بسم الله الرحمن الرحيم

(و ما أوتيتم من العلم إلا قليلا)

صدق الله العظيم
الاسراء اية 58

UPPER & LOWER MOTOR NEURON lesion

By

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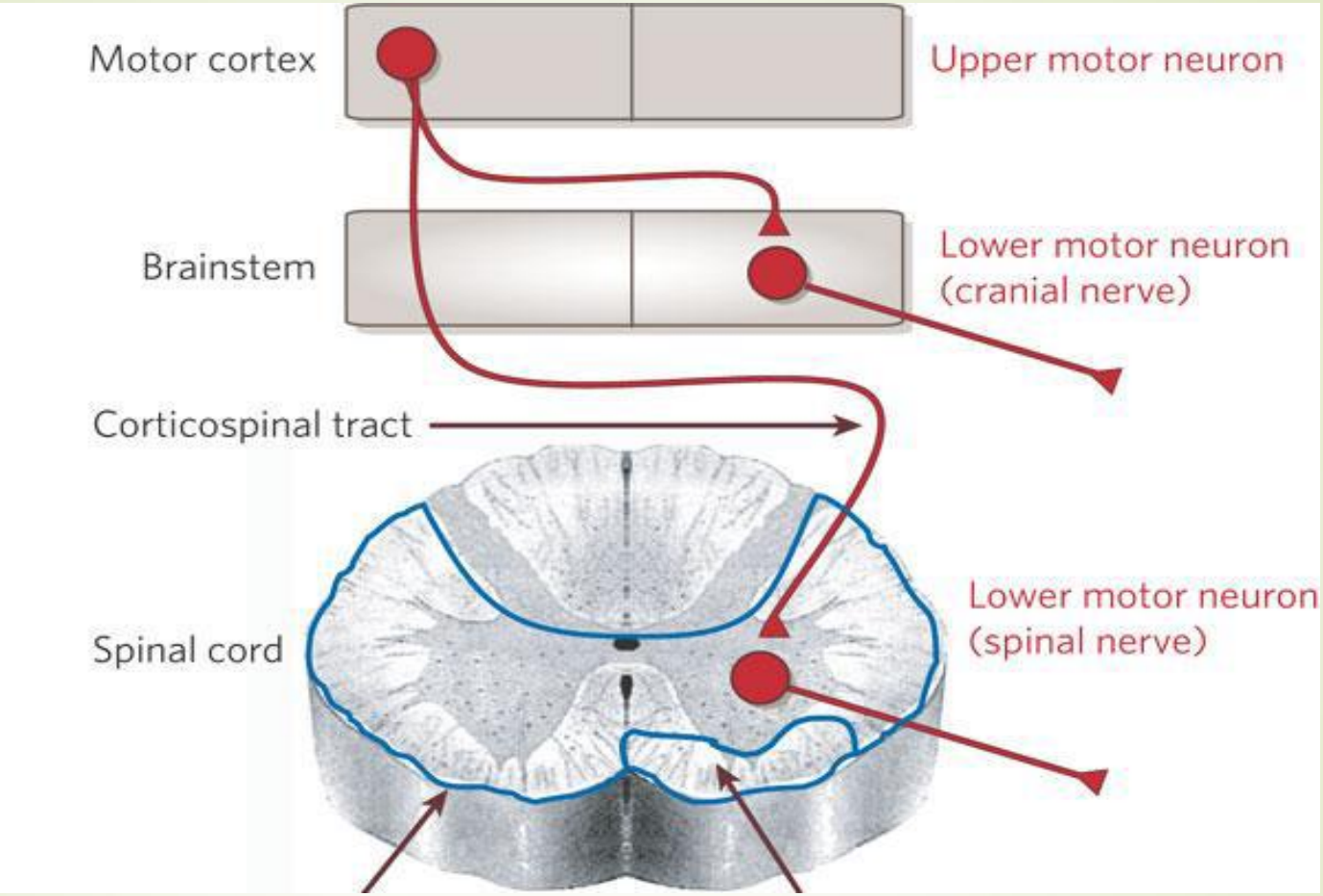
Objectives

- 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.



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

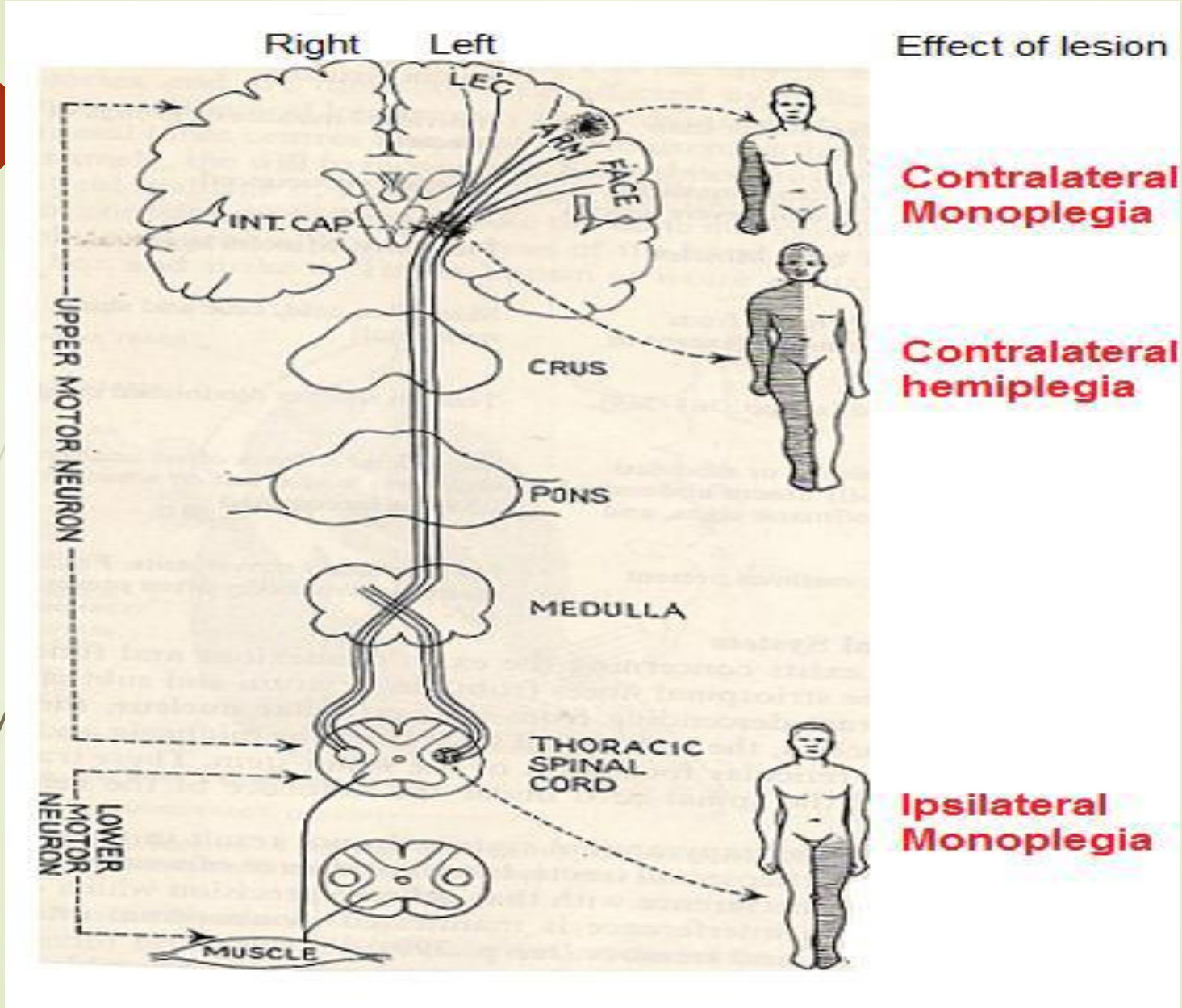
(e.g. nerve injury by trauma or compressive lesion)

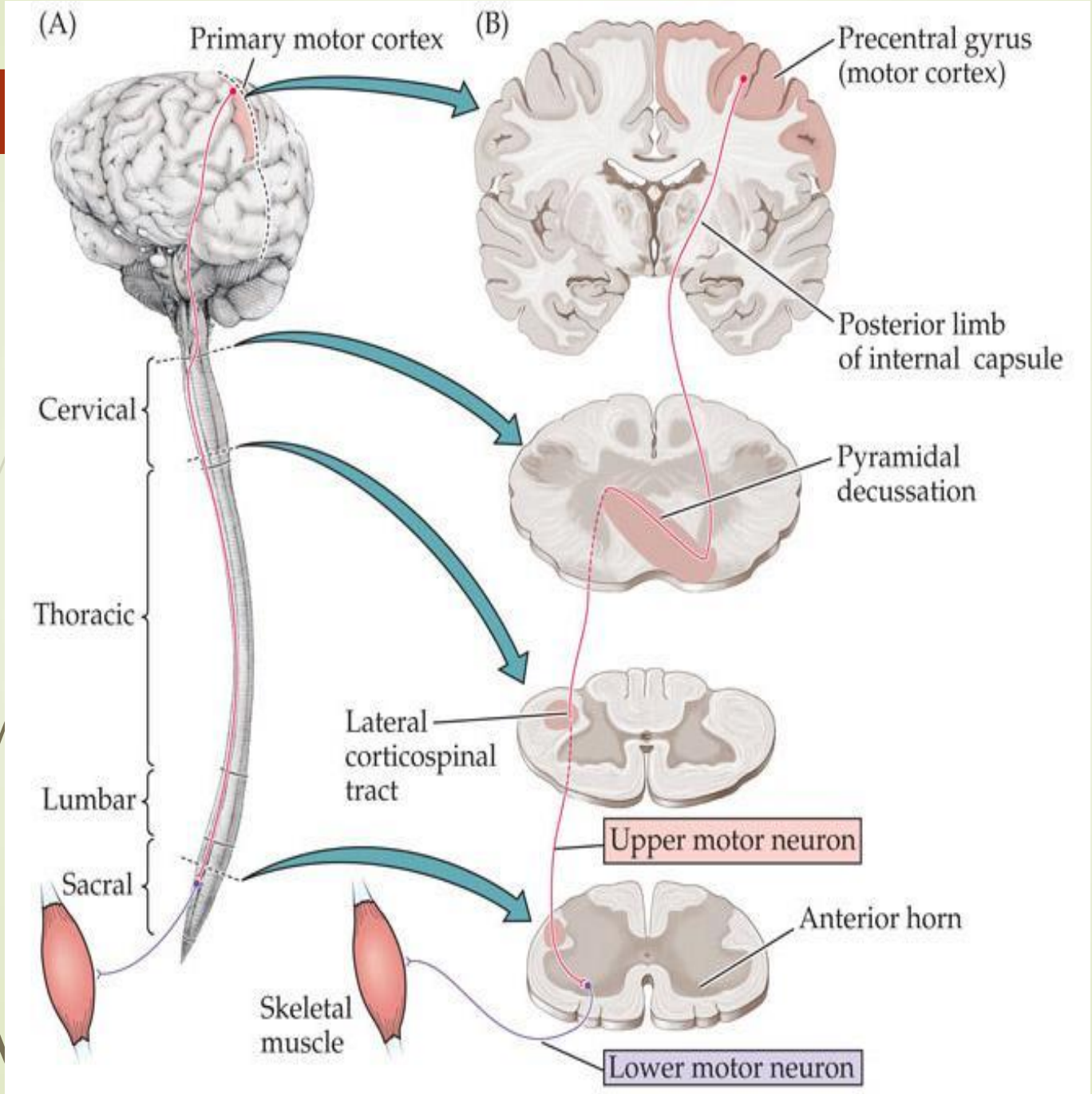
	UMNL	LMNL
1-extent of paralysis	widwspread	localized
2-site of paralysis	Opposite side to lesion	Same side of lesion
3-Tone of muscles	Spasticity (hypertonia) " clasp-knife spasticity	Hypotonia " flaccid paralysis
4- Deep reflexes	Brisk (exaggerated) tendon jerks	Diminished or absent
5- Superficial reflexes	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 minor 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

