

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

(وَمَا أُوتِيتُمْ مِنَ الْعِلْمِ إِلَّا قَلِيلًا)

صدق الله العظيم  
الاسراء اية ٥٨

# UPPER & LOWER MOTOR NEURON

## lesion

By

Dr Fawzia ALROuq

Ph.D

Assistant Professor of physiology

Consultant of neurophysiology

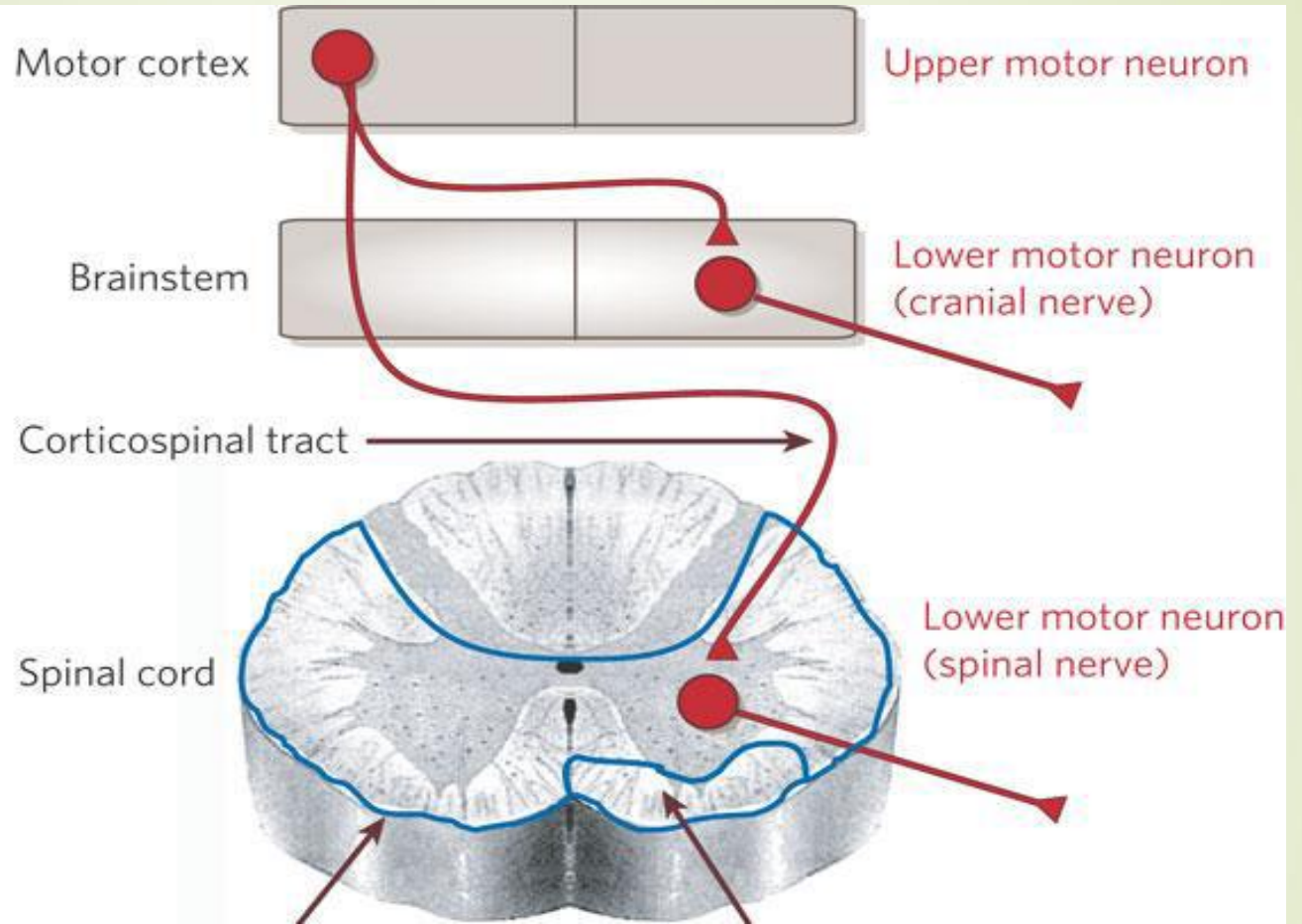
Department of Physiology

College of Medicine, King Saud University University



# 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)

	<u>UMNL</u>	<u>LMNL</u>
1-extent of paralysis	widwsread	localized
2-site of paralysis	Opposite side to lesion	Same side of lesion
3-Tone of muscles	Spasticity ( hypertonia ) ” clasp-knife spasticity	Hypotonia ” flaccid paralysis
4- Deep reflexes	Brisk ( exaggerated) tendon jerks	Diminished or absent
5- Superficial reflexes	absent	absent
6-Planter reflex	Extensor plantar reflex , Babinski sign ( dorsiflexion of the big toe and fanning out of the other toes ) , or just an upgoing toe .	Absent .
7-muscle waisting	No marked muscle wasting , but <u>minor</u> <u>wasting</u> 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: ➤

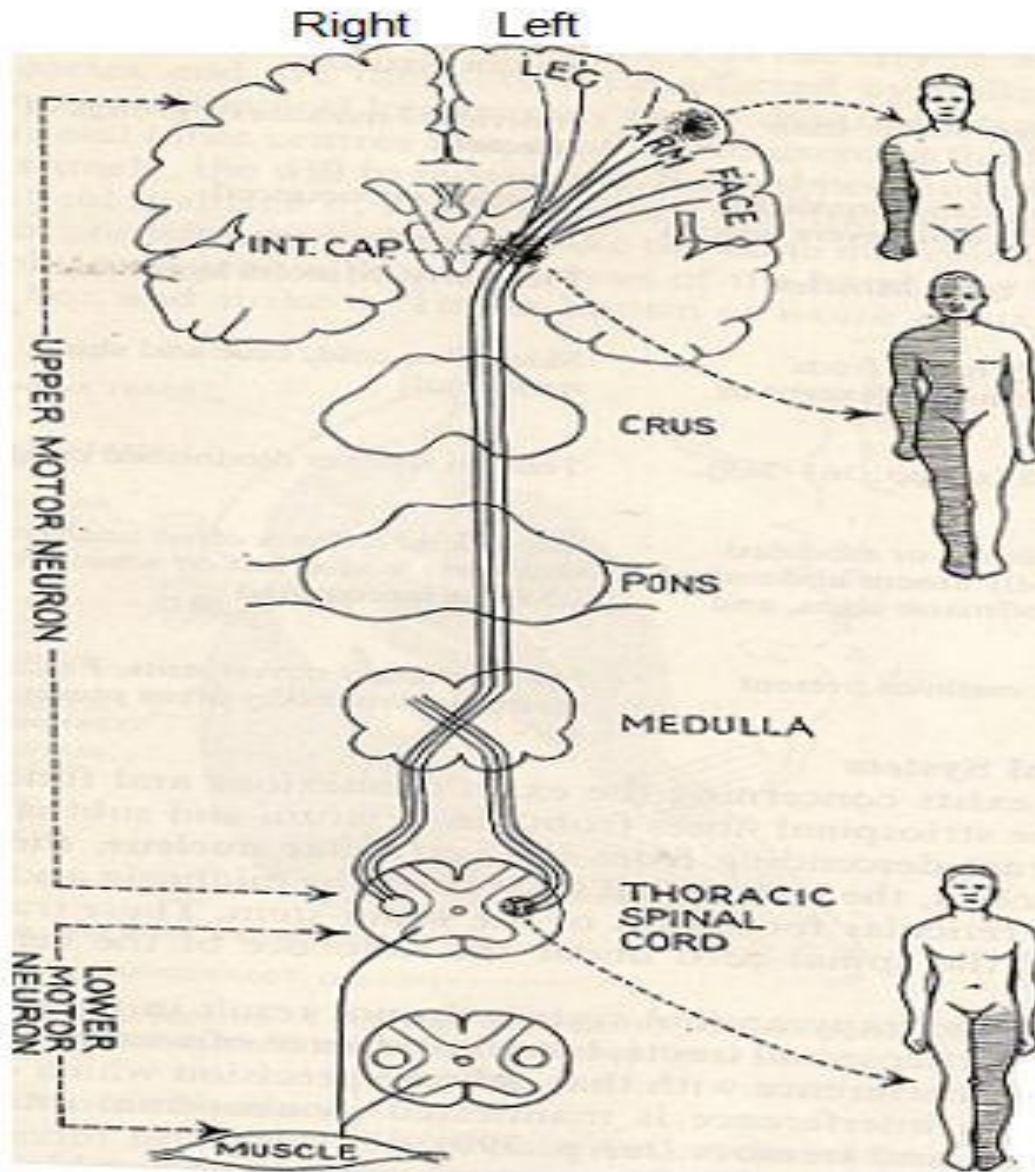
This leads to restricted paralysis\_e.g. contralateral monoplegia (paralysis of one limb because area 4 is widespread so it is rarely damaged completely. ➤

