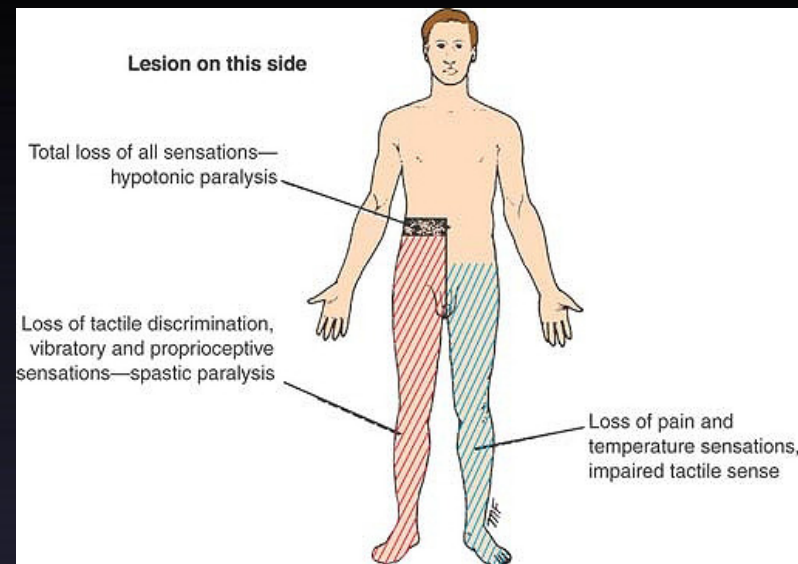


# UPPER AND LOWER MOTOR NEURON LESIONS



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King Saud University*

# OBJECTIVES

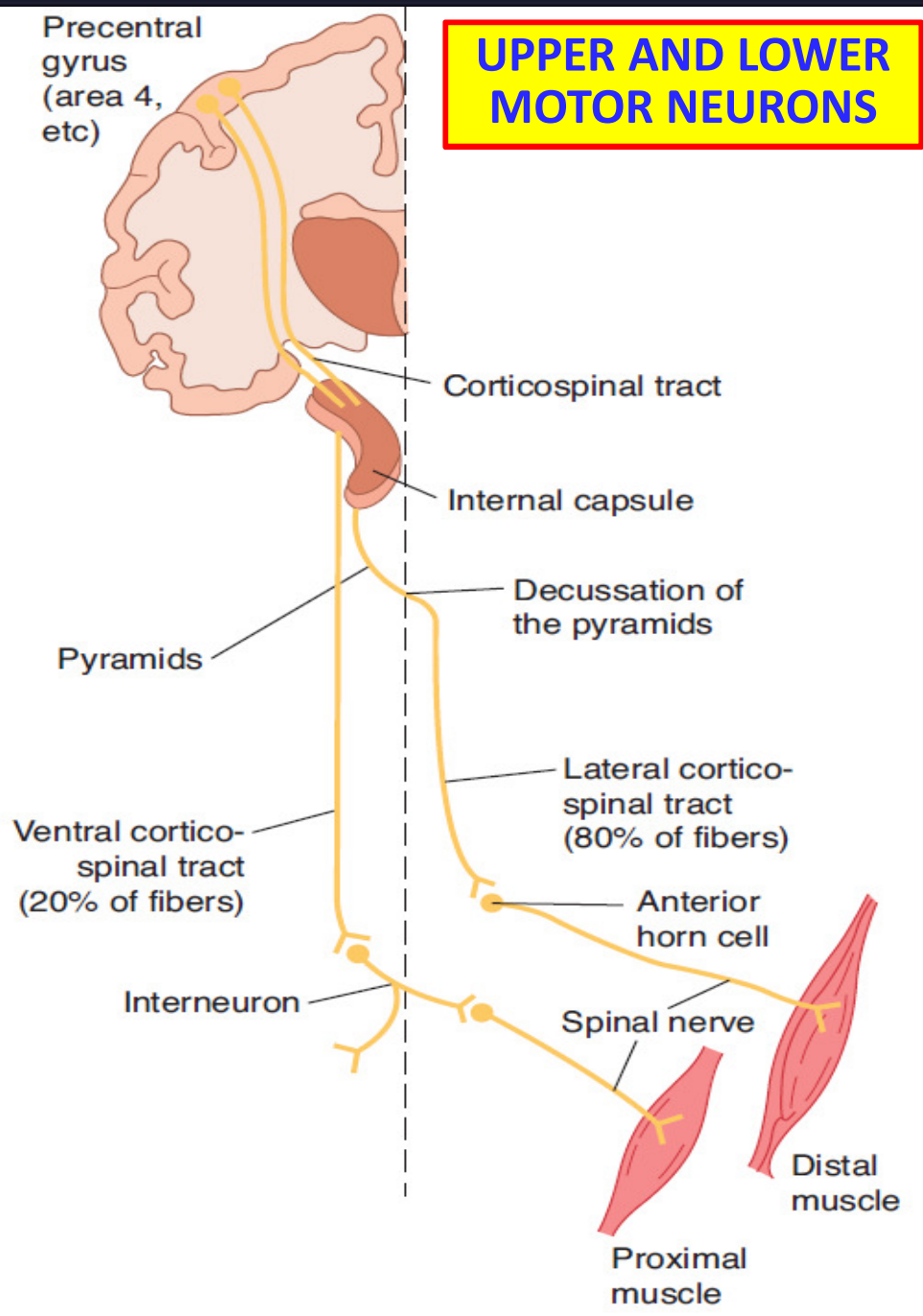
At the end of this lecture you should be able to

- ▶ Describe the functional anatomy of upper and lower motor neurons
- ▶ Describe and differentiate the features of upper and lower motor neuron lesions
- ▶ Explain features of Brown Sequard Syndrome
- ▶ Correlate the site of lesion with pattern of loss of sensations
- ▶ Describe facial, bulbar and pseudobulbar palsy