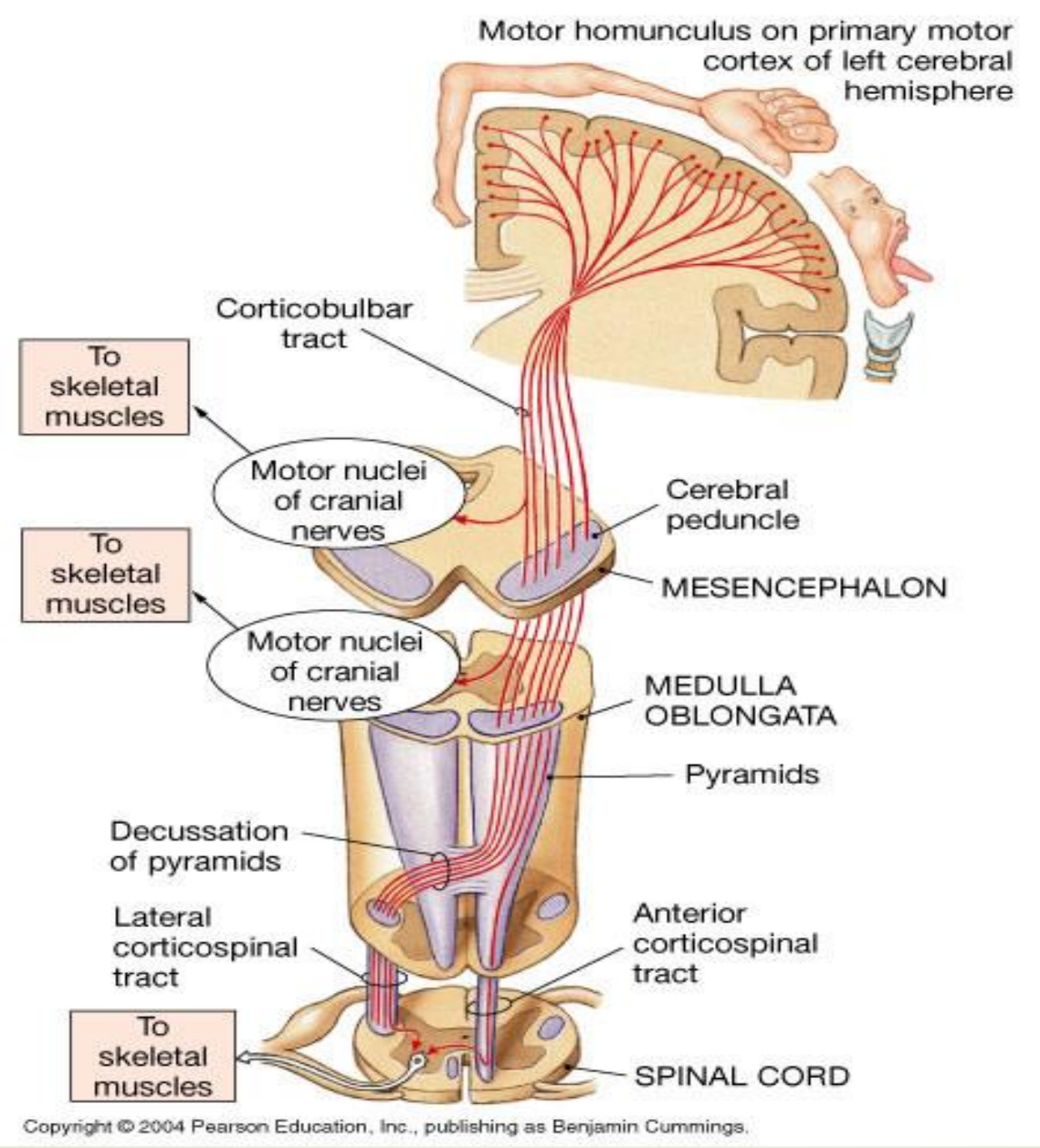
The effect of a lesion in different parts of the motor system

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. contralateral monoplegia (paralysis of one limb because area 4 is widespread so it is rarely damaged completely.
- **2- In the corona radiata:**
- This leads to contralateral monoplegia or hemiplegia, depending on the extent of the lesion.
- **3- In the internal capsule:**
- This often leads to contralateral hemiplegia because almost all fibers are injured
-


Right
Left
Effect of lesion
LEG
ARM
FACE
INT. CAP
CRUS
PONS
MEDULLA
THORACIC SPINAL CORD
UPPER MOTOR NEURON
LOWER MOTOR NEURON
MUSCLE
Contralateral Monoplegia
Contralateral hemiplegia
Ipsilateral Monoplegia


(A)
Primary motor cortex
(B)
Precentral gyrus
(motor cortex)
Posterior limb
of internal capsule
Cervical
Thoracic
Lumbar
Sacral
Pyramidal
decussation
Lateral
corticospinal
tract
Upper motor neuron
Anterior horn
Skeletal
muscle
Lower motor neuron


Motor homunculus on primary motor cortex of left cerebral hemisphere
Corticobulbar tract
To skeletal muscles
Motor nuclei of cranial nerves
Cerebral peduncle
To skeletal muscles
MESENCEPHALON
Motor nuclei of cranial nerves
MEDULLA OBLONGATA
Pyramids
Decussation of pyramids
Lateral corticospinal tract
Anterior corticospinal tract
To skeletal muscles
SPINAL CORD
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Extrapyramidal Tracts

Extrapyramidal Tracts

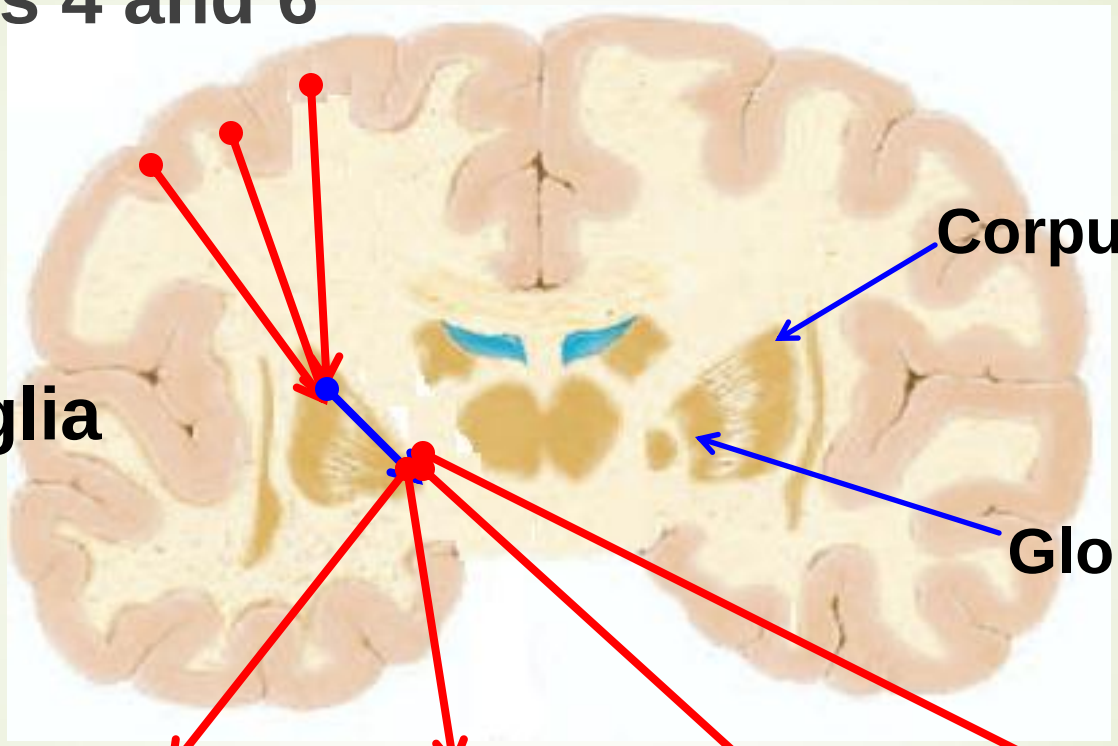
- **Origin:**
- From **area (6) and area (4)** → descends to **corpus striatum** → **Globus pallidus**→ from the globus pallidus fibers pass to;

1. **Reticular formation**
2. **Vestibular nuclei**
3. **Red nucleus**
4. **Tectum** of midbrain.

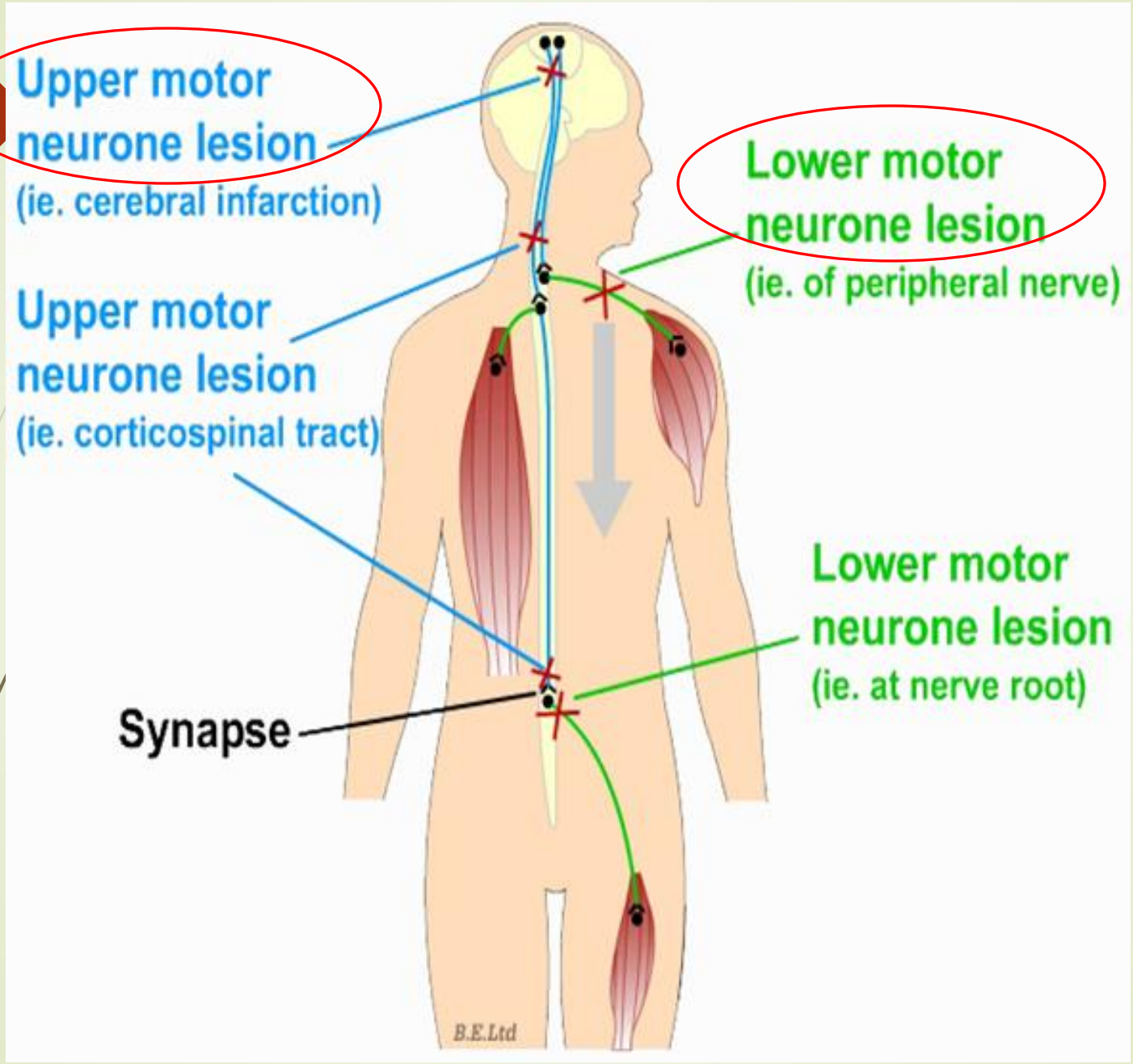
- From these nuclei the extrapyramidal tracts arise