## 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 ➤



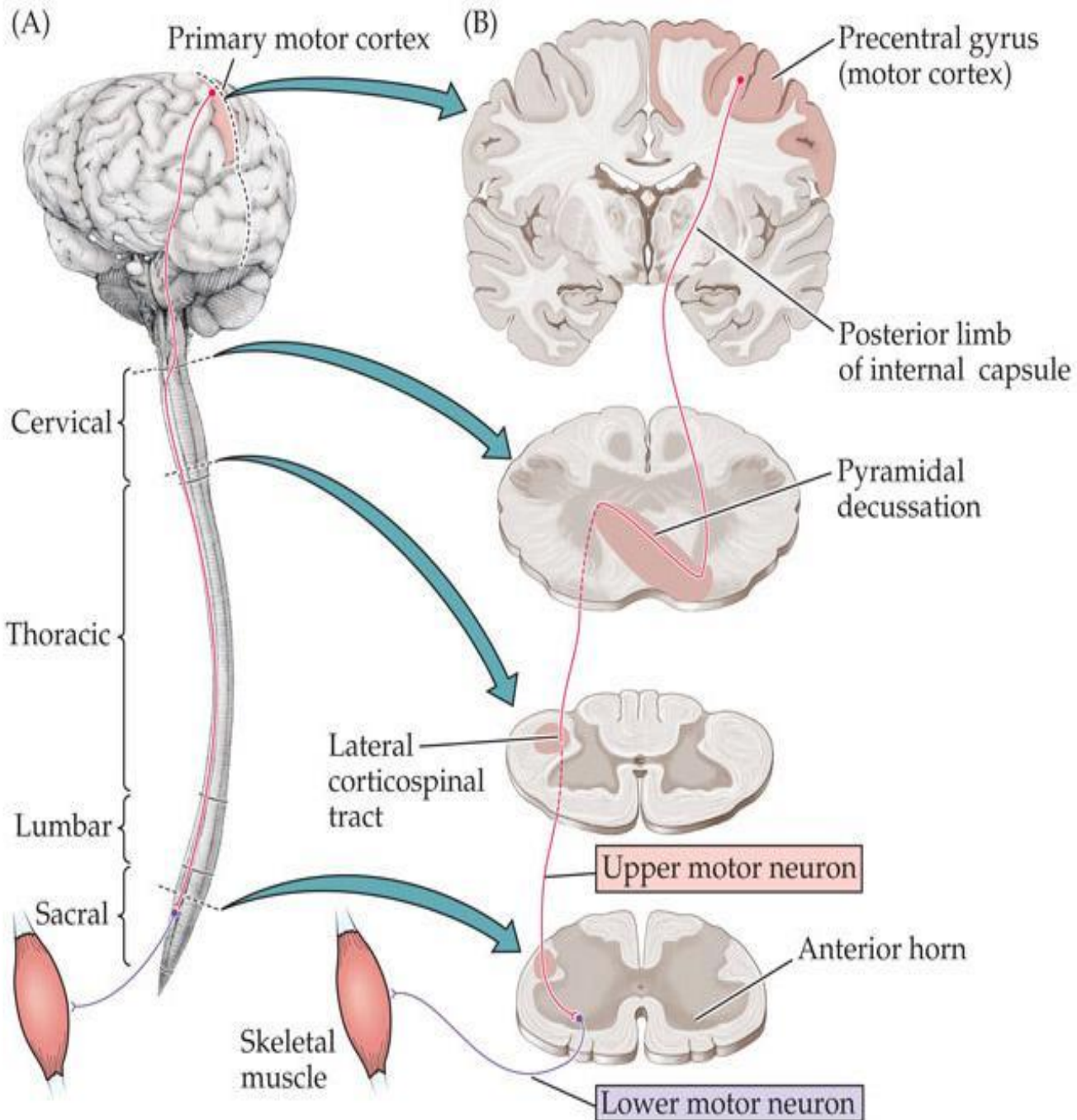
Effect of lesion

**Contralateral Monoplegia**

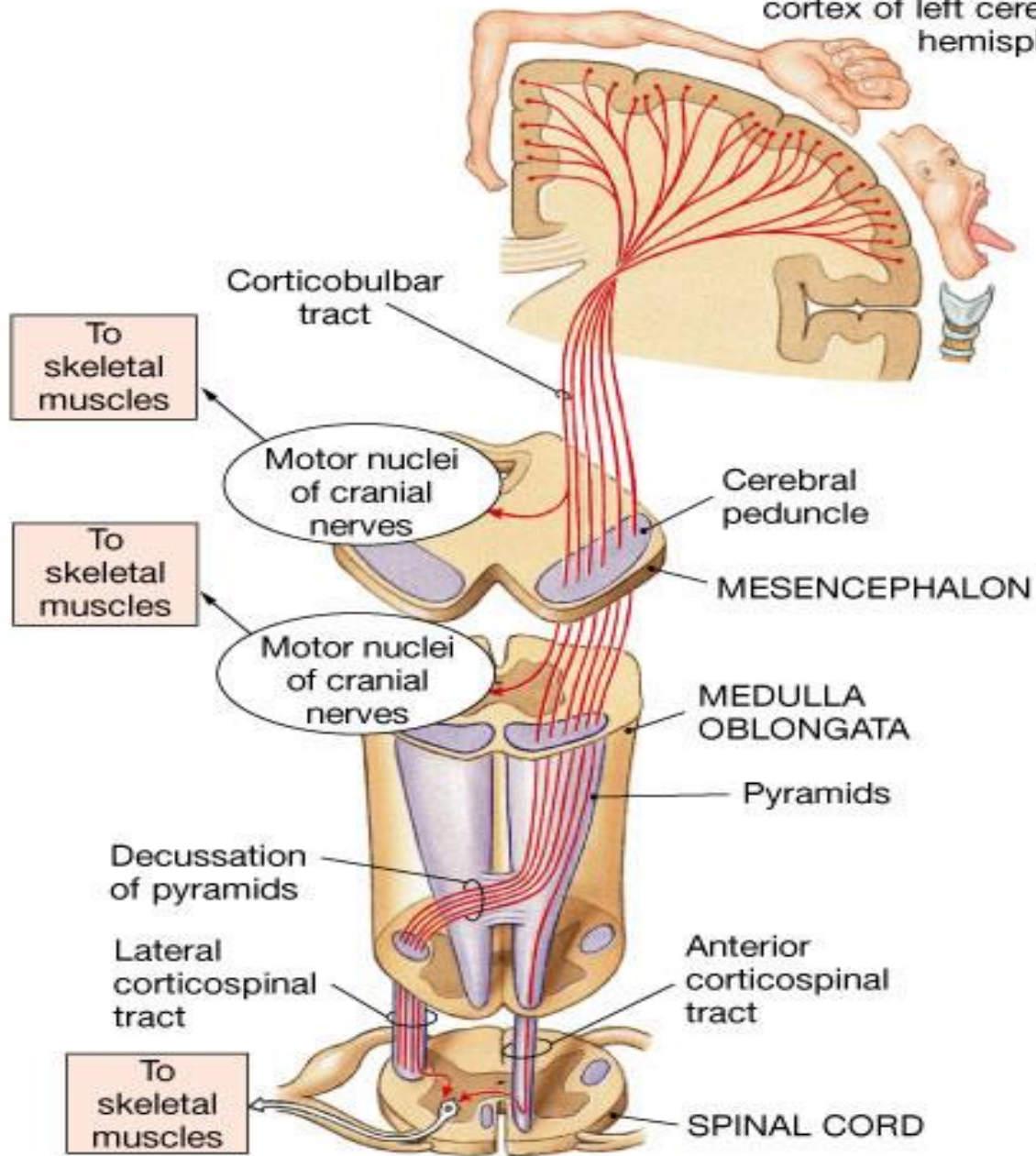
**Contralateral hemiplegia**

**Ipsilateral Monoplegia**





Motor homunculus on primary motor cortex of left cerebral hemisphere



# Extrapyramidal Tracts



# Extrapyramidal Tracts

## Origin:

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

Reticular formation

Vestibular nuclei

Red nucleus

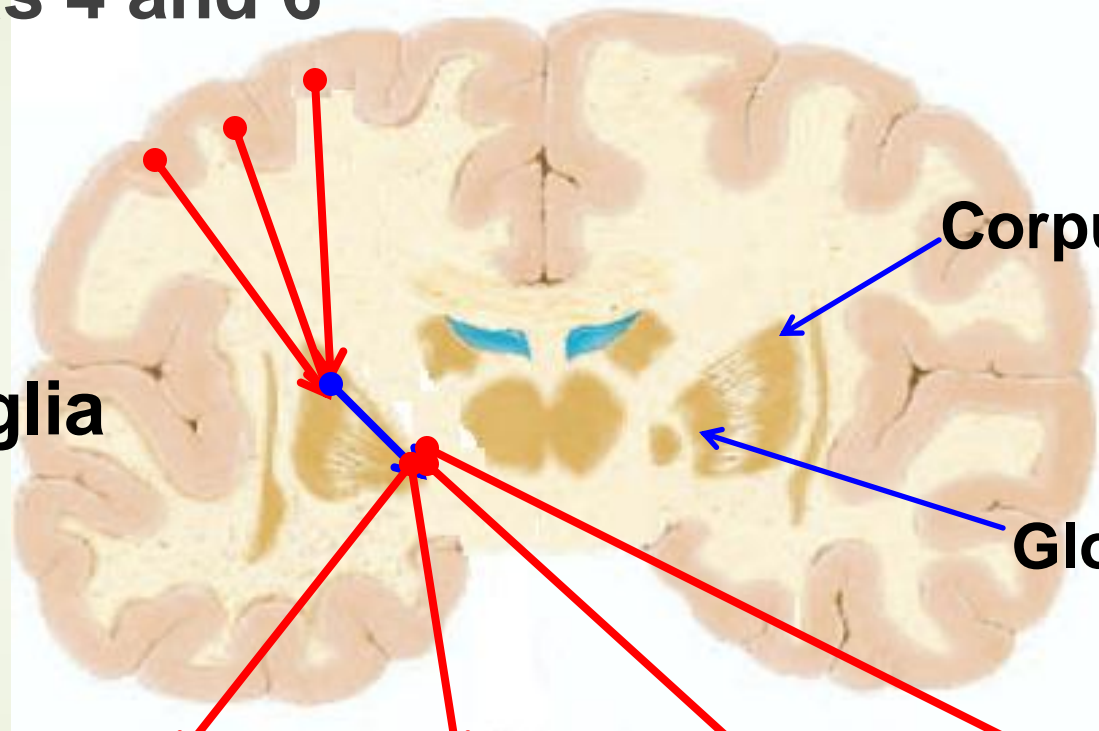
Tectum of midbrain.

From these nuclei the extrapyramidal tracts arise