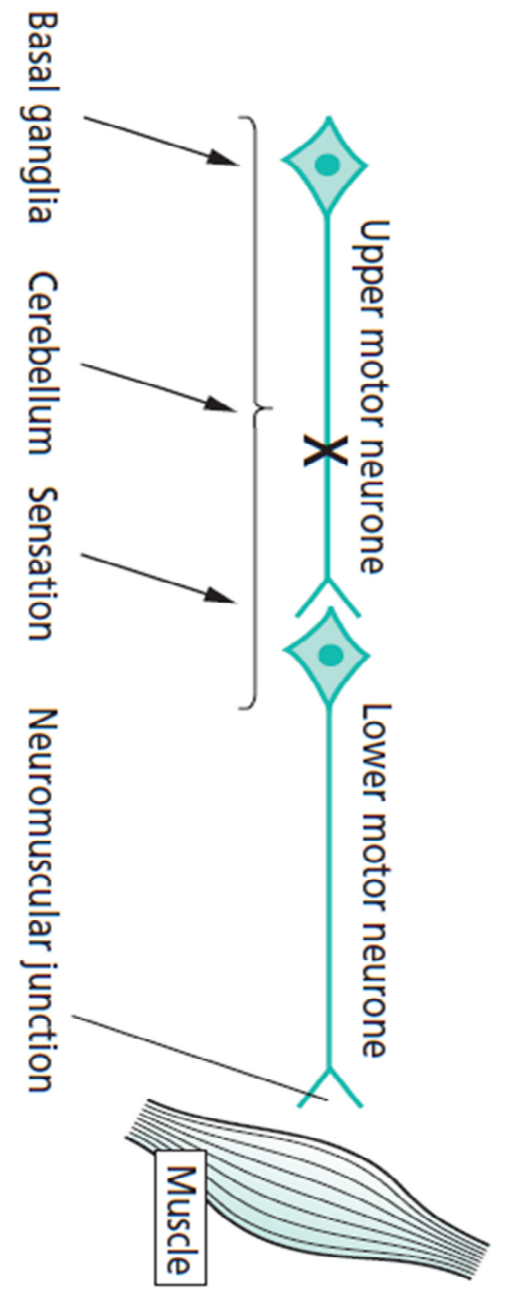




# UPPER AND LOWER MOTOR NEURONS



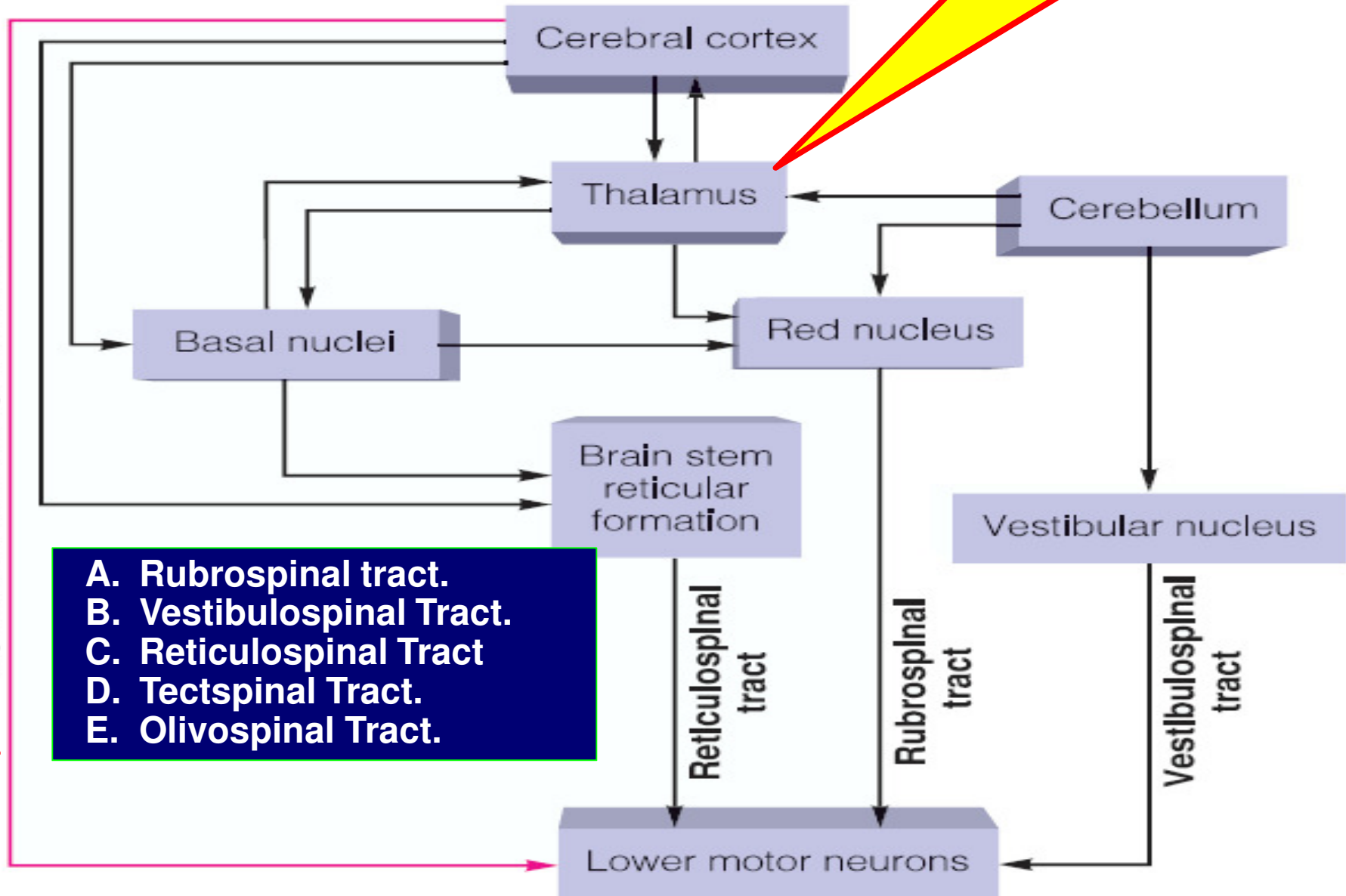
## Upper motor neurone



**UMNs control lower LMNs through two different pathways**

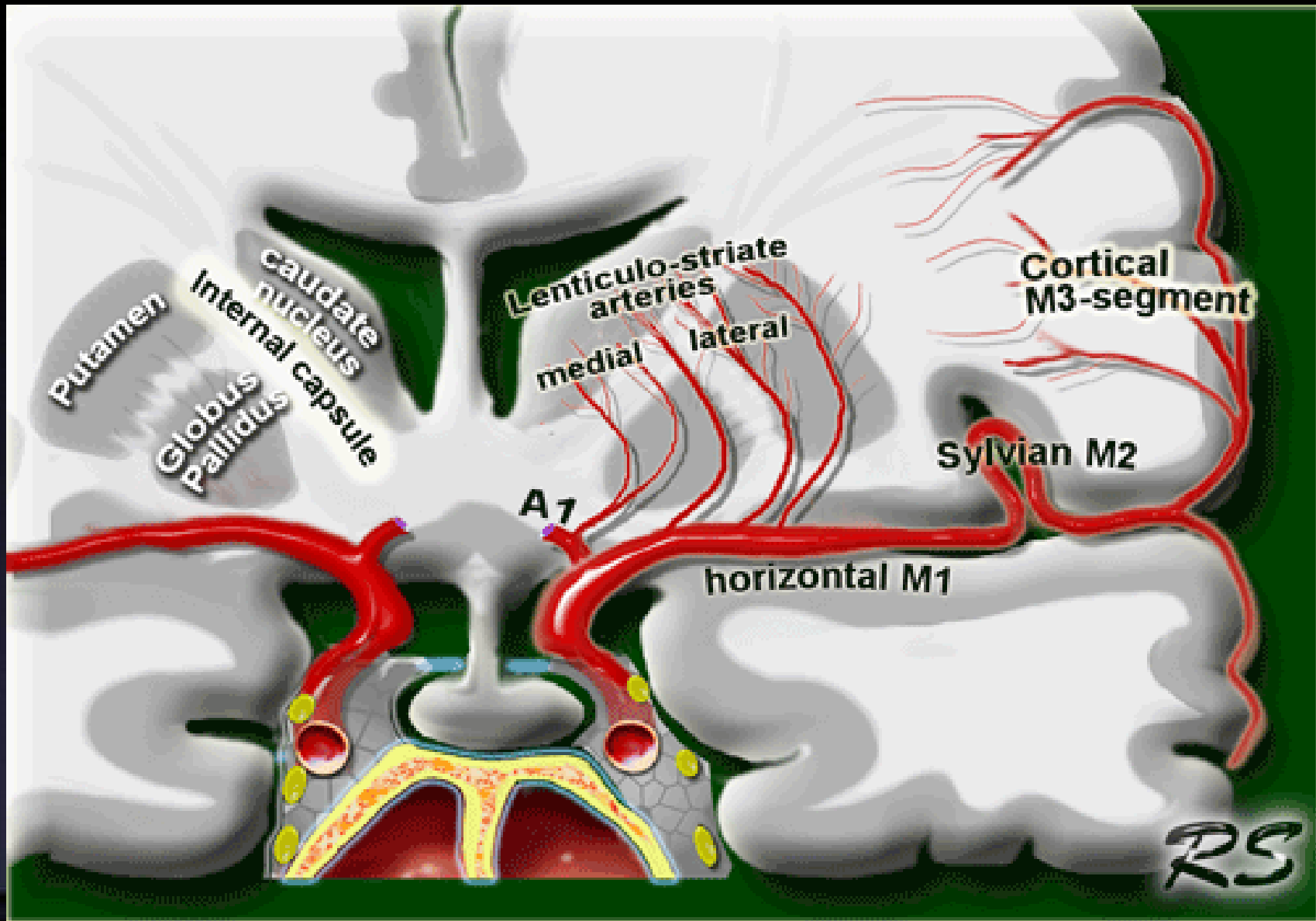
**Extrapyramidal motor system**

**Pyramidal system**



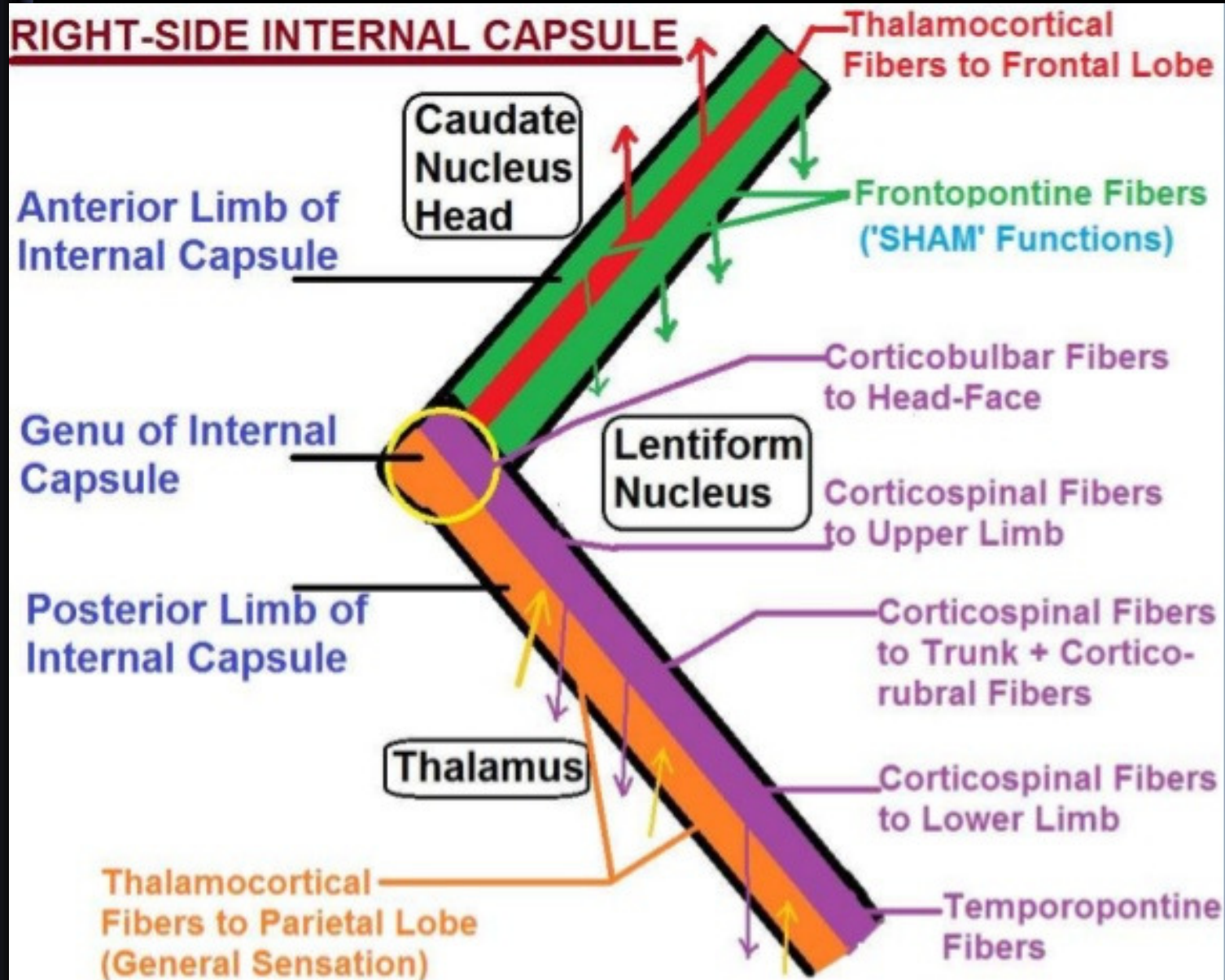
- A. Rubrospinal tract.
- B. Vestibulospinal Tract.
- C. Reticulospinal Tract
- D. Tectospinal Tract.
- E. Olivospinal Tract.

# Lenticulostriate Arteries from the middle cerebral artery



# INTERNAL CAPSULE

## RIGHT-SIDE INTERNAL CAPSULE



Internal Capsule – Tracts

# Causes of UMNL & LMNL

## Upper Motor Neuron Lesion

Can result from

- Cerebral stroke by hemorrhage, thrombosis or embolism
- Spinal cord transection or hemisection (Brown-Sequard syndrome)