Extrapyramidal Tracts

Motor areas 4 and 6



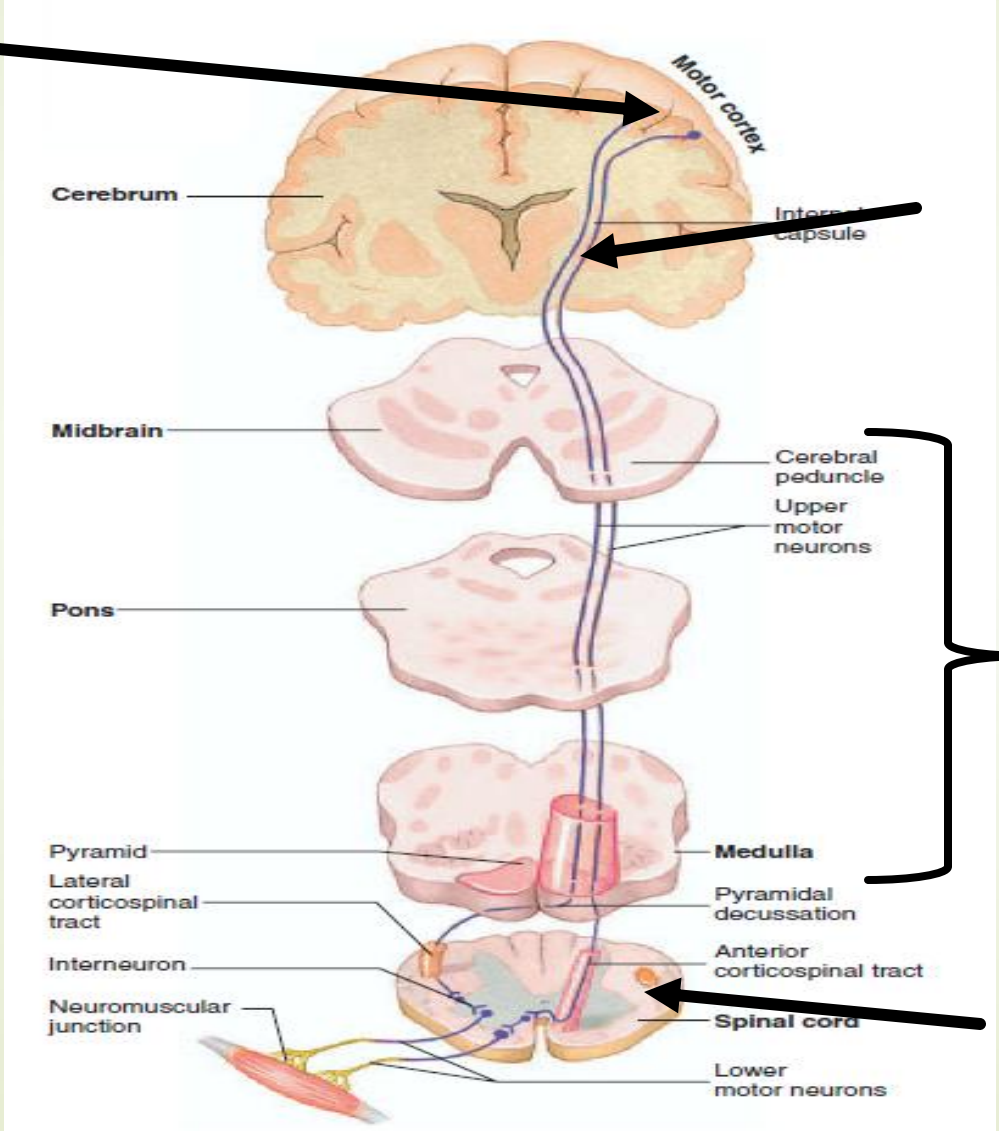
Upper and Lower Motor Neuron Lesion (UMNL and LMNL)


Upper motor neurone lesion
(ie. cerebral infarction)
Upper motor neurone lesion
(ie. corticospinal tract)
Synapse
Lower motor neurone lesion
(ie. of peripheral nerve)
Lower motor neurone lesion
(ie. at nerve root)
B.E.Ltd

Upper Motor Neuron Lesion (UMNL)

Upper Motor Neuron Lesion

MOTOR CORTEX



INTERNAL CAPSULE

BRAIN STEM

SPINAL CORD

Upper Motor Neuron Lesion

Def.,

- It is the damage of **upper motor neuron** in the higher center or the **descending motor tract**.

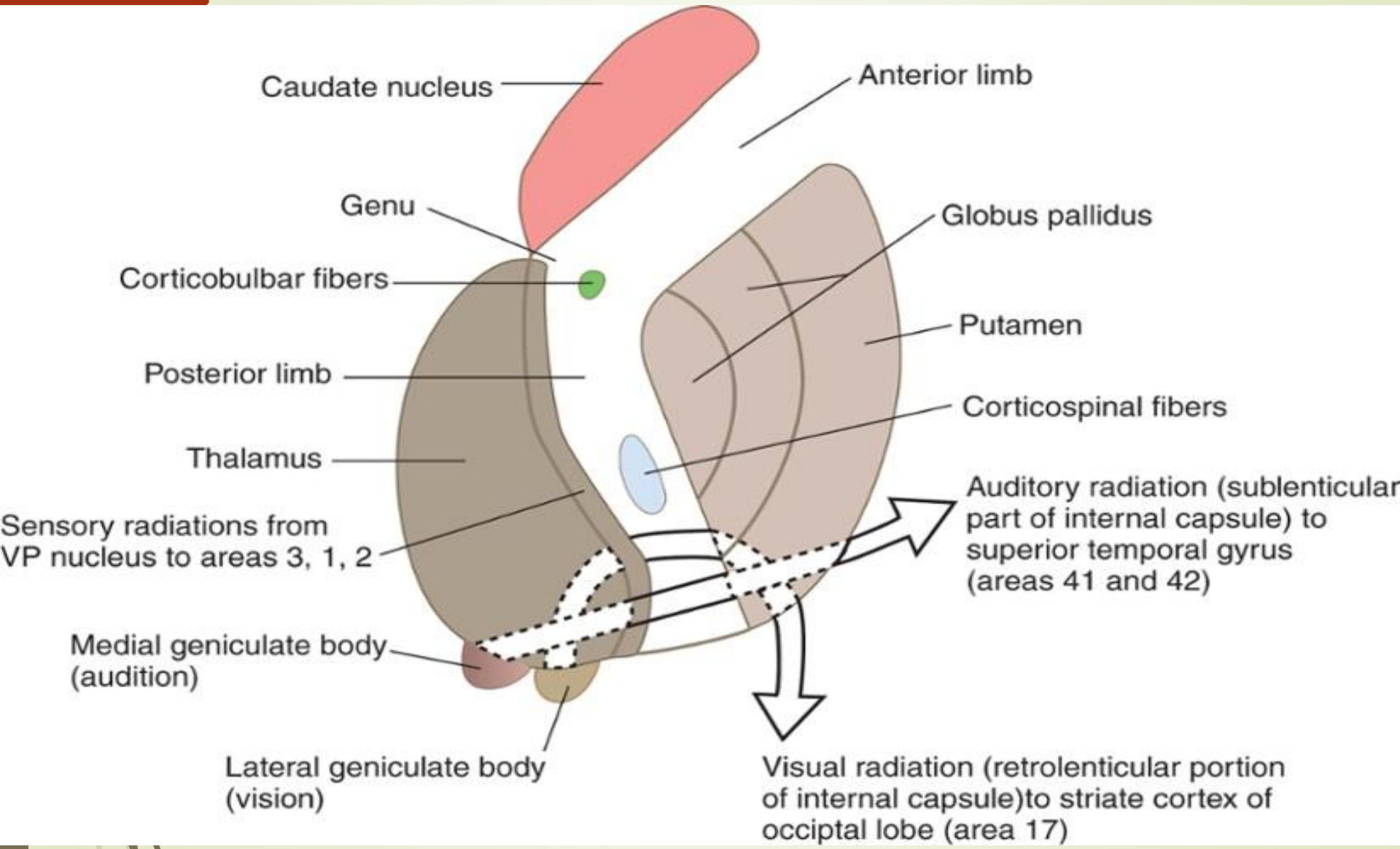
Causes

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

Sites:

- **Most common** site of UMNL is the **internal capsule**.