# Extrapyramidal Tracts

Motor areas 4 and 6

Basal Ganglia



Corpus striatum

Globus pallidus

RF

Vest. Nuclei

Red Nucl.

Tectum

Ret.Spin T.

Vest.Spin.T.

Rubrospin. T.

Tectospin. T.



# **Upper and Lower Motor Neuron Lesion (UMNL and LMNL)**

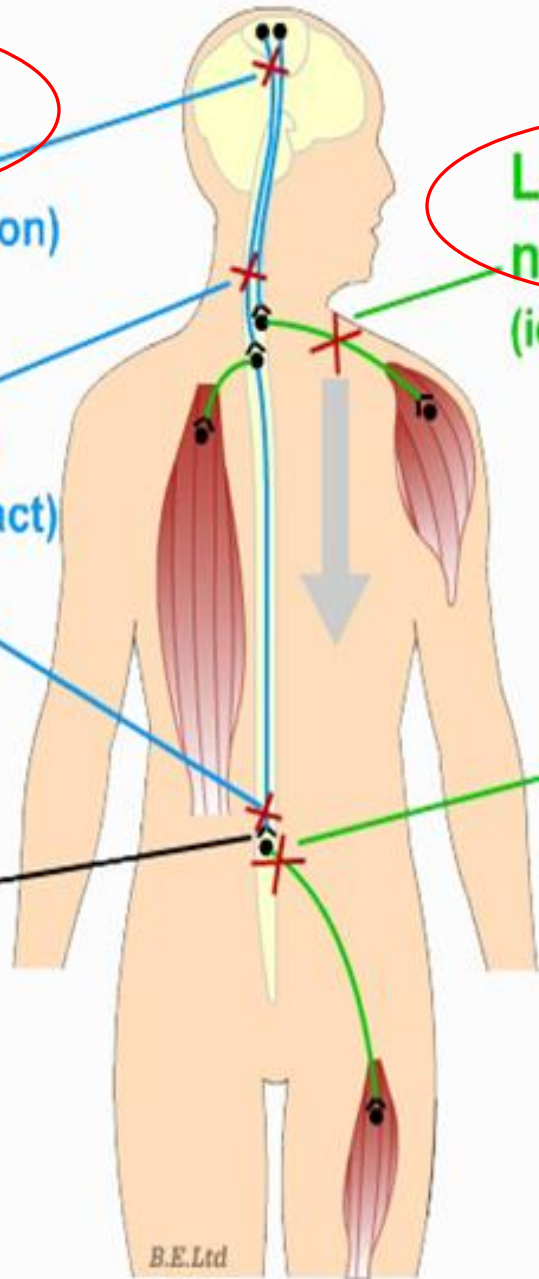
**Upper motor neurone lesion**  
(ie. cerebral infarction)

**Upper motor neurone lesion**  
(ie. corticospinal tract)

**Synapse**

**Lower motor neurone lesion**  
(ie. of peripheral nerve)

**Lower motor neurone lesion**  
(ie. at nerve root)



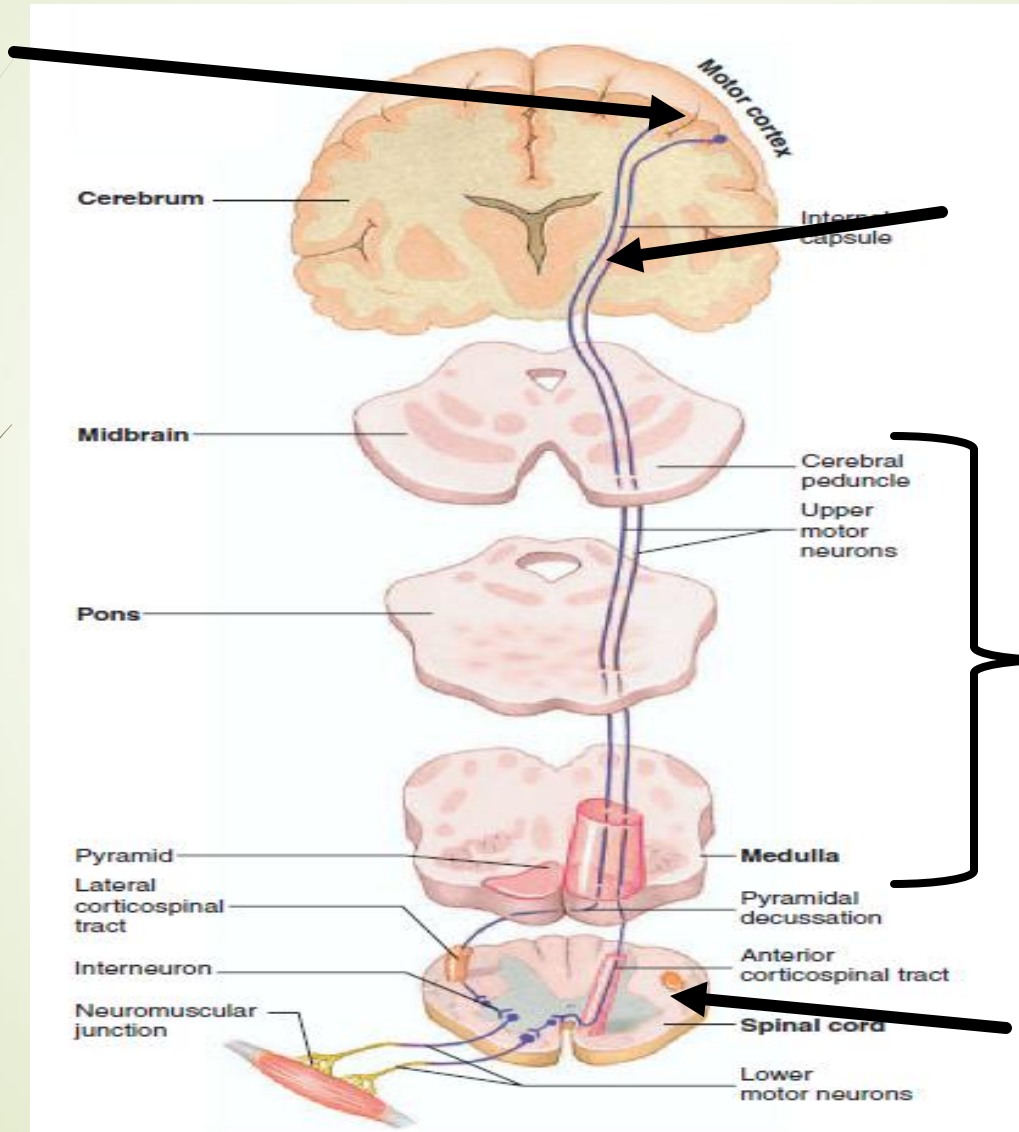
# Upper Motor Neuron Lesion (UMNL)