## Lower Motor Neuron Lesion

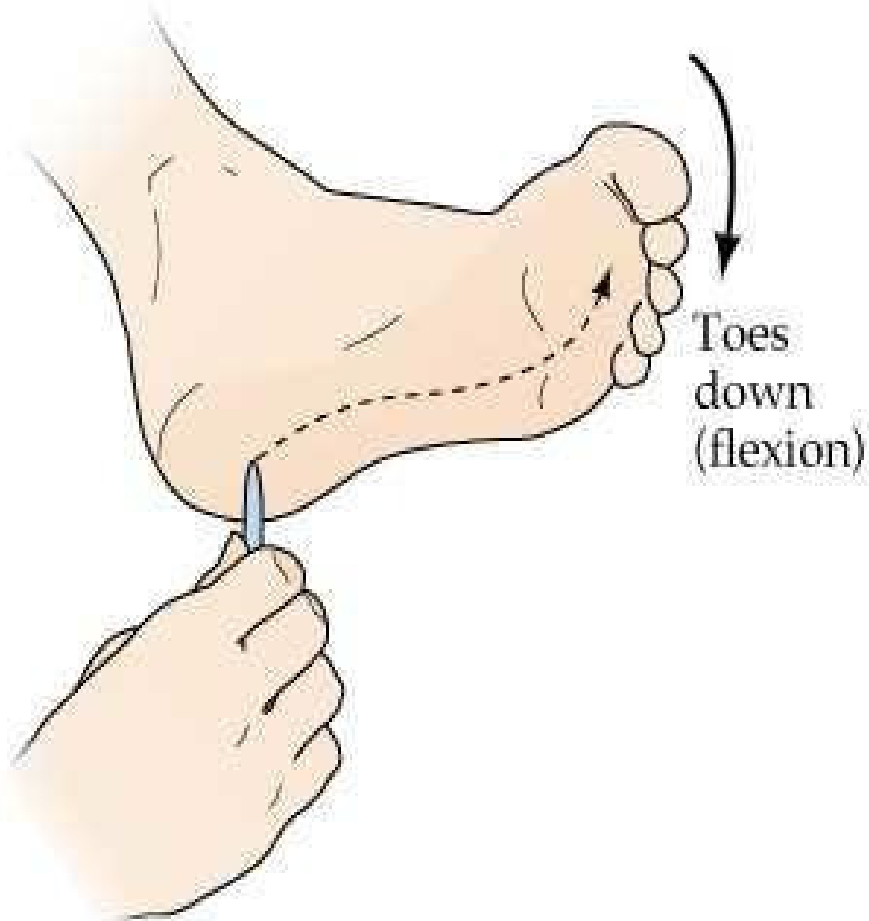
Can result from

- Anterior horn cell lesions (e.g. poliomyelitis, motor neuron disease)
- Spinal root lesions or peripheral nerve lesion (e.g. nerve injury by trauma or compressive lesion)

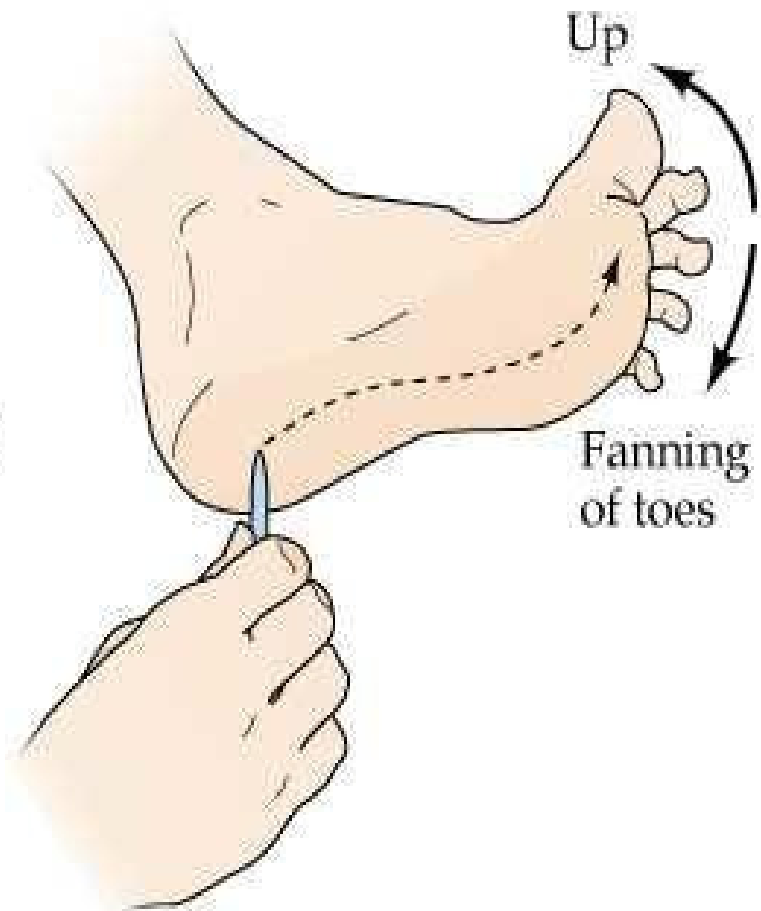


# COMAPRISON UPPER & LOWER MOTOR NEURON LESIONS

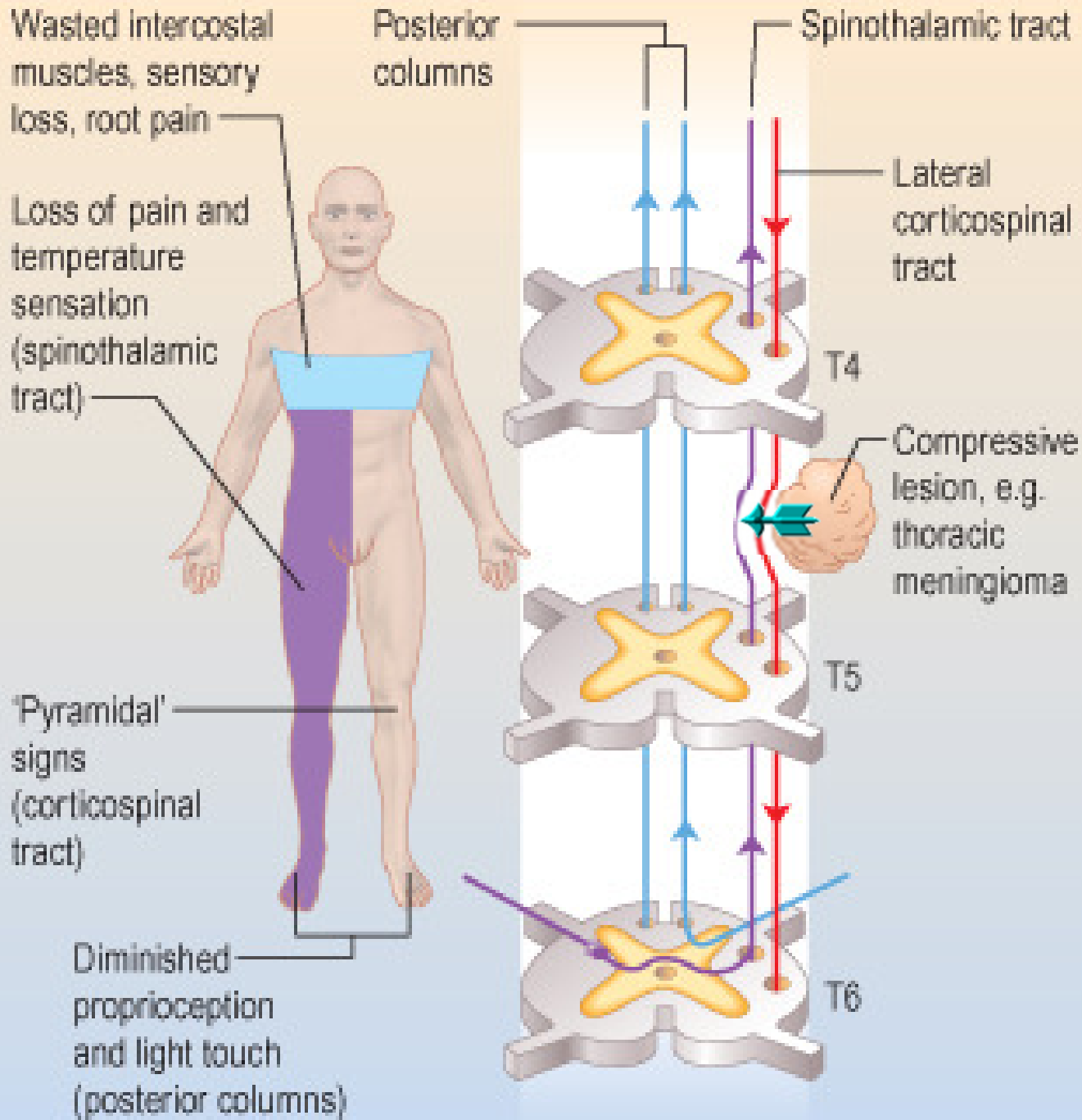
	<u>UMN LESION</u>	<u>LMN LESION</u>
Pattern	Paralysis affect movements	Individual muscle or group of muscles are affected.
Wasting	not pronounced (70-80)	Pronounced (20-30)
Tone	Spasticity Muscles hypertonic (Clasp Knife).	Tendon reflexes diminished or absent.
Tendon reflexes	Brisk / increased	diminished or absent.
Superficial reflexes	Absent	Absent
NCV	Normal	Decreased
Denervation potentials (Fibrillations) [ON EMG ONLY]	Absent	Present
Fasciculations (Visible)	Absent	Present
Trophic changes	Less	Pronounced in skin & Nails
clonus	Present	Absent
Babinski's sign	Extensor plantar response (Positive)	flexor or absent plantar responses