Internal Capsule



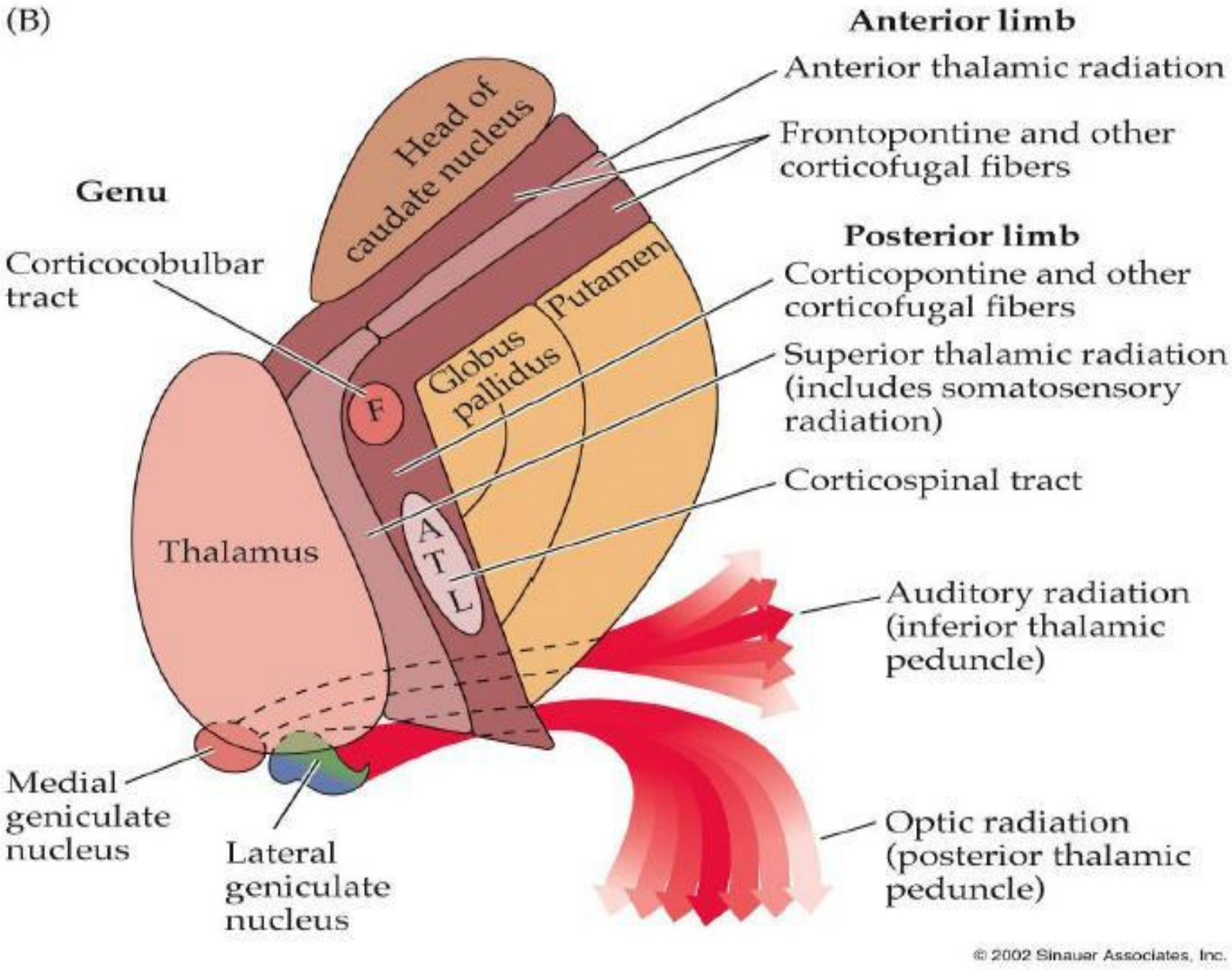
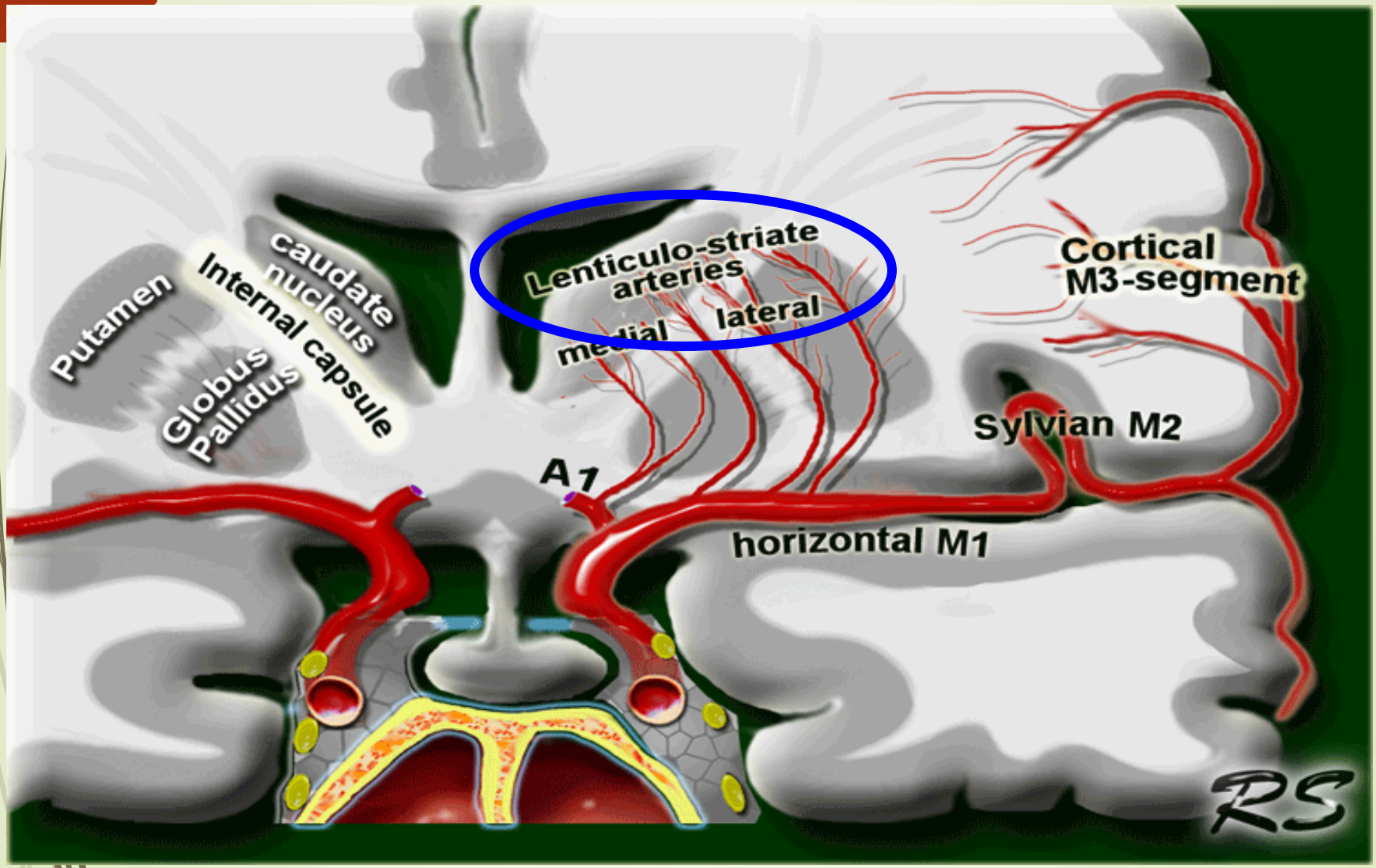
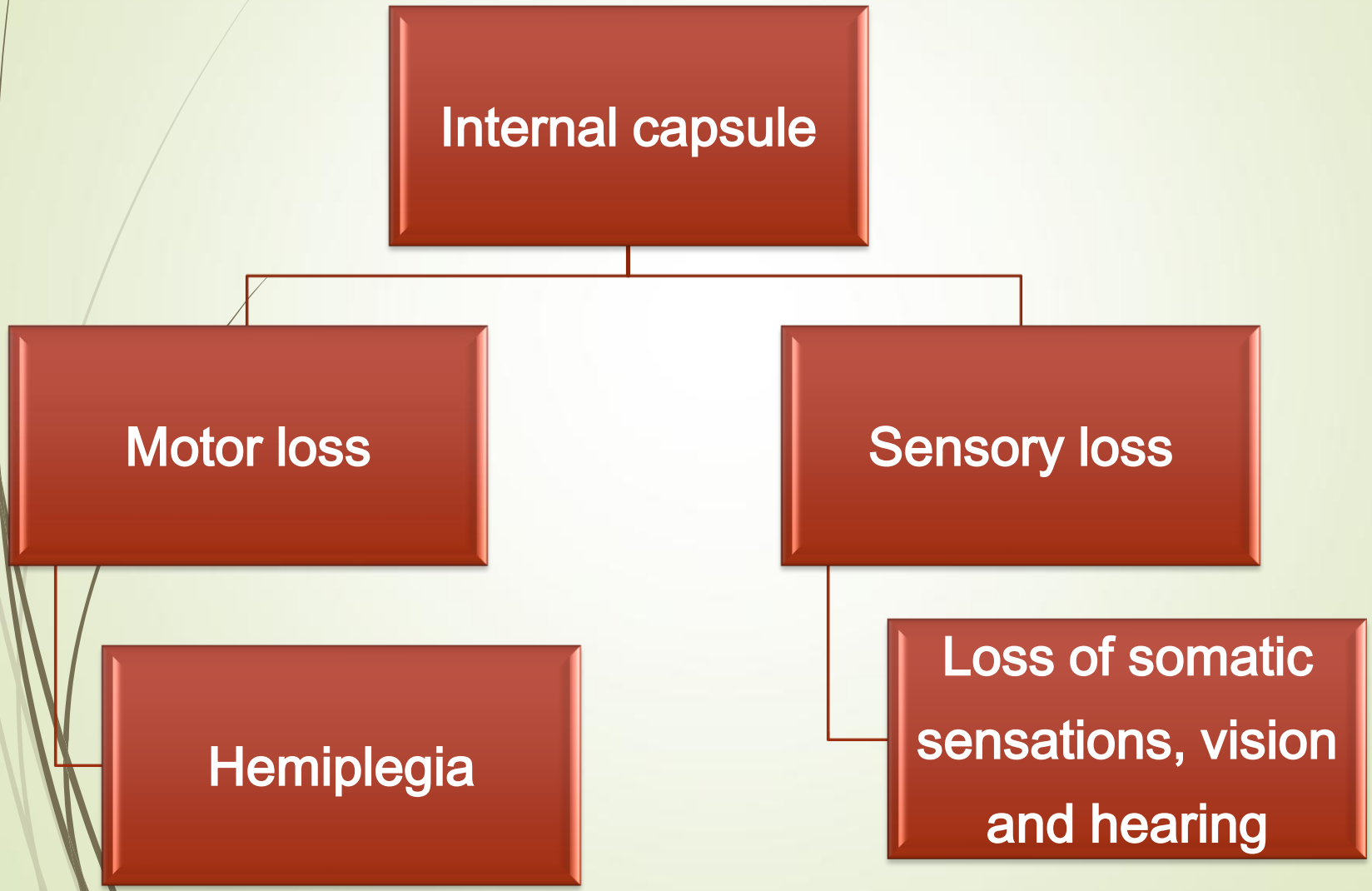


Figure showing Corticospinal & Corticobulbar Fibers in Internal capsule

Thrombosis of Lenticulostriate artery



Damage of Posterior Limb of Internal Capsule



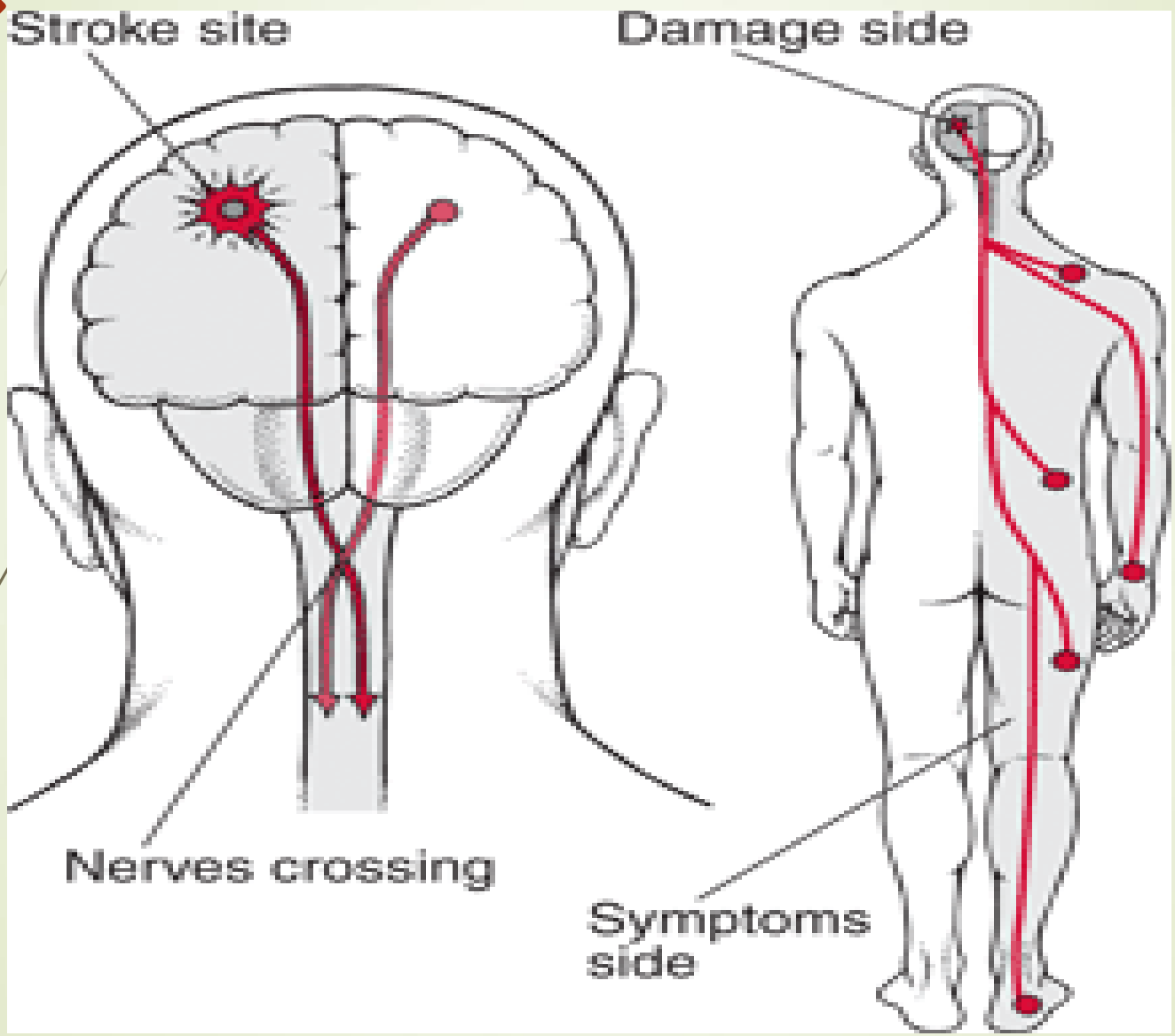
Motor Loss (Hemiplegia)