# 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

Trauma .۱

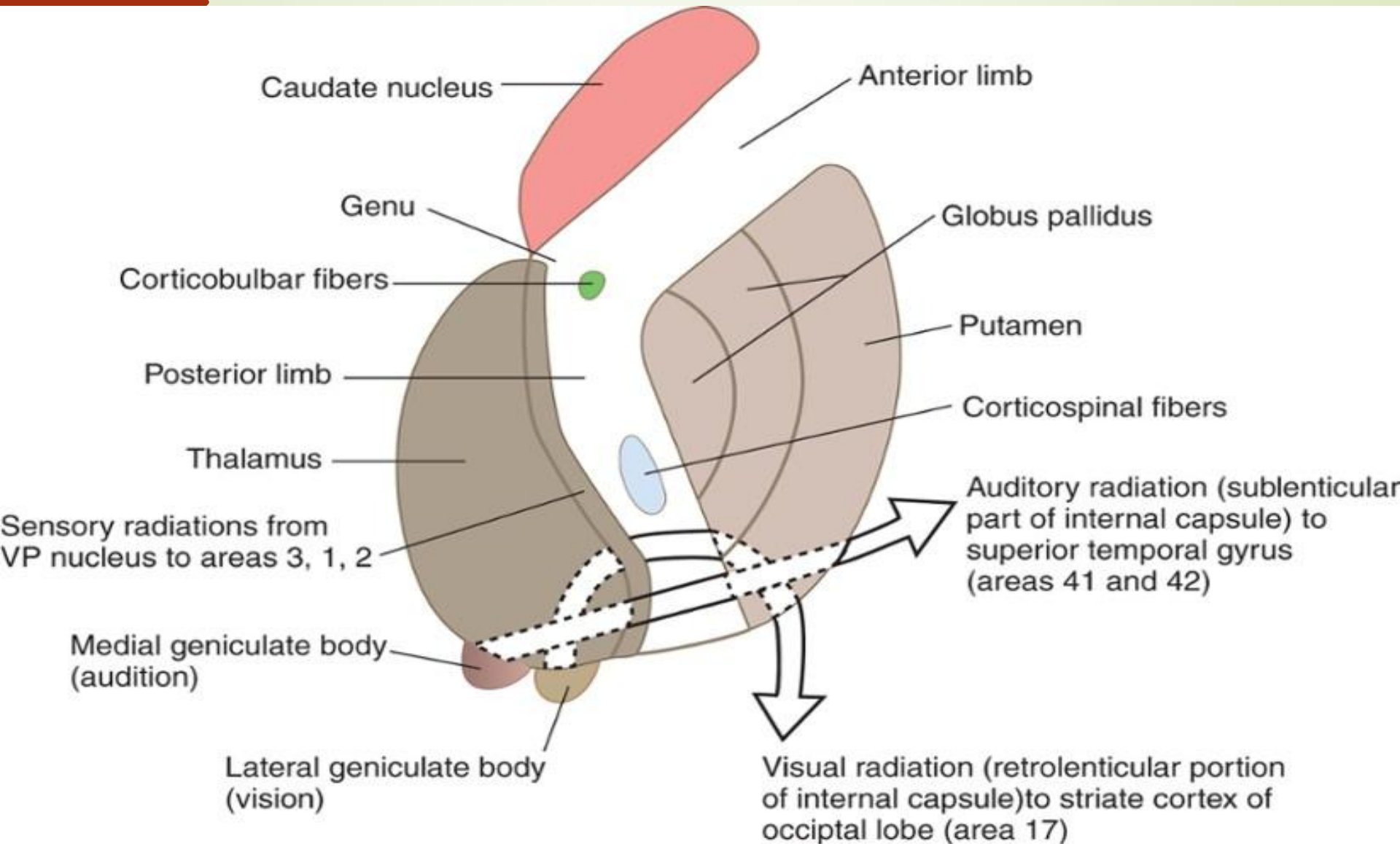
Tumour .۲

Vascular disorders as thrombosis or hemorrhage. .۳

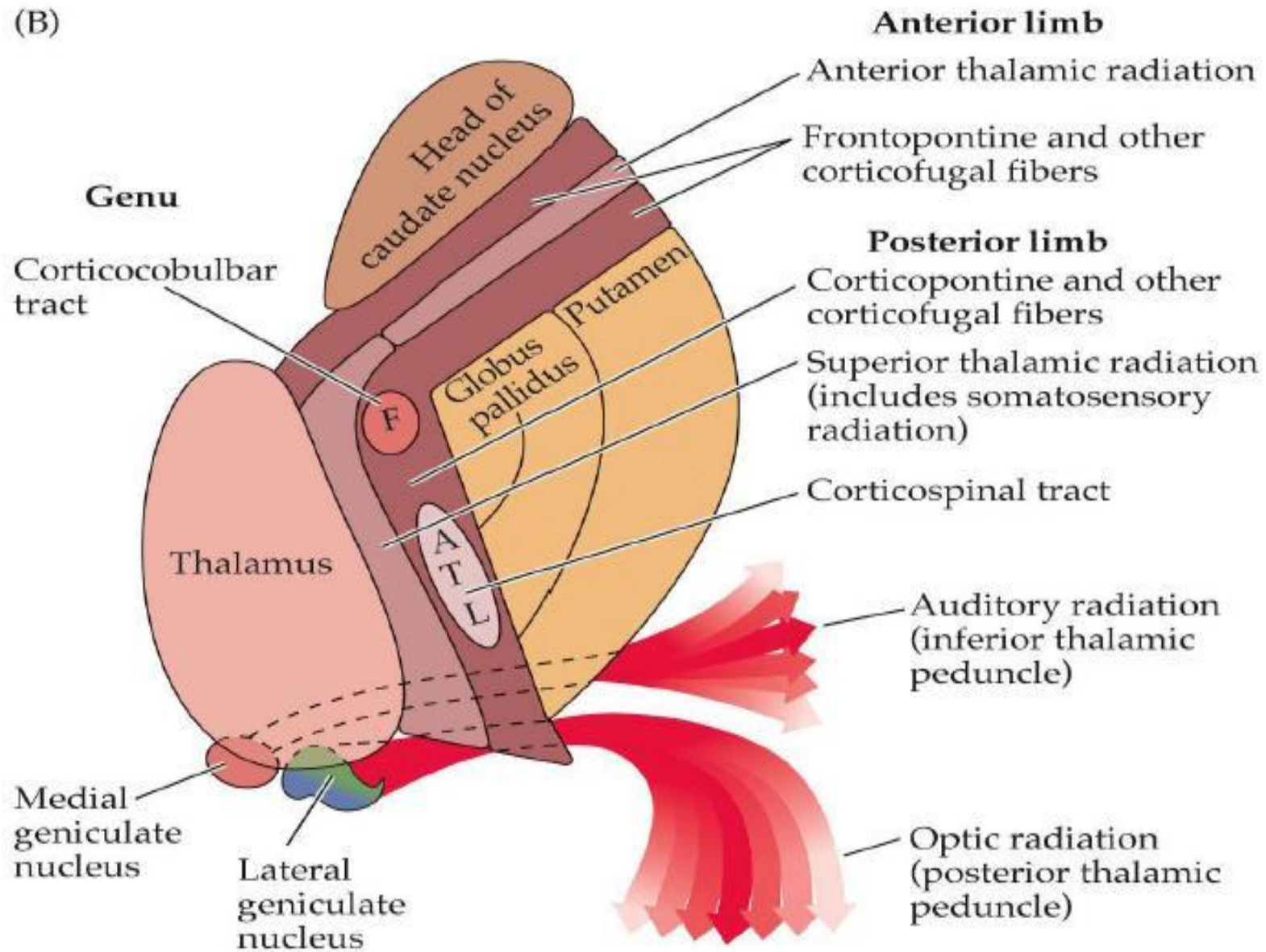
## Sites:

Most common site of UMNL is the internal capsule. ➡

# Internal Capsule



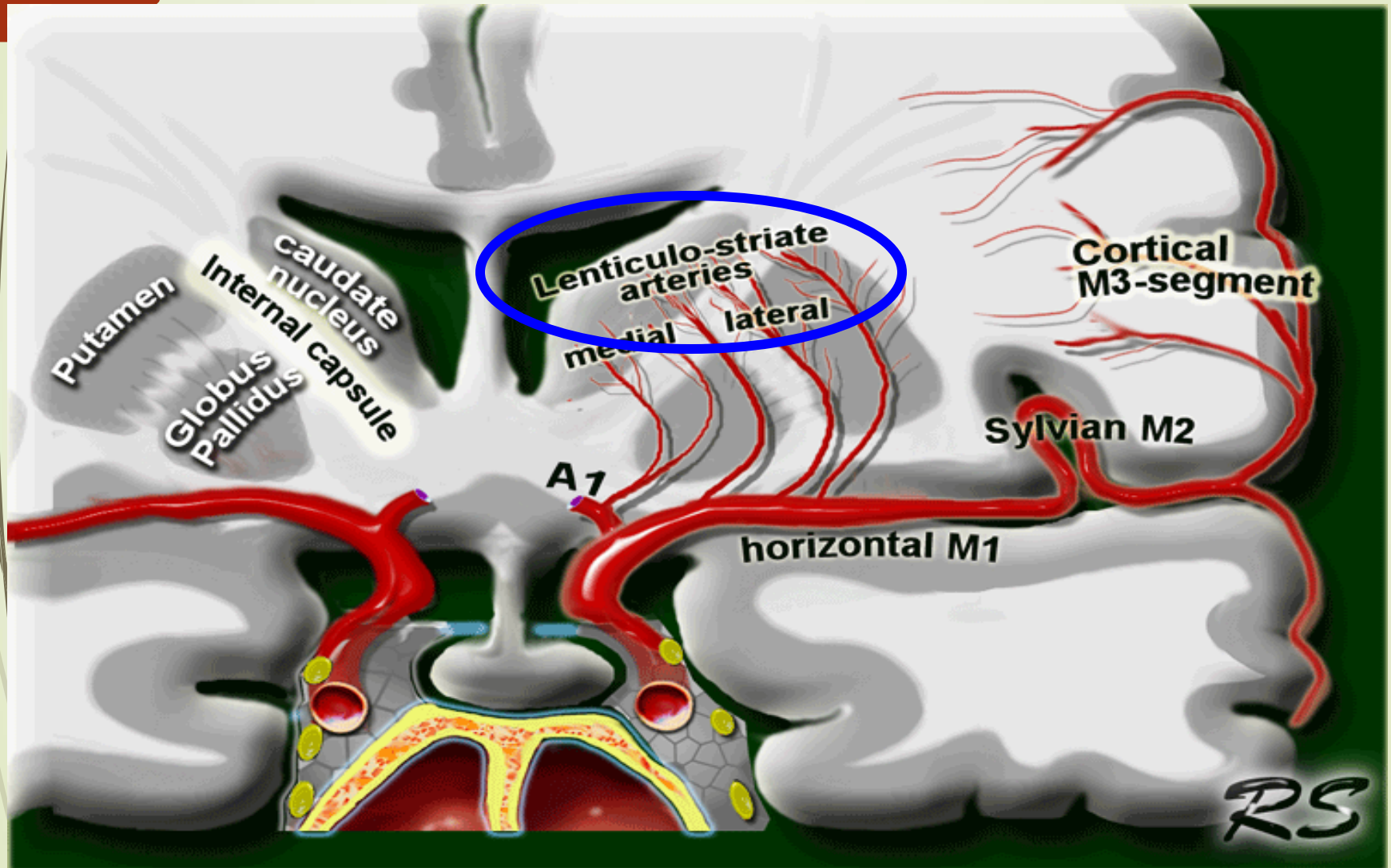
(B)



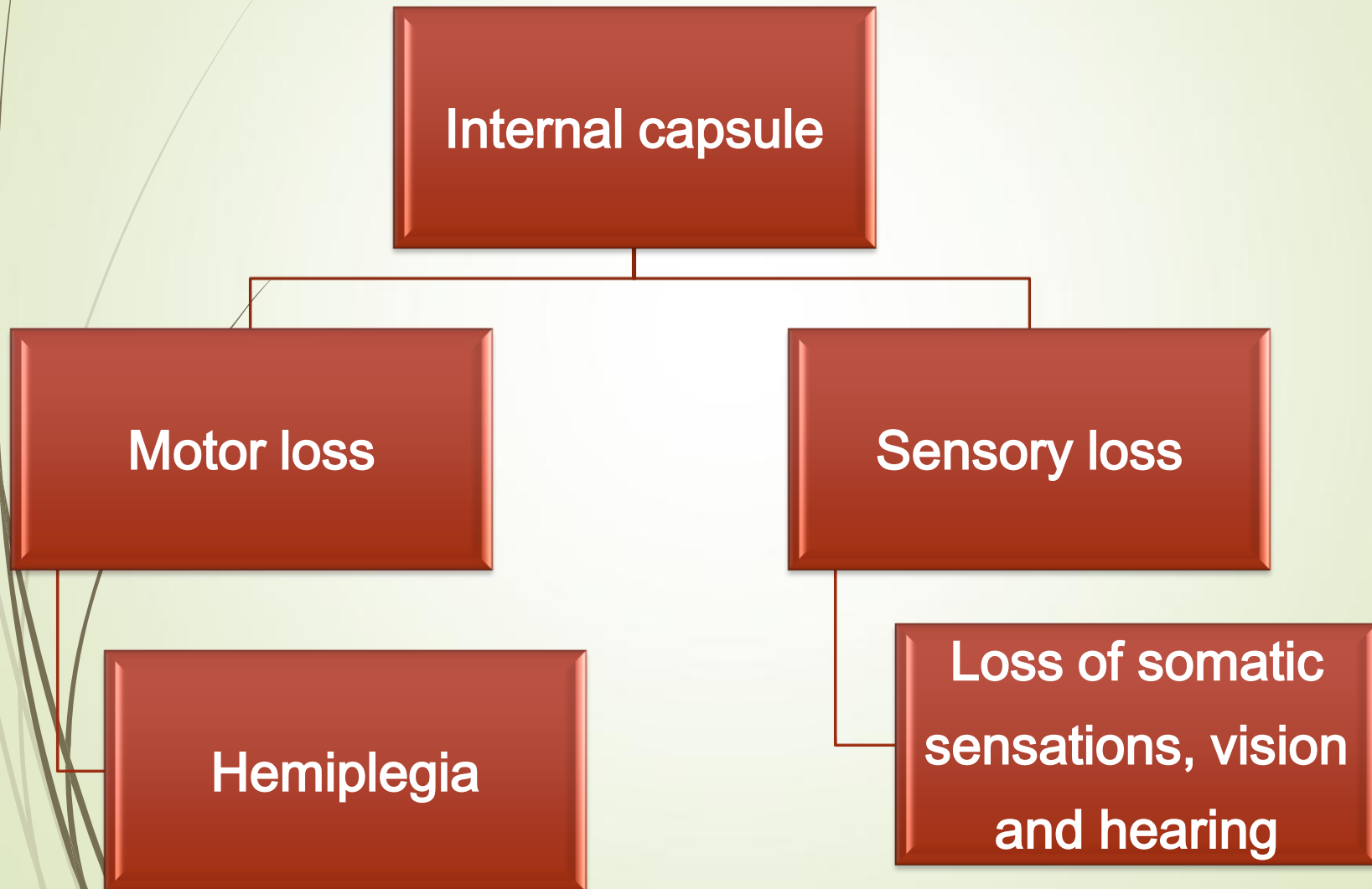
© 2002 Sinauer Associates, Inc.

Figure showing Corticospinal & Corticobulbar Fibers in Internal capsule

# Thrombosis of Lenticulostriate artery



# Damage of Posterior Limb of Internal Capsule



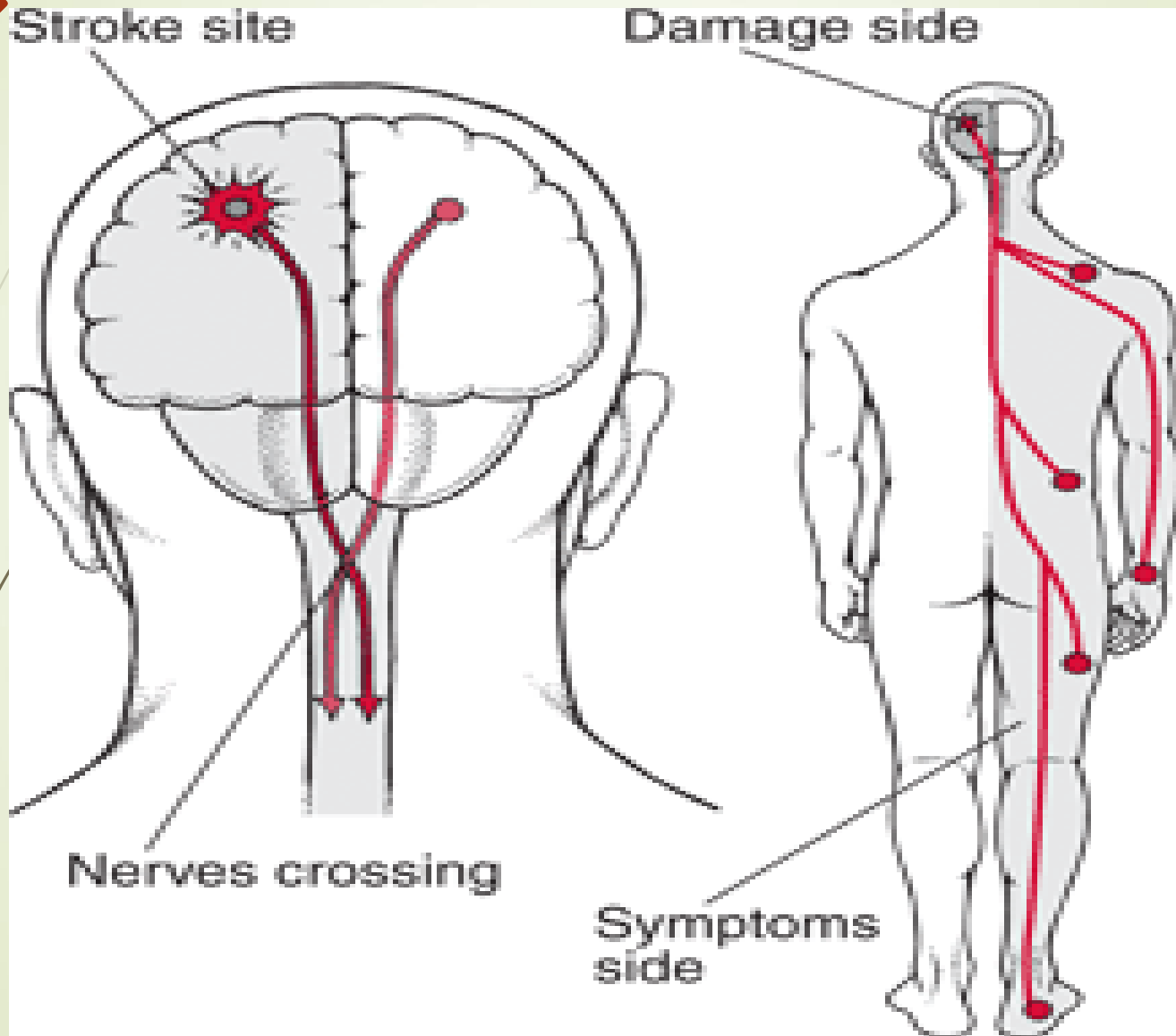
# Motor Loss (Hemiplegia)