Normal plantar response



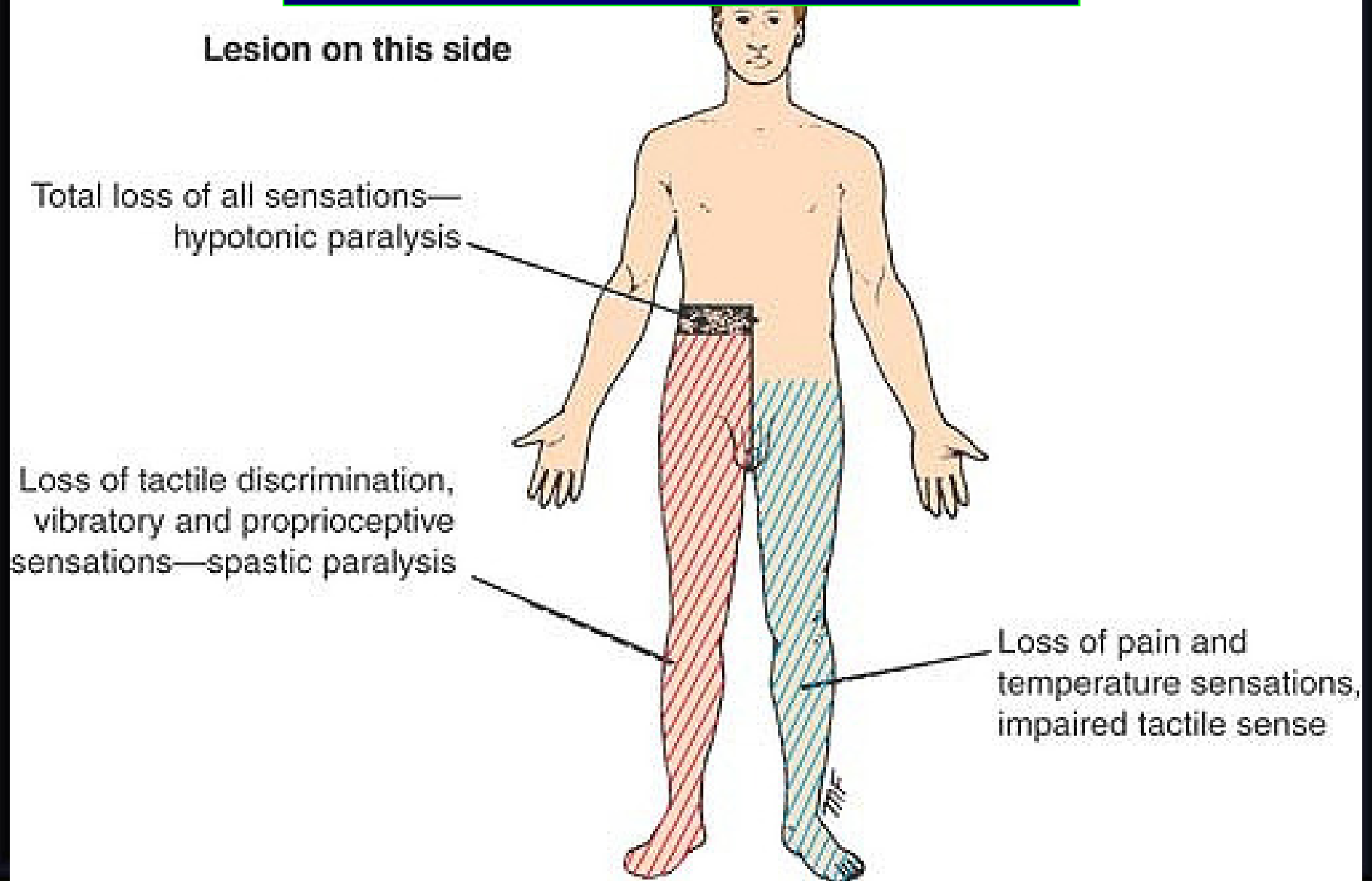
Extensor plantar response  
(Babinski sign)



# Brown Sequard syndrome

## HEMISECTION OF SPINAL CORD

Lesion on this side



# 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)

1. **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.

2. **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.