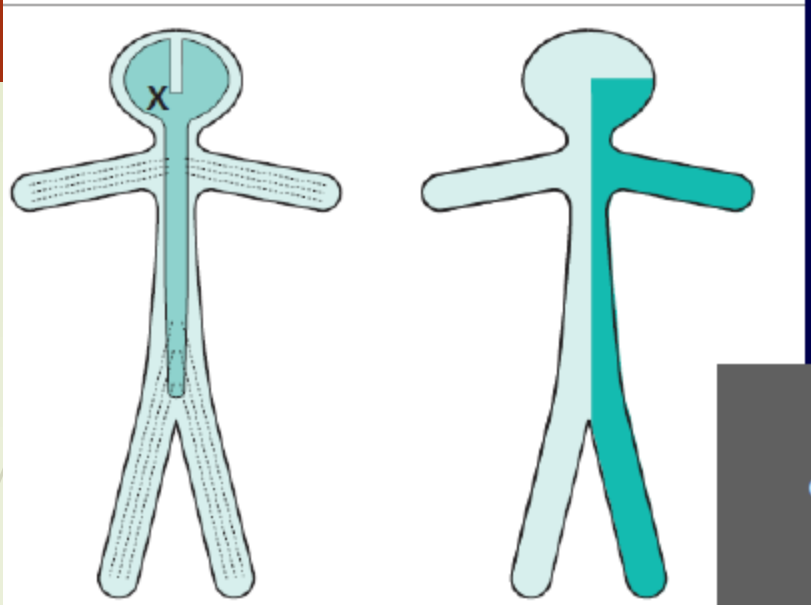
1. **Contralateral paralysis** (loss of only voluntary movements) of the **distal ms** of the limbs, lower facial ms and ms of the tongue.

2. **Contralateral paresis** (weakness i.e., the ms retains some movements) of the axial ms and upper facial ms.

- **Axial ms** are supplied by **descending motor tracts other than CBS** whereas ms of the **upper face are ipsilaterally** innervated by CBS tract.

Motor Loss (Hemiplegia or UMNL)

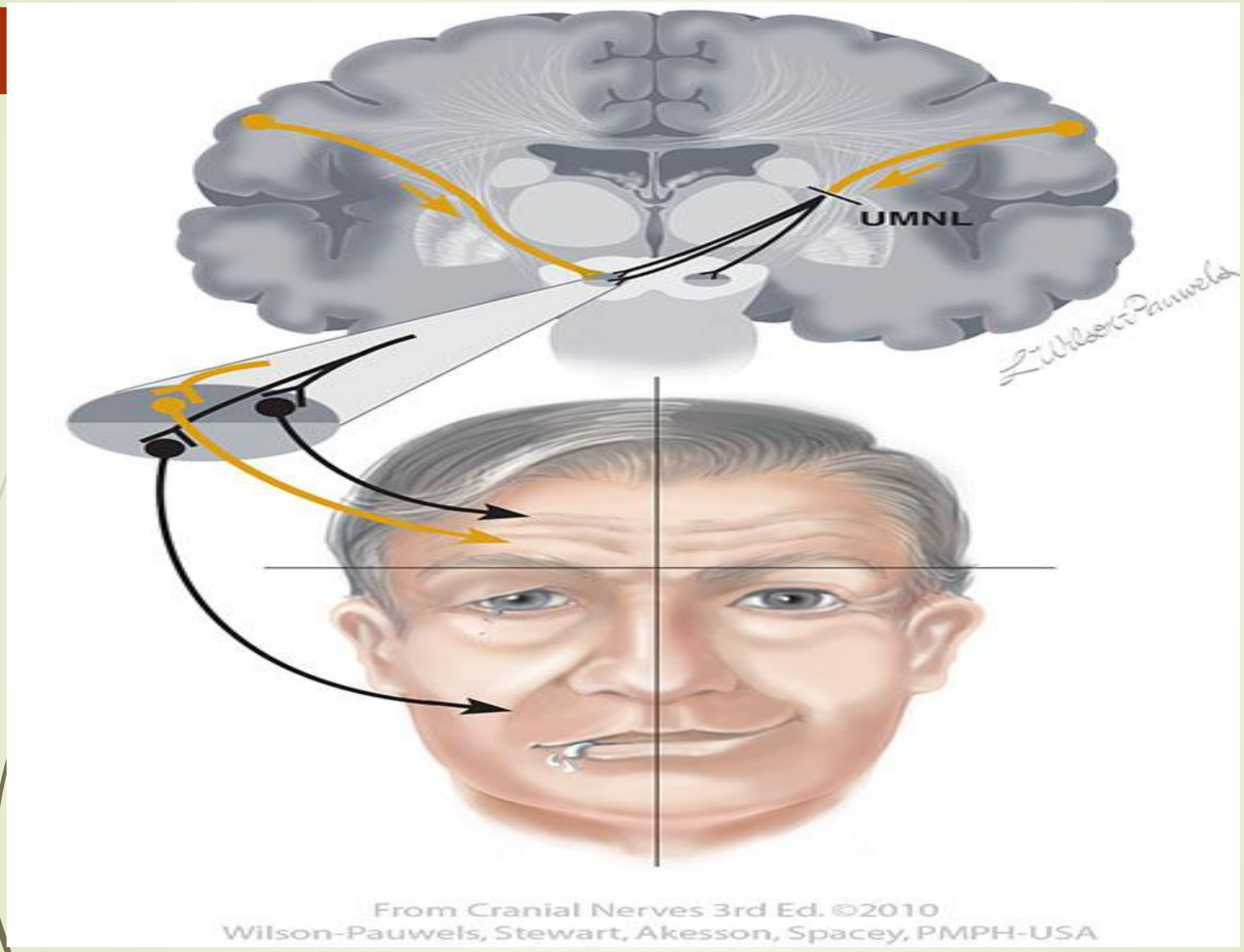




Contralateral Hemiparesis

Contralateral hemiparesis

Lesions situated deep in the cerebral hemisphere, in the region of 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 significant contralateral sensory loss (hemianaesthesia) and visual loss (homonymous hemianopia), in addition to the hemiparesis.


UMNL
From Cranial Nerves 3rd Ed. ©2010
Wilson-Pauwels, Stewart, Akesson, Spacey, PMPH-USA

Motor Loss (Hemiplegia)

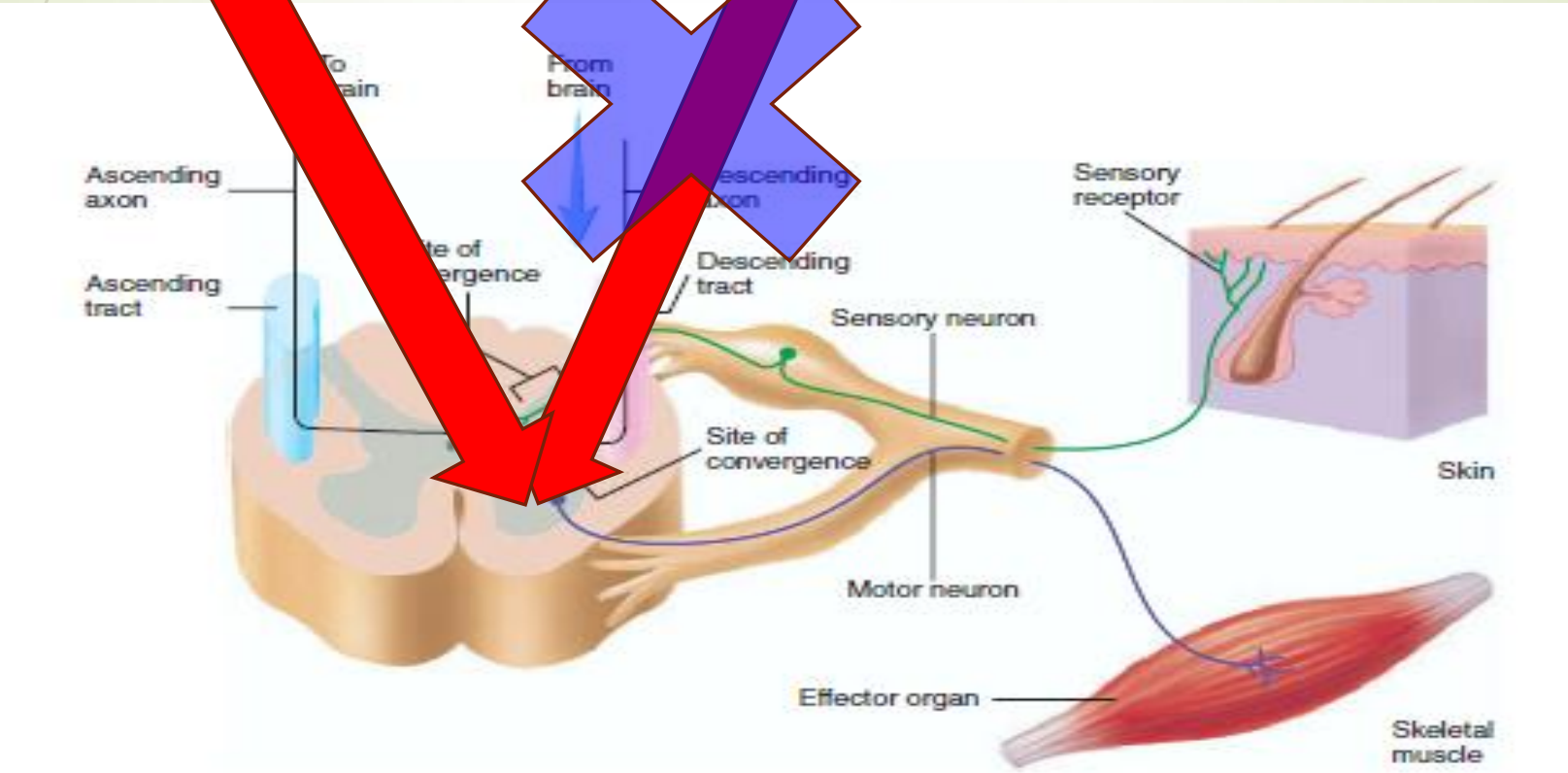
3) **Spasticity (increased ms tone)** of the skeletal ms due to increased **supraspinal facilitation** to γ-motor neurons.