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

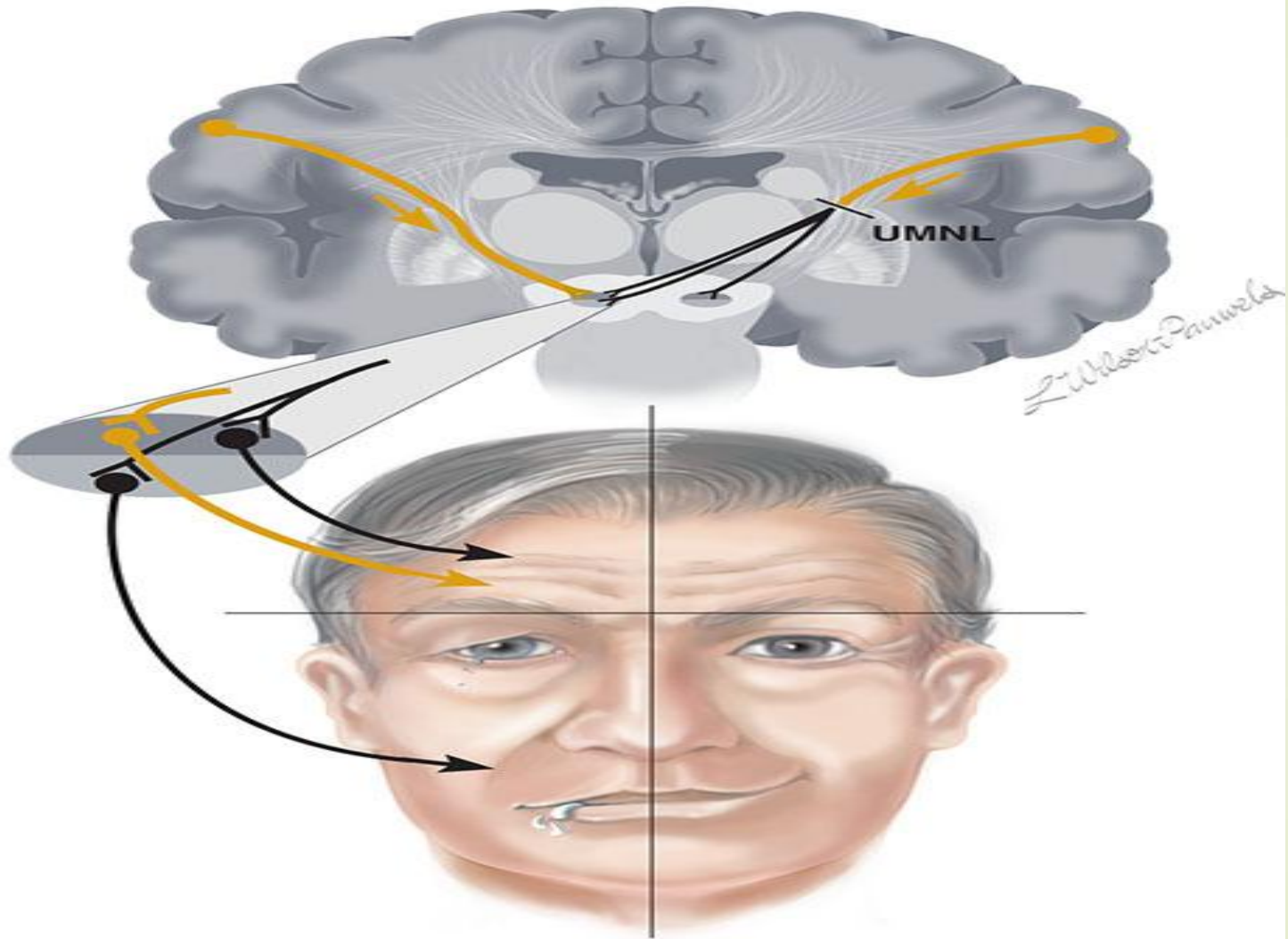
**Contralateral paresis** (weakness i.e., the ms retains some movements) of the axial ms and upper facial ms. .2

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)







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  $\gamma$ -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.

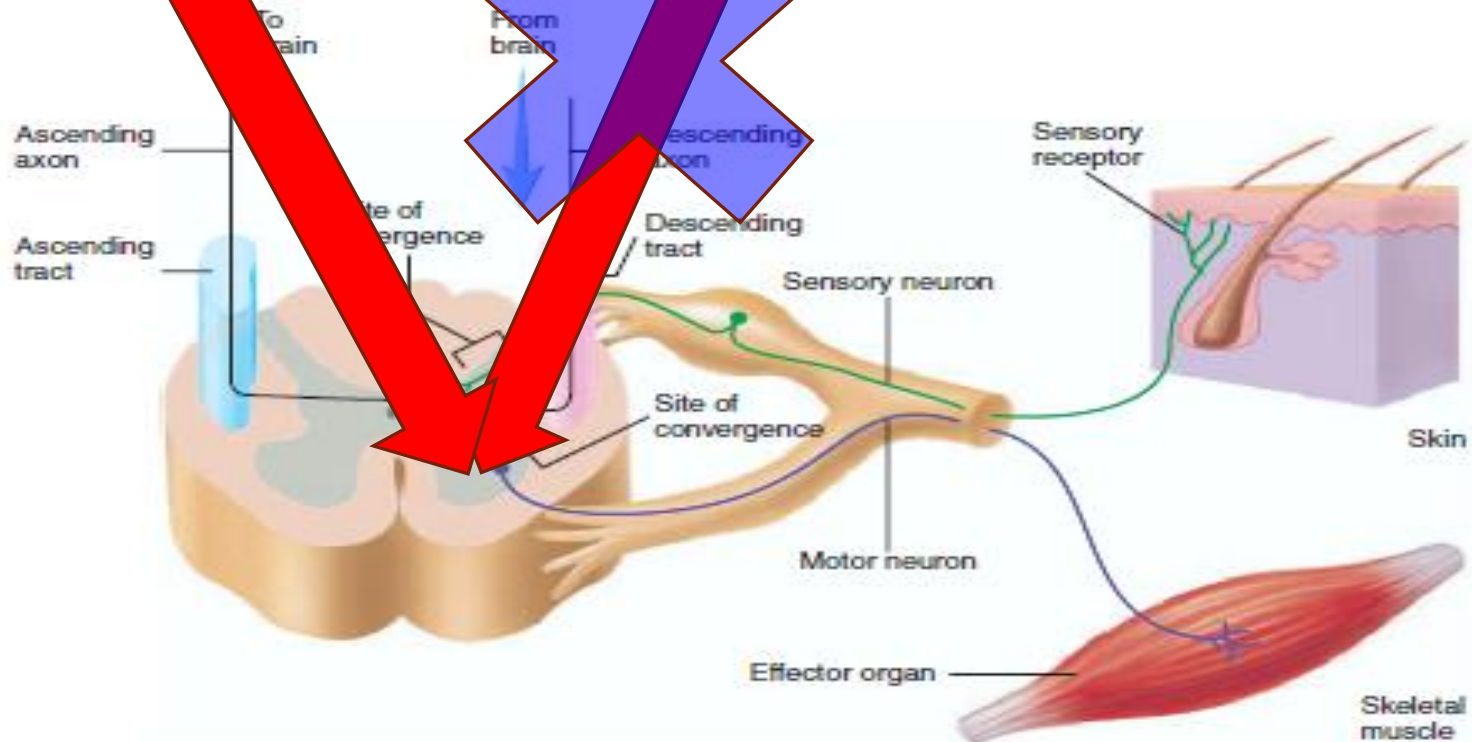
This spasticity is of the **clasp-knife type**