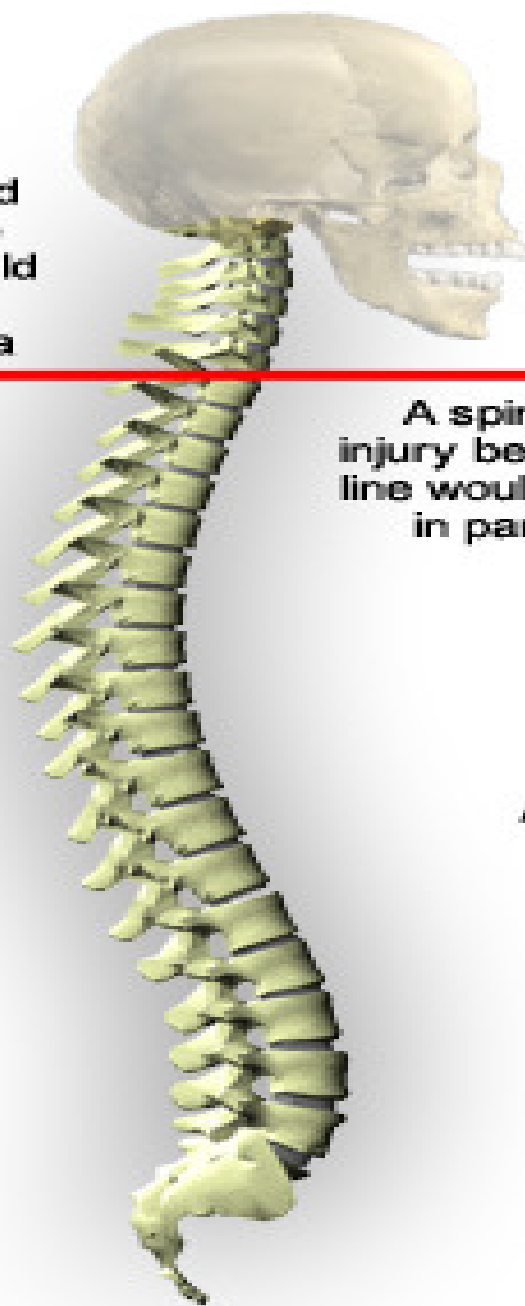
3. **Ipsilateral band of cutaneous anesthesia** in the segment of the lesion. This results from the destruction of the **posterior root** and its entrance into the spinal cord at the level of the lesion.

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

5. **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.

6. **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.

A spinal cord injury above this line would result in quadraplegia



A spinal cord injury below this line would result in paraplegia

Posterior  
(Rear)

Anterior  
(Front)

[www.apparelized.com](http://www.apparelized.com)

**Upper cervical cord lesions produce quadriplegia and weakness of the diaphragm**

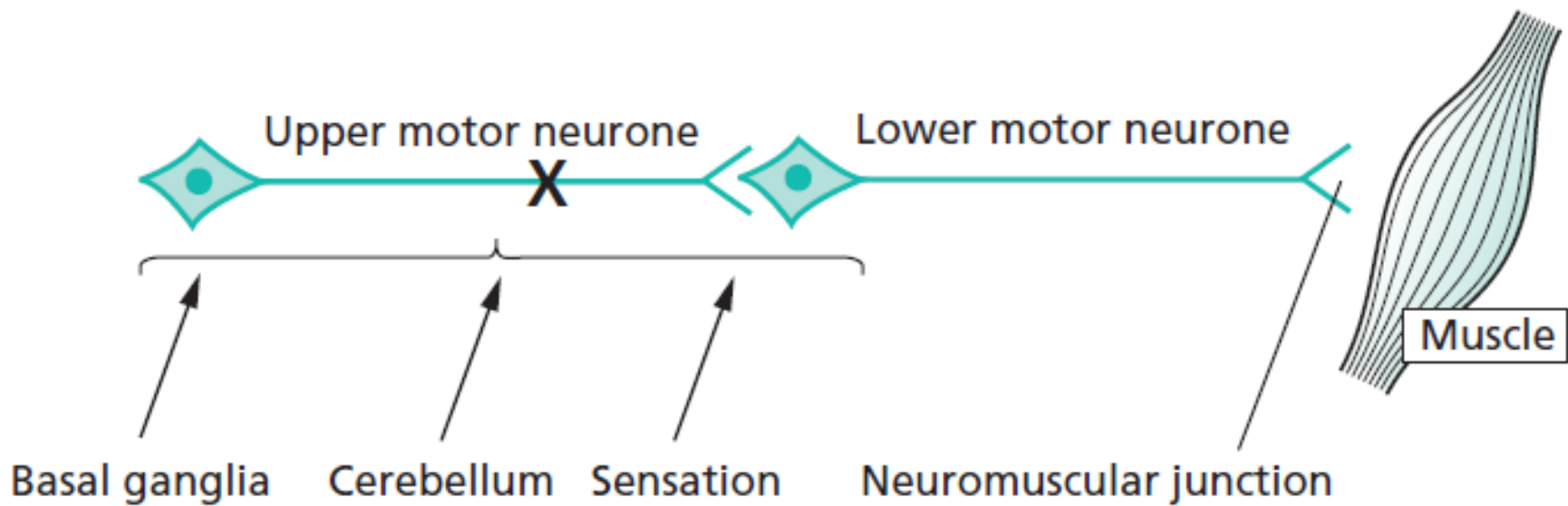
**Lesions at C4-C5 produce quadriplegia**