- 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**

Spasticity

Facilitatory RF and VST

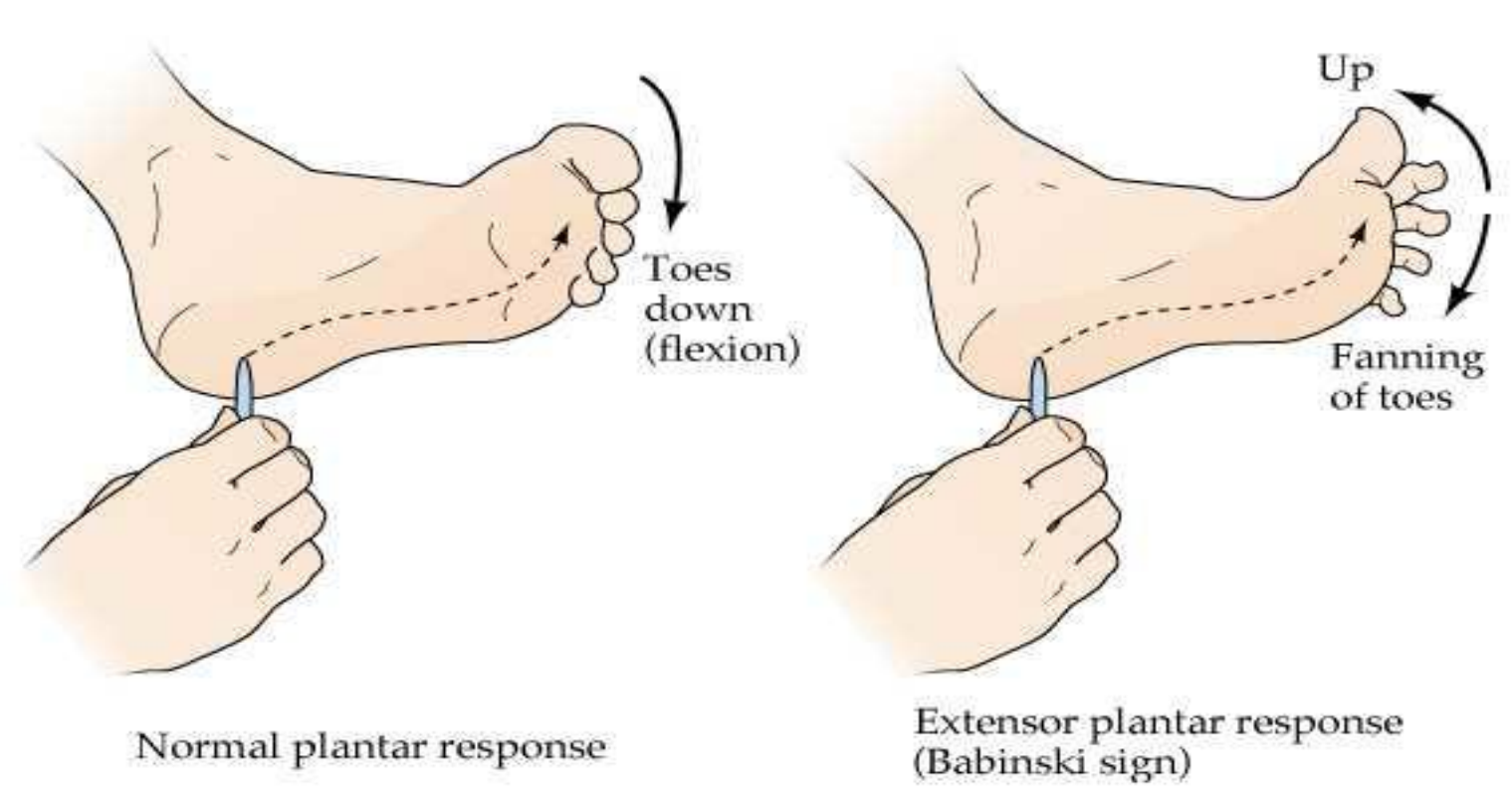
Inhibitory RF



Motor Loss (Hemiplegia)

4) **Exaggerated tendon jerk & clonus**: due to increased supraspinal facilitation.

5) **Positive Babinski's sign**



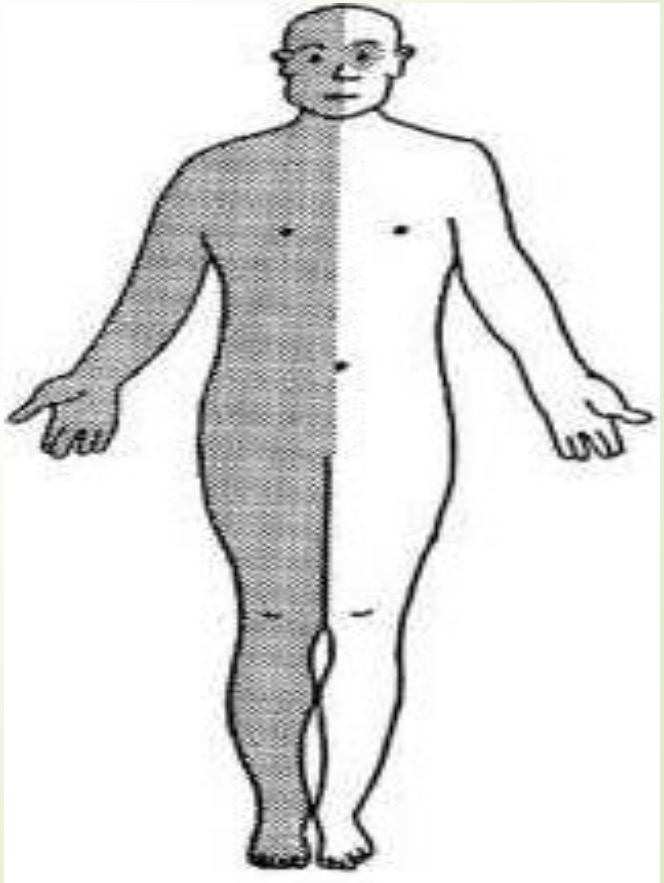
Motor Loss (Hemiplegia)

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

7) **Normal response** of the paralyzed ms to **electric stimulation**

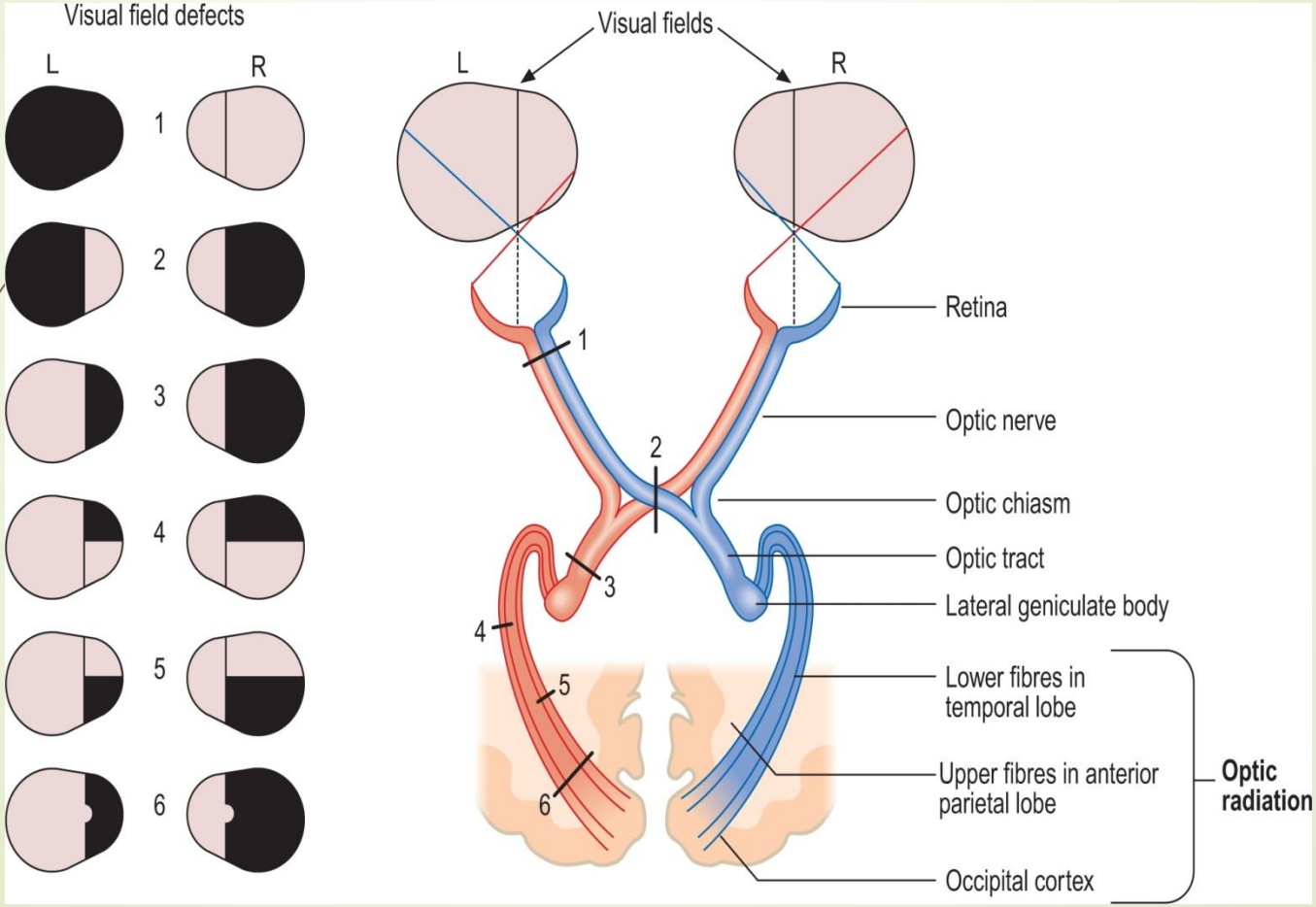
Sensory Loss

1) **Contralateral hemianaesthesia** i.e. loss of all sensations on the **opposite side** of the body



Sensory Loss

2) Contralateral homonymous hemianopia i.e., loss of vision in the two opposite halves of the field of vision



Sensory Loss

3) **Bilateral diminution** of hearing acuity.

- **No complete loss** of hearing as both ears are bilaterally represented in both cortices.

Lower Motor Neuron Lesion (LMNL)

Lower Motor Neuron Lesion

Def.,

- 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

Trauma

Neuropathy

Effects of LMNL

I) Structural changes

- In Nerve (degeneration and regeneration
- In muscle (atrophy and increase Ach receptors

II) Functional changes

1. Flaccid paralysis
2. Fasciculation and fibrillation
3. Dennervation supersensitivity
4. Reaction of degeneration

Functional Effects of LMNL

A) 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

Functional Effects of LMNL

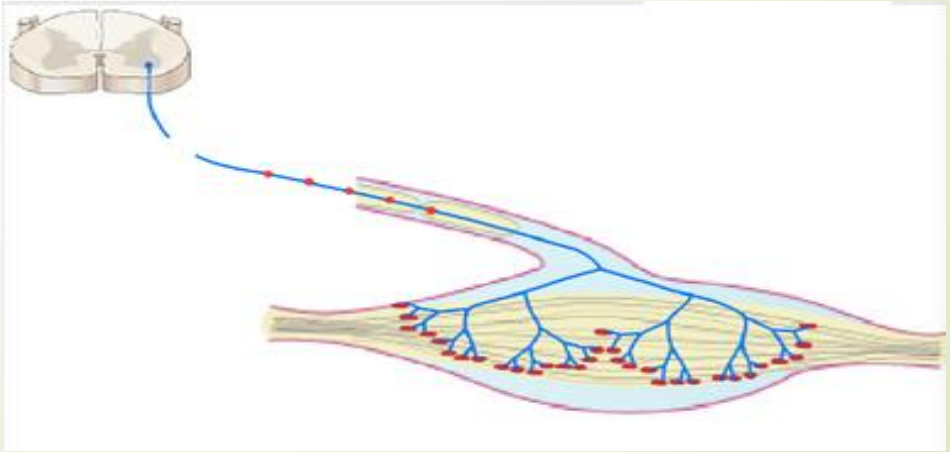
B) **Fasiculations and fibrillations:**

- Appears **few days or weeks** after denervation
- Disappear when the motor nerve completely **degenerates** or **successful re-innervation** of the ms occurs.

