerebellar**)
- Spastic paresis below lesion (**Lat. corticospinal**)
- Flaccid paralysis (Vent. horn destruction)
- Dermatomal anesthesia (Dorsal horn destruction)

Contralateral Loss:

- Loss of pain and temp (lat. spinothalamic)
- Loss of crude touch and Pressure (Vent. spinothalamic)
- Minor contralat. muscle Weakness (Vent. corticospinal)
- Leg ataxia (Vent. Spinocerebellar)



Cont.

- Ipsilateral lower motor neuron paralysis in the segment of the lesion and muscular atrophy. These signs are caused by damage to the neurons on the anterior gray column and possibly by damage to the nerve roots of the same segment.

- Ipsilateral spastic paralysis below the level of the lesion.
  - An ipsilateral Babinski sign is present, and depending on the segment of the cord damaged, an ipsilateral loss of the superficial abdominal reflexes and cremasteric reflex occurs.
  - All these signs are due to loss of the corticospinal tracts on the side of the lesion. Spastic paralysis is produced by interruption of the descending tracts other than the corticospinal tracts.

- Ipsilateral band of cutaneous anesthesia in the segment of the lesion. This results from the destruction of the posterior root and its entrance into the spinal cord at the level of the lesion.

- Ipsilateral loss of tactile discrimination and of vibratory and proprioceptive sensations below the level of the lesion. These signs are caused b destruction of the ascending tracts in the posterior white column on the same side of the lesion.

- Contralateral loss of pain and temperature sensations below the level of the lesion. This is due to destruction of the crossed lateral spinothalamic tracts on the same side of the lesion.
  - Because the tracts cross obliquely, the sensory loss occurs two or three segments below the lesion distally.

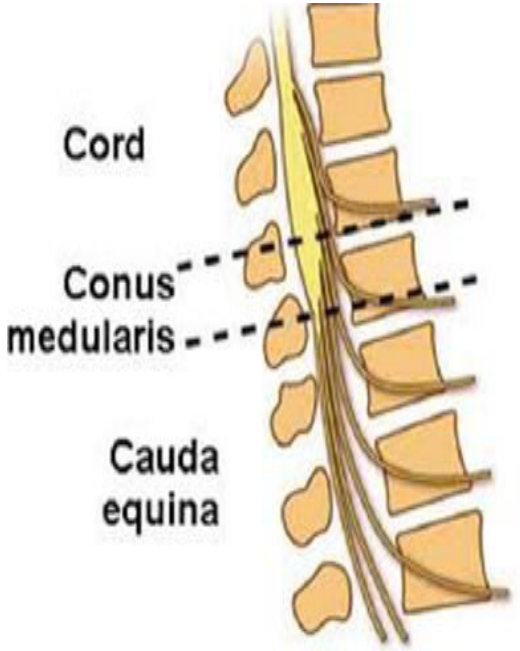
- Contralateral but not complete loss of tactile sensation below the level of the lesion. This condition is brought about by destruction of the crossed anterior spinothalamic tracts on the side of the lesion. Here, again, because the tracts cross obliquely, the sensory impairment occurs two or three segments below the level of the lesion distally. The contralateral loss of tactile sense is incomplete because discriminative touch traveling in the ascending tracts in the contralateral posterior white column remains intact.

Comparison between bulbar and pseudobulbar palsy

Bulbar palsy	Psedobulbar palsy
B/L LMN defect of IX-XII cranial nerves	**B/L UMN defect** of IX-XII cranial nerves
Dysphagia (liquid>solid), nasal regurgitation, slurred speech	Dysphagia, dysarthria, emotional lability
Nasal speech, wasted tongue with fasciculation, absent gag reflex	Slow indistinct speech, spastic tongue, brisk jaw jerk Frontal release signs

Cauda equina and conus medullaris lesions

Conus medullaris	cauda equina
B/L saddle anaesthesia	asymmetric leg weakness and sensory loss
Prominent bowel, bladder symptoms, impotence	Relative sparing of bowelbladder function
Bulbocavernous (S2-s4) and anal reflexes (s4-s5) are absent	Variable areflexia in lower extremities
Muscle strength largely preserved	Low back and radicular pain



Extramedullary and intramedullary syndromes

Extramedullary lesions	Intramedullary lesion
• radicular pain is often prominent • there is early sacral sensory loss (lateral spinothalamic tract) • spastic weakness in the legs (corticospinal tract) due to the superficial location of leg fibers in the corticospinal tract • Early UMN signs	• Tend to produce poorly localized burning pain rather than radicular pain • Spare sensation in the perineal and sacral areas ("sacral sparing"), reflecting the laminated configuration of the spinothalamic tract with sacral fibers outermost; corticospinal tract signs appear later. • Late UMN signs

Thank you!

اعمل لترسم بسمة، اعمل لتمسح دمعة، اعمل و أنت تعلم أن الله لا يضيع أجر من أحسن عملا.

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QUIZ

اقتراحات وشكاوي

References:

- Females' and Males' slides.
- Guyton and Hall Textbook of Medical Physiology (Thirteenth Edition.)