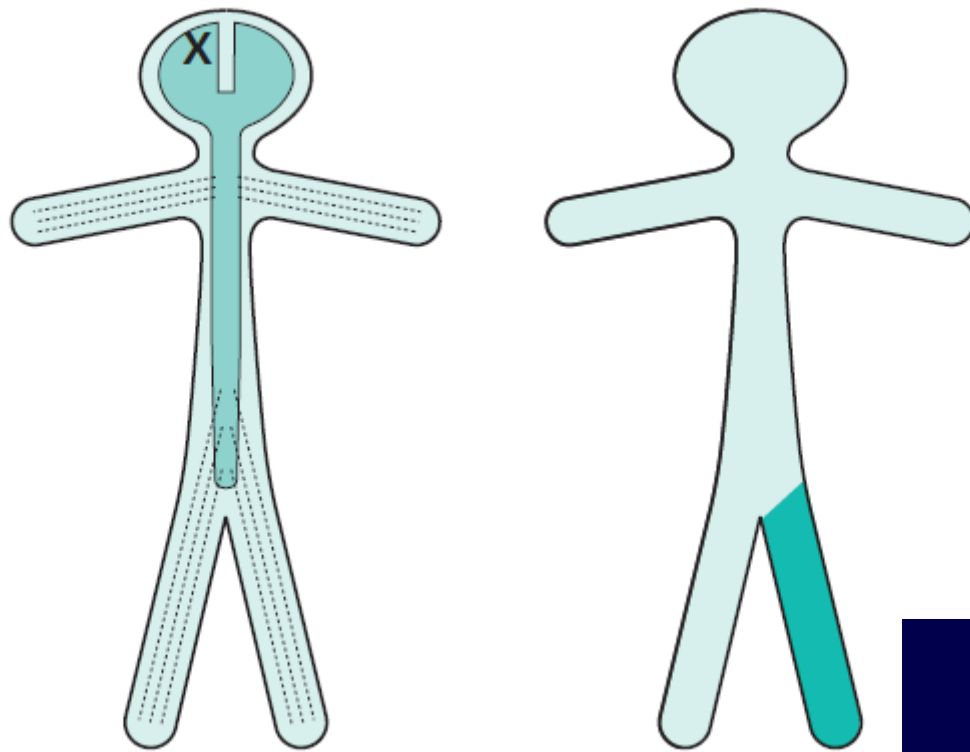
**Hemiparesis means weakness**

**Hemiplegia means total paralysis**



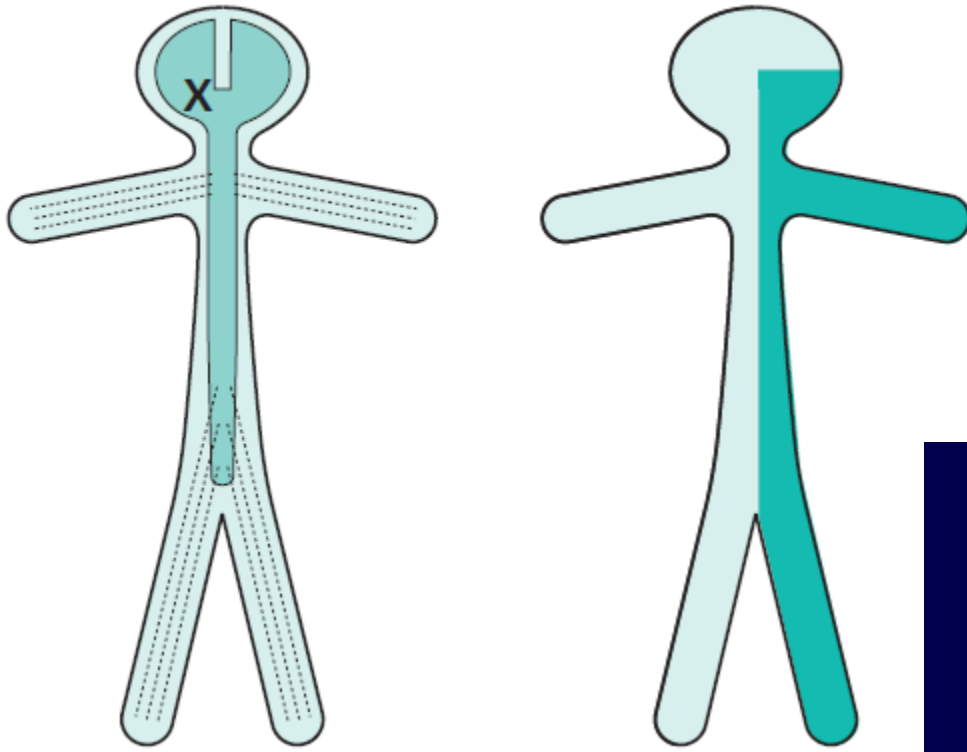
## Upper motor neurone





## Contralateral Monoparesis

**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.



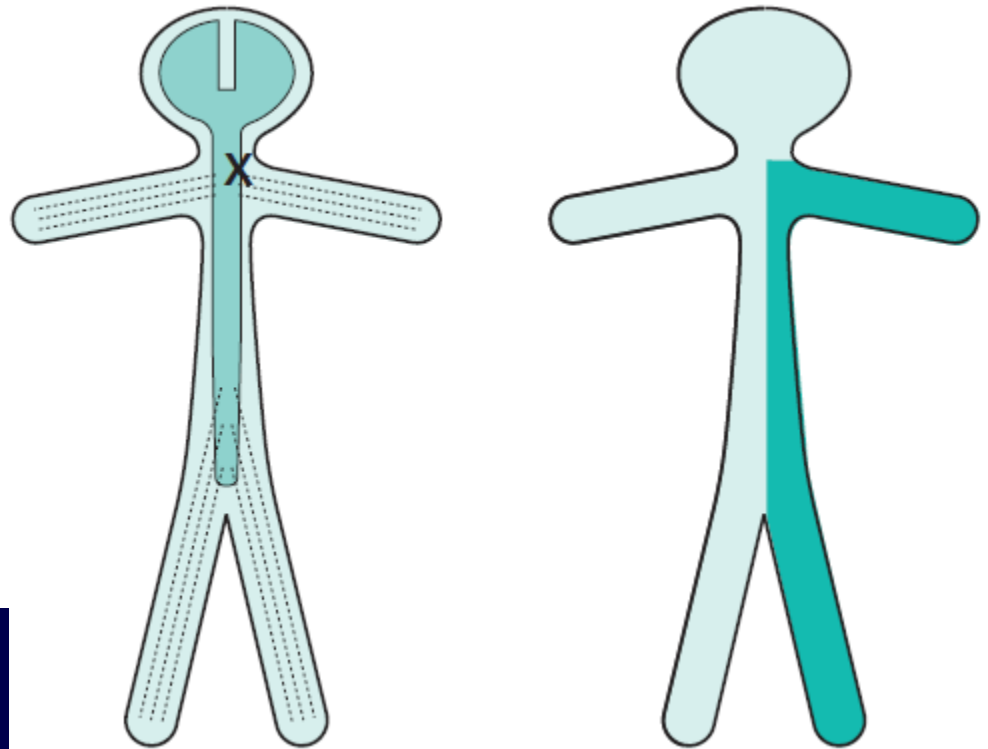
## Contralateral Hemiparesis

### 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.

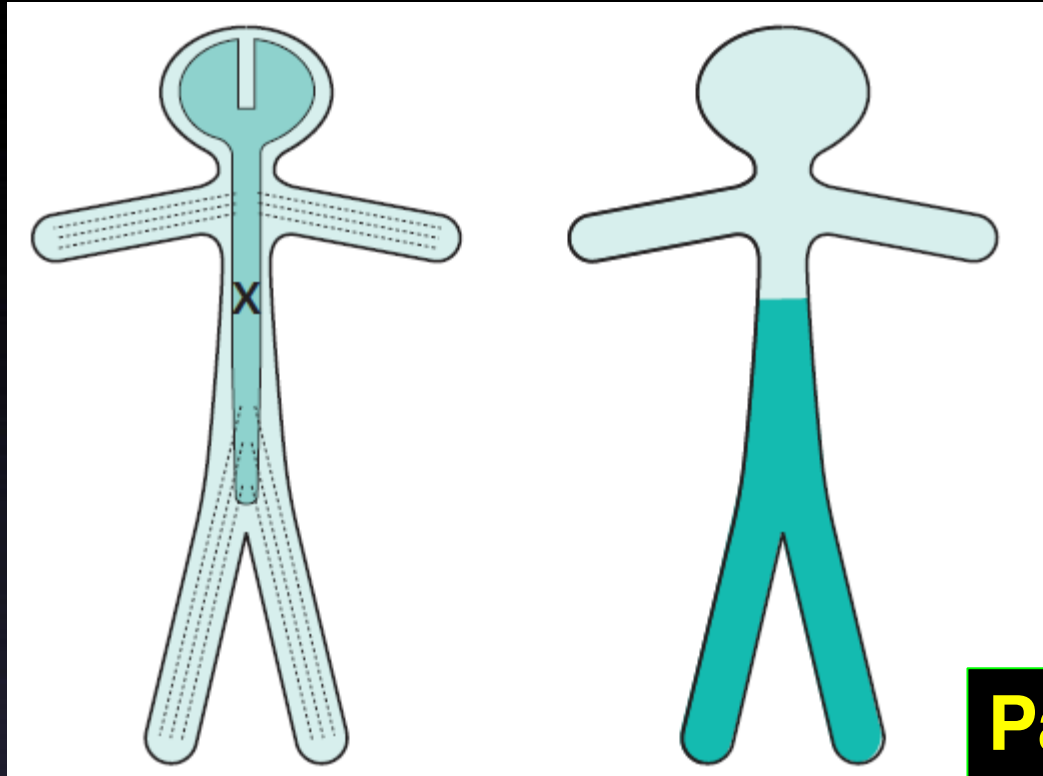
### **Ipsilateral hemiparesis**

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 neuron signs in both legs, often asymmetrically since the pathology rarely affects both sides of the spinal cord equally.