Functional Effects of LMNL

B) 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

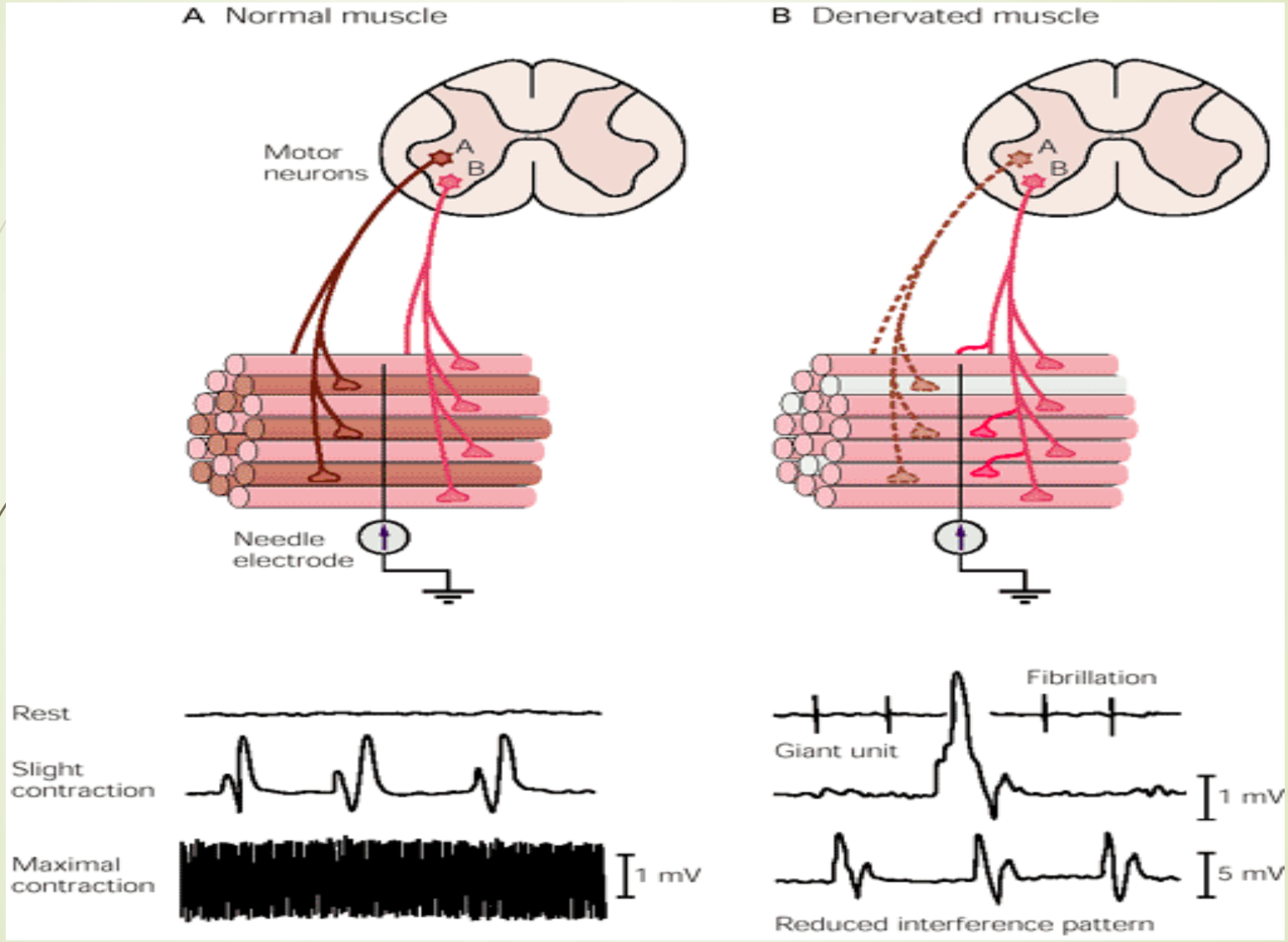


Functional Effects of LMNL

B) 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 **electromyogram (EMG).**

Fibrillations



Functional Effects of LMNL

c) **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.

UMNL and 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.

UMNL and LMNL

	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

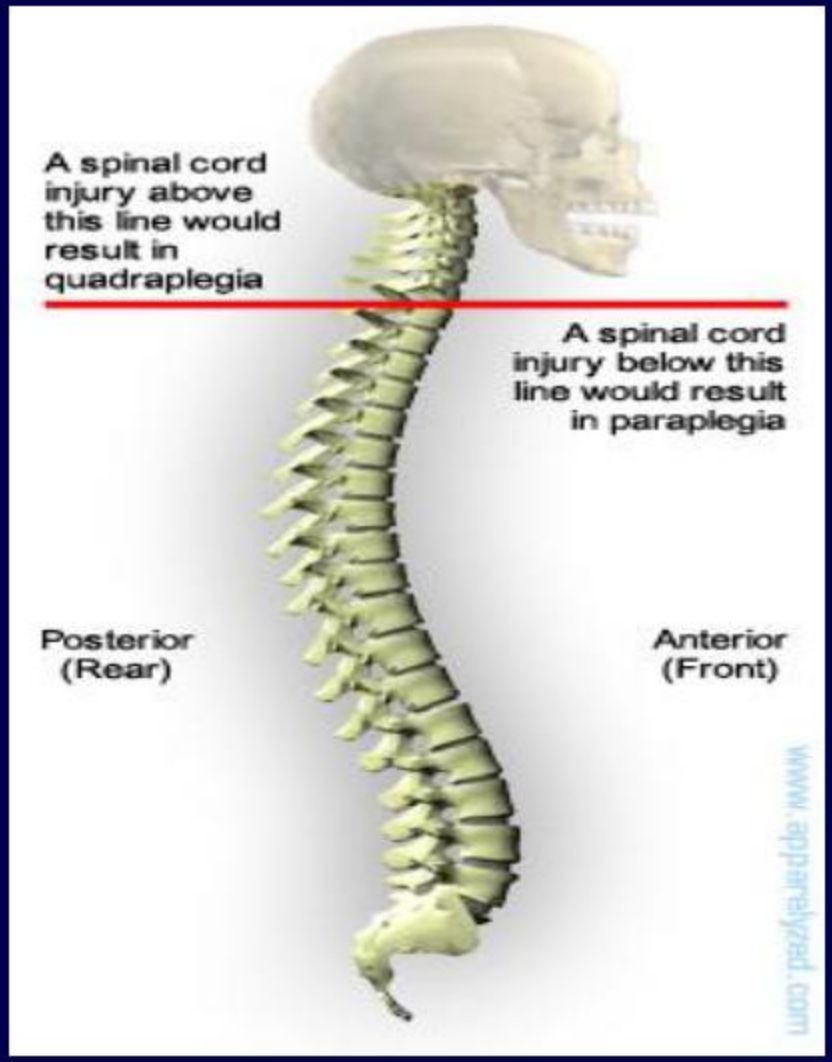
UMNL and LMNL

	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

Spinal cord lesions

Types

- Complete:
  - No function or sensation below injury site
  - Both Sides equally affected
  - Can Result in quadriplegia
- Incomplete:
  - Some function and/or sensation below injury site
  - One side may be more affected than other



Upper cervical cord lesions produce quadriplegia and weakness of the diaphragm

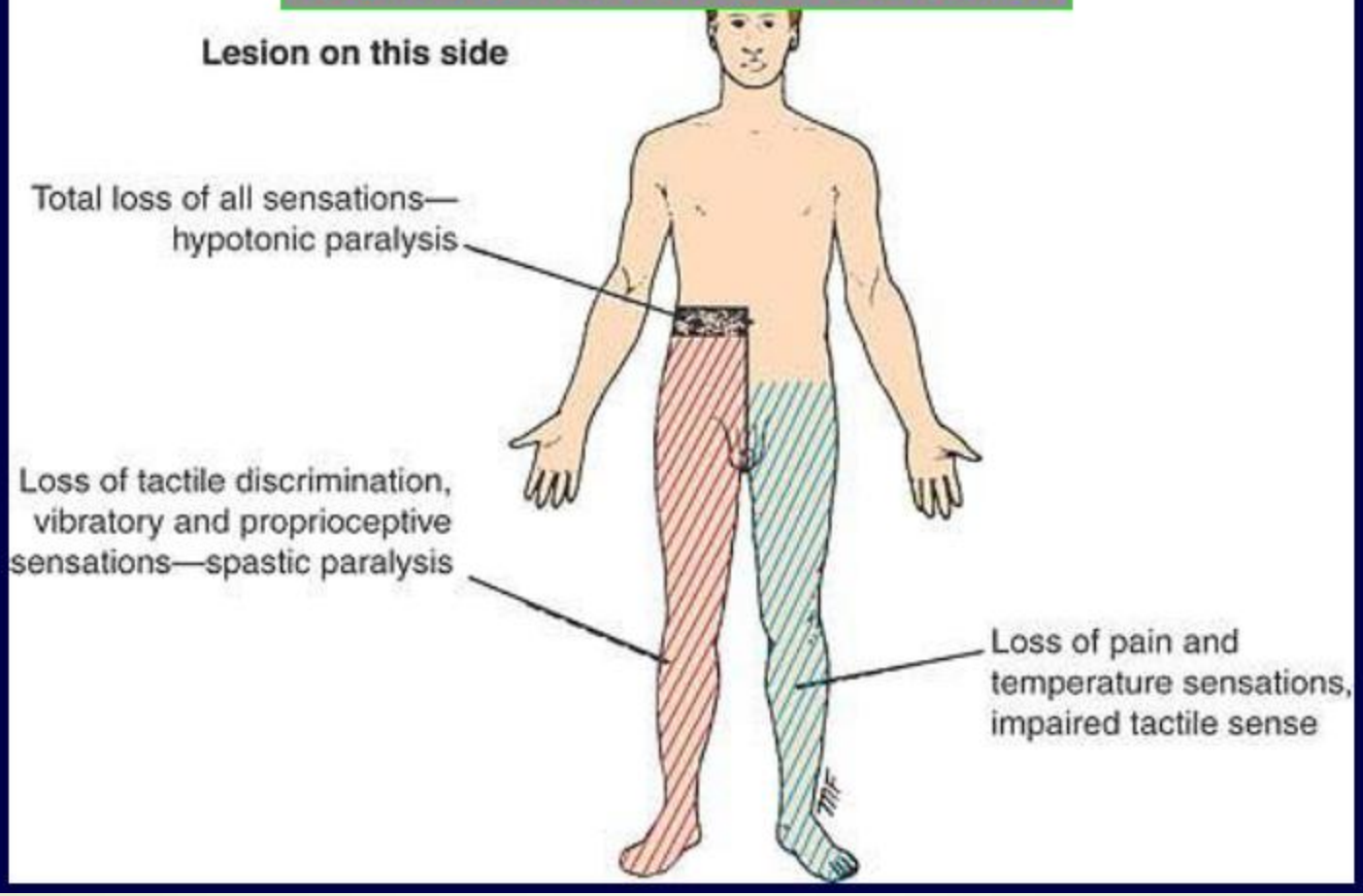
Lesions at C4-C5 produce quadriplegia

Hemiparesis means weakness

Hemiplegia means total paralysis

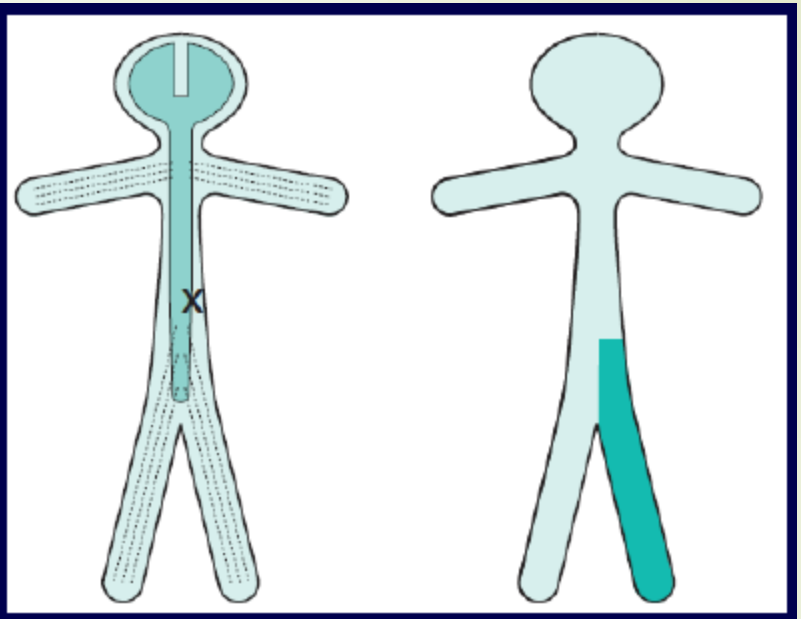
Brown Sequard syndrome

HEMISECTION OF SPINAL CORD



Ipsilateral Monoparesis

A unilateral lesion in the spinal cord below the level of the neck produces upper motor neurone weakness in one leg. There may be 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.