# Spasticity

Facilitatory RF  
and VST

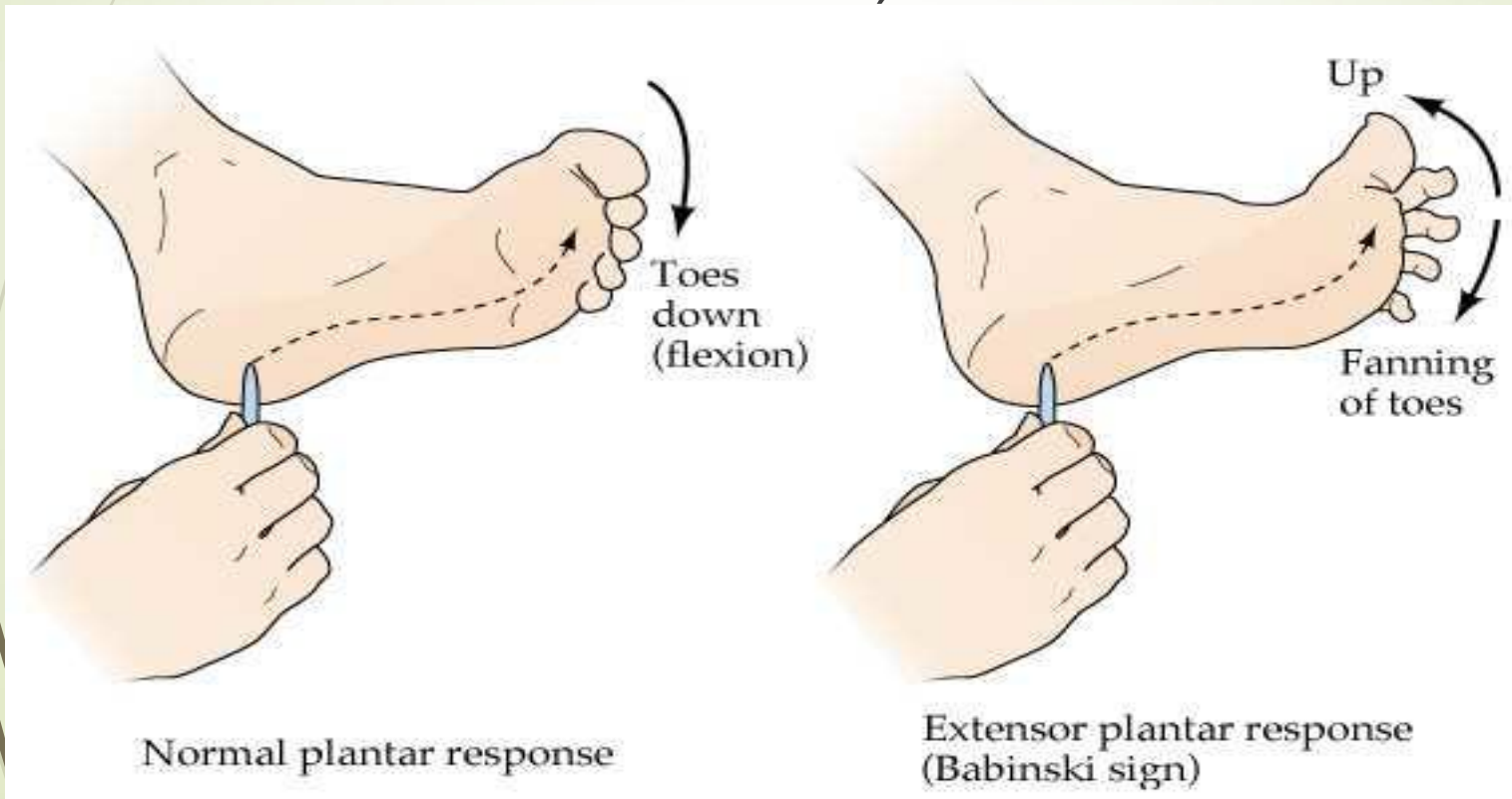
Inhibitory RF



# 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

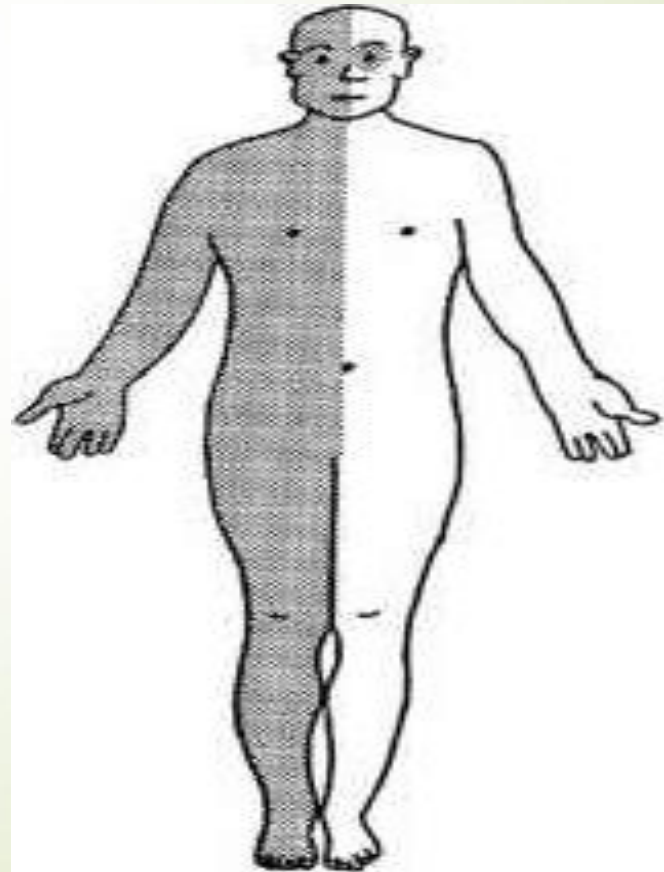
a) **Faradic current** produces clonic or tetanic contractions

b) **Galvanic current** produces contractions that occur only at closing (make) and opening (break) of the circuits.

**CCC > ACC > AOC > COC**

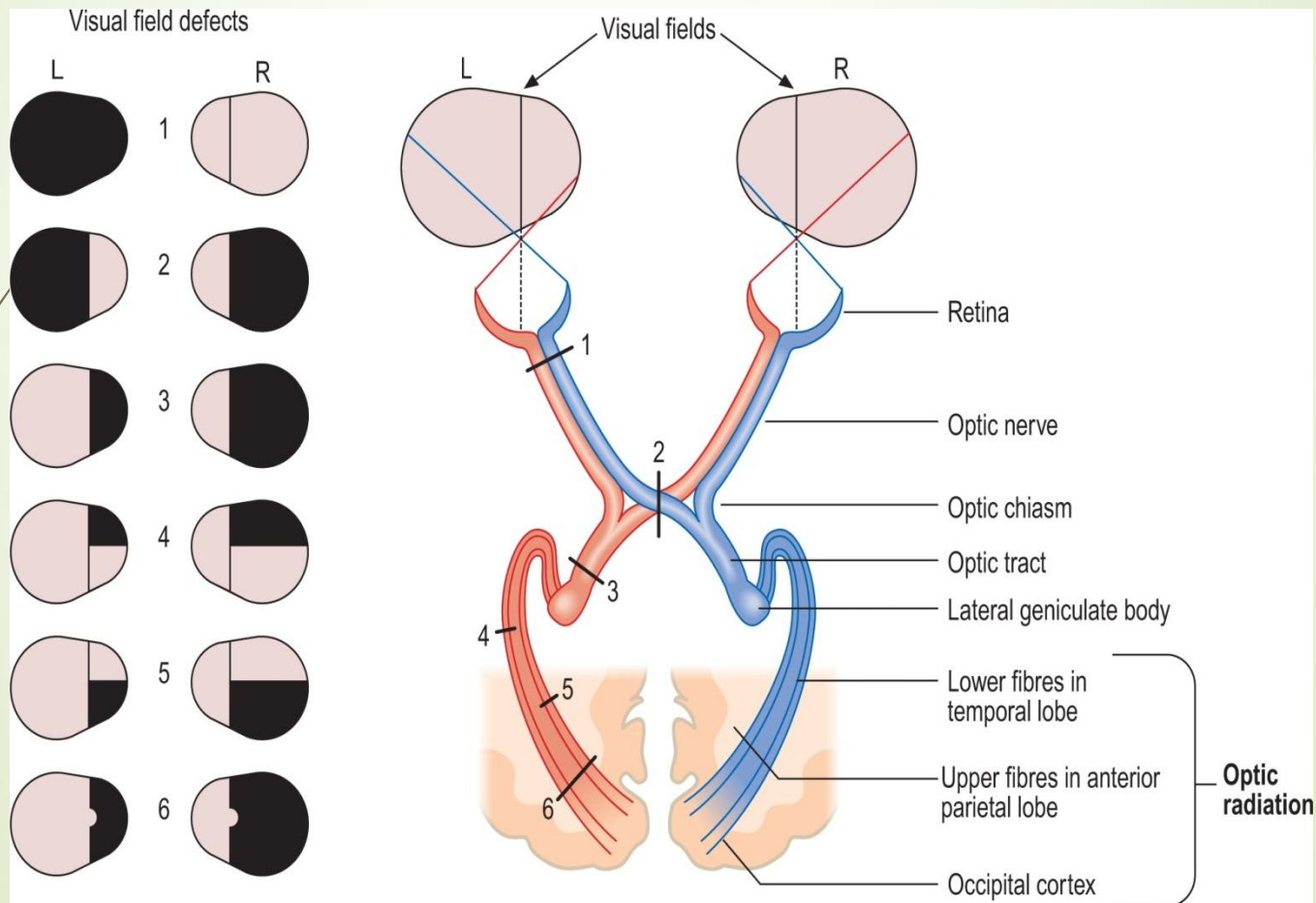
# Sensory Loss

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. Denervation 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