Ipsilateral Monoparesis

Brown Sequard syndrome

HEMISECTION OF SPINAL CORD

Ipsilateral Loss:

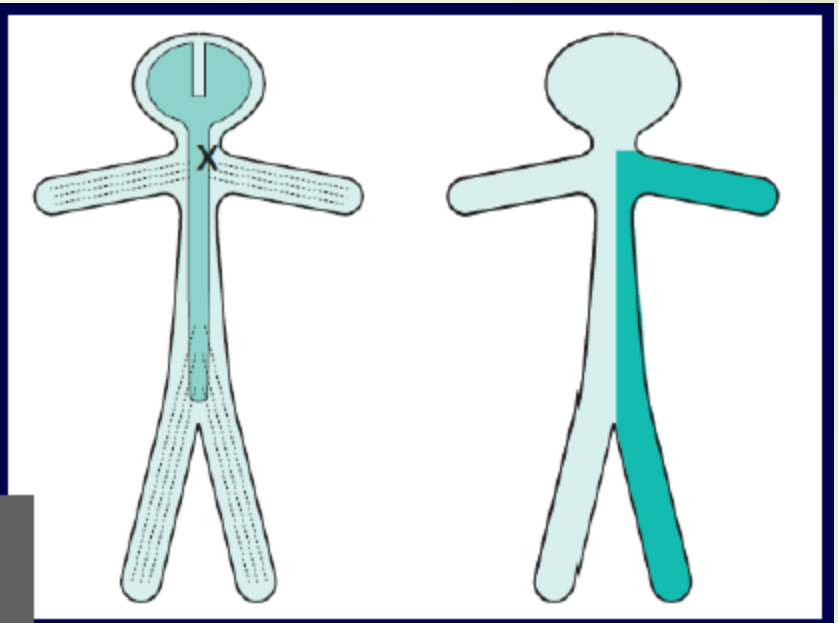
- Fine touch, Vibration, Proprioception (Dorsal Column)
- Leg Ataxia (Dorsal Spinocerebellar)
- Spastic Paresis below lesion (Lat Corticospinal)
- Flaccid Paralysis (Vent horn destruction)
- Dermatomal Anesthesia (Dorsal Horn destruction)

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)

Ipsilateral hemiparesis

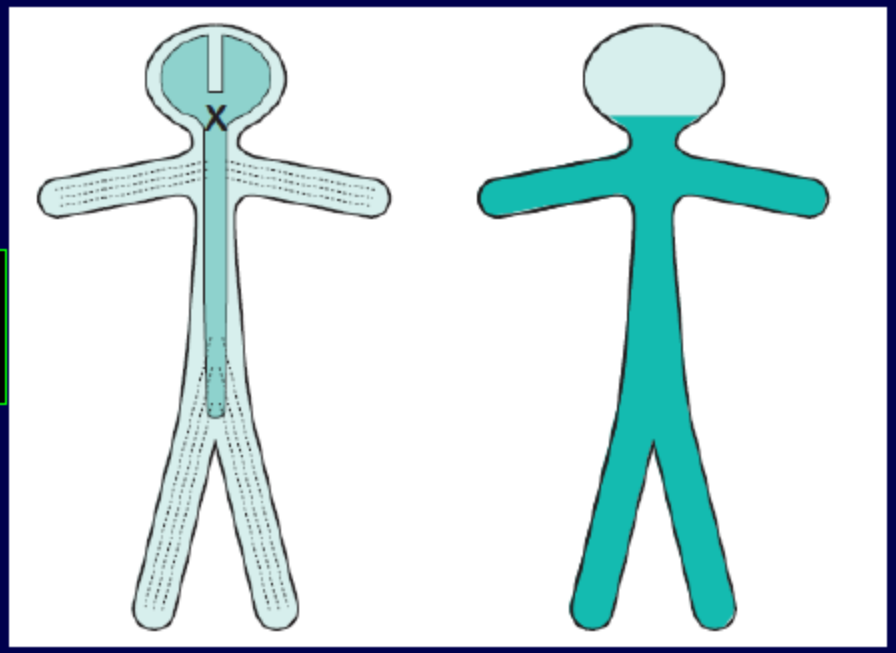
A unilateral high cervical cord lesion will produce a hemiparesis similar to that which is caused by a contralateral cerebral hemisphere lesion, except that the face cannot be involved in the hemiparesis, vision will be normal, and the same dissociation of sensory loss (referred to above) may be found below the level of the lesion.



Ipsilateral Hemiparesis

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.

Tetraparesis or Quadriparesis



Tetraparesis or quadriparesis, if the lesion is in the upper cervical cord or brainstem.

Bulbar palsy

- **B/L LMN** defect of IX-XII cranial nerves
- Dysphagia (liquid>solid), nasal regurgitation, slurred speech
- Nasal speech, wasted tongue with fasciculation, absent gag reflex

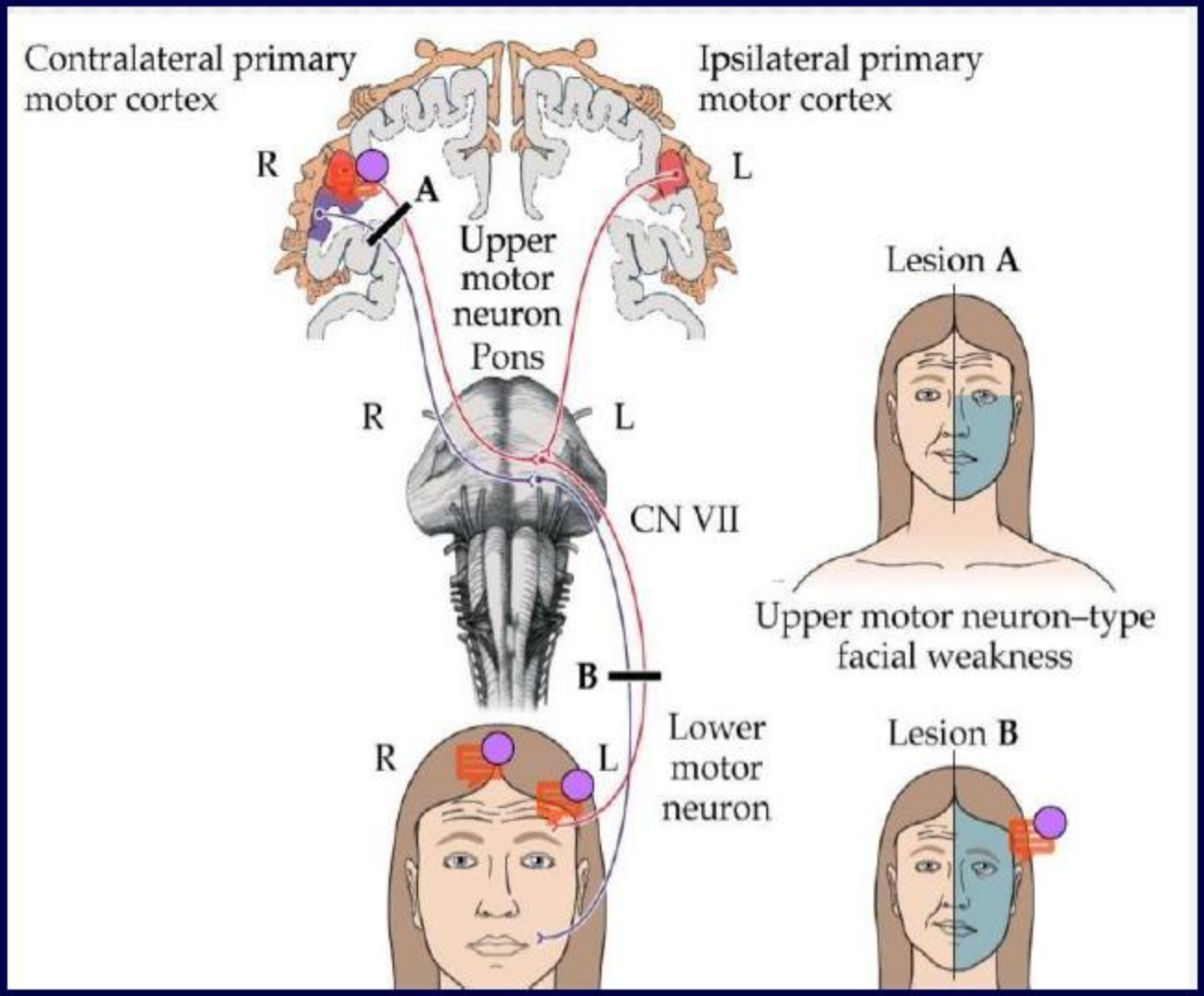
Pseudobulbar palsy-

- **B/L UMN defect** of IX-XII cranial nerves
- Dysphagia, dysarthria, emotional lability
- Slow indistinct speech, spastic tongue, brisk jaw jerk
- Frontal release signs

VIIth Cranial Nerve lesion

UMN VIIth Cranial Nerve lesion: 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.

LMN VIIth Cranial Nerve lesion 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.


Contralateral primary motor cortex
Ipsilateral primary motor cortex
R
A
L
Upper motor neuron
Pons
R
L
CN VII
B
R
L
Lower motor neuron
Lesion A
Upper motor neuron–type facial weakness
Lesion B

Motor neuron disease

- Selectively affect motor neurons, that control voluntary muscle activity
- Types-
  - **Amyotrophic lateral sclerosis- UMN+LMN**
  - **Primary lateral sclerosis- UMN**
  - **Progressive muscular atrophy- LMN**
  - **Bulbar palsy- bulbar LMN**
  - **Pseudobulbar palsy- bulbar UMN**

Spinal cord

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

BLADDER CONTROL

Cortical:

- Post-central lesions cause loss of sense of bladder fullness.
- Pre-central lesions cause difficulty initiating micturition.
- Frontal lesions cause socially inappropriate micturition.

Spinal cord

- **Bilateral UMN lesions (pyramidal tracts) cause**
- **urinary frequency and incontinence. The bladder is small and**
- **hypertonic, i.e. sensitive to small changes in intravesical pressure.**
- **Frontal lesions can also cause a hypertonic bladder.**

LMN

- **Sacral lesions (conus medullaris, sacral root and pelvic**
- **nerve – bilateral) cause a flaccid, atonic bladder that overflows**
- **(cauda equina), often unexpectedly.**

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- **nerve – bilateral) cause a flaccid, atonic bladder that overflows**
- **(cauda equina), often unexpectedly.**


THANKS