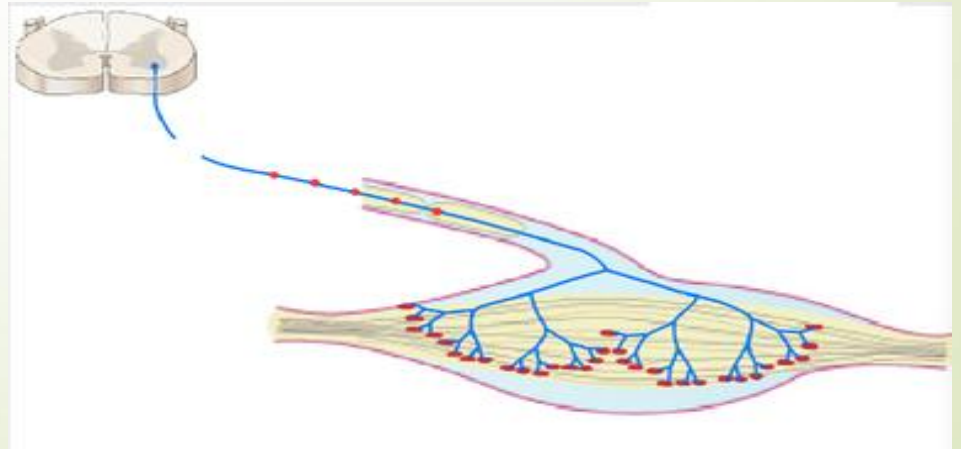
## B) Fasciculations and fibrillations:

- 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) Fasciculations :

- 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



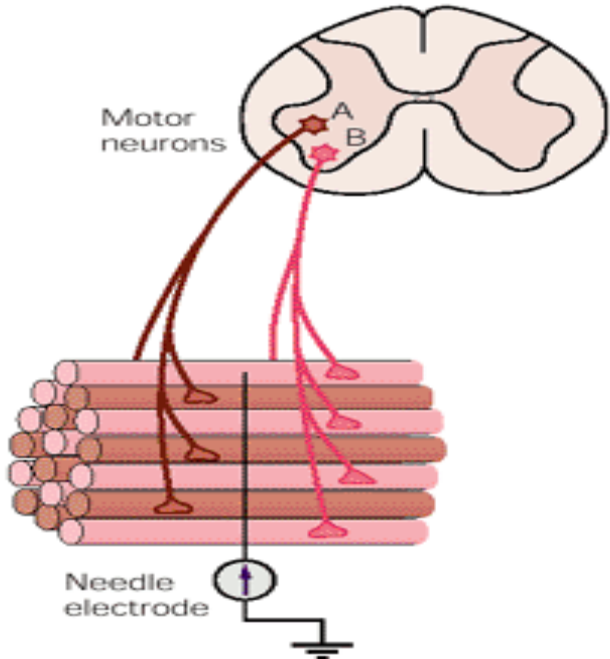
# Functional Effects of LMNL

## 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

A Normal muscle



Rest



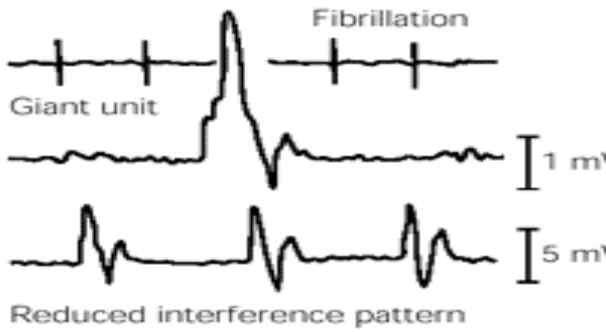
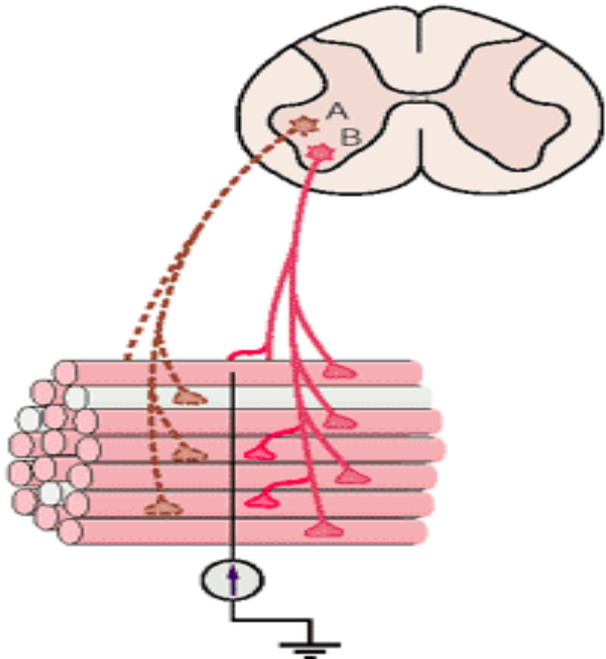
Slight contraction



Maximal contraction



B Denervated muscle





# 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
<b>Cause</b>	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
<b>Characters</b>		
<b>1- Paralysis</b>	<ul style="list-style-type: none"><li>* On the opposite side of the body (contralateral)</li><li>* Widespread affecting half of the face, upper &amp; lower limbs</li><li>* Poor recovery</li></ul>	<ul style="list-style-type: none"><li>* On the same side of the lesion</li><li>* Localized to muscles supplied by the affected segment only</li><li>* Recovery may occur.</li></ul>

# UMNL and LMNL

	UMNL	LMNL
<b>2- Muscle tone</b>	<ul style="list-style-type: none"><li>* Hypertonia of the spastic type in the paralyzed muscle</li><li>* <u>Klasp</u> knife type: resistance to passive movement then sudden release</li><li>* Cause: loss of inhibitory effect of the cortical extrapyramidal area &amp; ↑↑ facilitatory impulses on the <math>\gamma</math> motor neurons <math>\Rightarrow</math> facilitation of stretch reflex</li></ul>	<ul style="list-style-type: none"><li>* Hypotonia or <u>Atonia</u>: Flaccid paralysis (loss of tone in paralyzed muscles)</li><li>* Cause: interruption of stretch reflex</li></ul>
<b>3- Deep reflexes</b>	<ul style="list-style-type: none"><li>* Exaggerated deep reflexes on the affected side: (e.g. knee &amp; ankle jerks).</li><li>* Clonus is present.</li><li>* Cause: release of stretch reflex from cerebral inhibition</li></ul>	<ul style="list-style-type: none"><li>* Absent deep reflexes in muscles supplied by the affected segments or motor nerves</li></ul>

# UMNL and LMNL

	UMNL	LMNL
<b>4- Superficial reflex</b>	<ul style="list-style-type: none"> <li>* Lost on the affected side.</li> <li>* Cause: loss of supra-spinal facilitation</li> <li>* Abdominal &amp; <u>ceremasteric</u> reflexes: absent</li> <li>* The planter reflex <math>\Rightarrow</math> +ve <u>Babiniski's sign.</u></li> </ul>	<ul style="list-style-type: none"> <li>* Lost on the affected segments only</li> </ul>
<b>5- Muscle wasting</b>	<ul style="list-style-type: none"> <li>* Not significant</li> <li>* Cause: paralyzed muscles are still innervated and can contract reflexly.</li> <li>* Spasticity saves muscle from wasting.</li> </ul>	<ul style="list-style-type: none"> <li>* Marked (disuse atrophy)</li> <li>* Cause: muscles cannot contract neither reflexly nor voluntary</li> </ul>
<b>6- Fasciculations</b>	Absent	Present Visible spontaneous contractions of bundles of fibers in the affected ms



**THANKS**