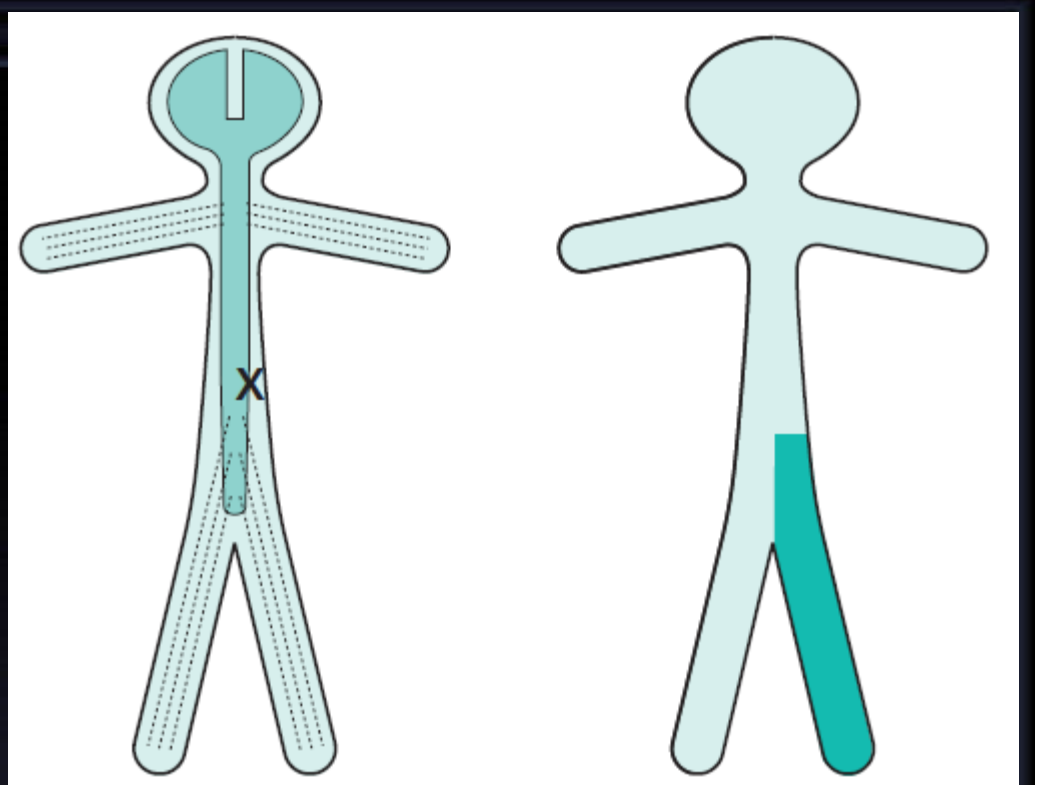


**Paraparesis**

**Paraparesis**, if the lesion is at or below the cervical portion of the spinal cord.

## **Ipsilateral Monoparesis**

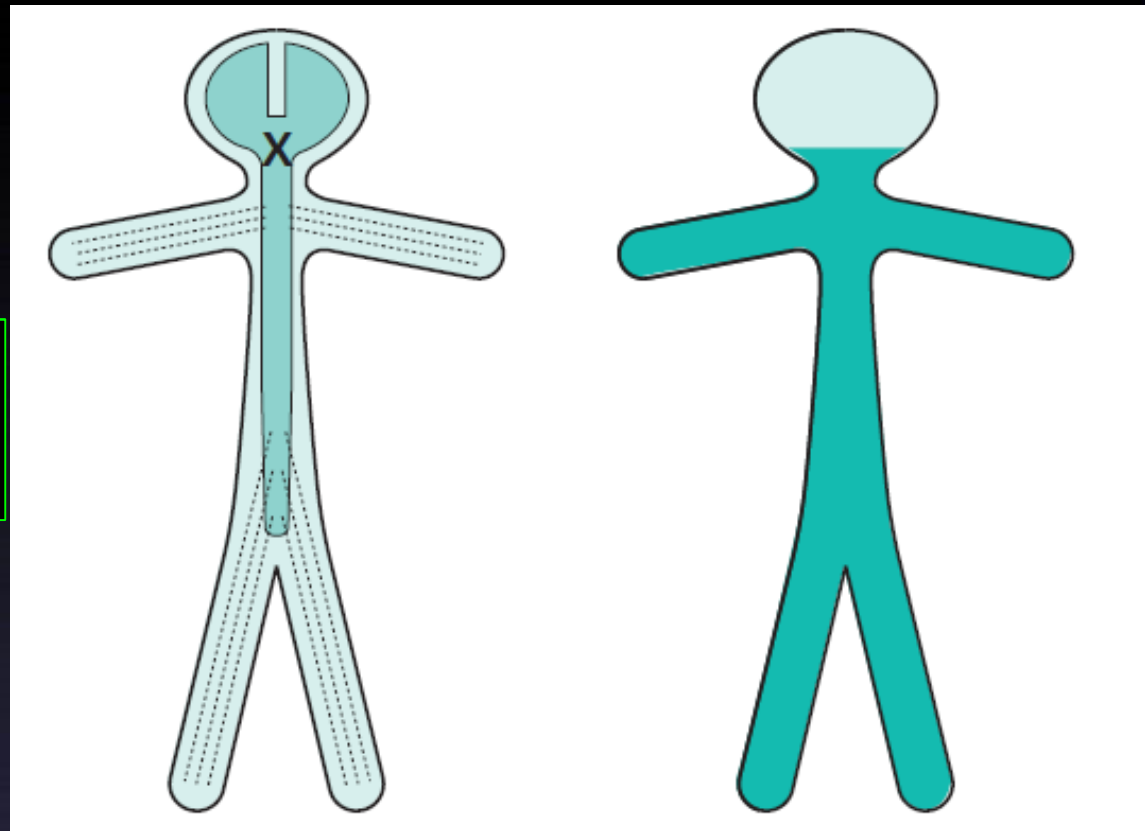
A unilateral lesion in the spinal cord below the level of the neck produces upper motor neurone weakness in one leg. There may be posterior column (position sense) sensory loss in the same leg, and spinothalamic (pain and temperature) sensory loss in the contralateral leg. This is known as dissociated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



**Ipsilateral Monoparesis**

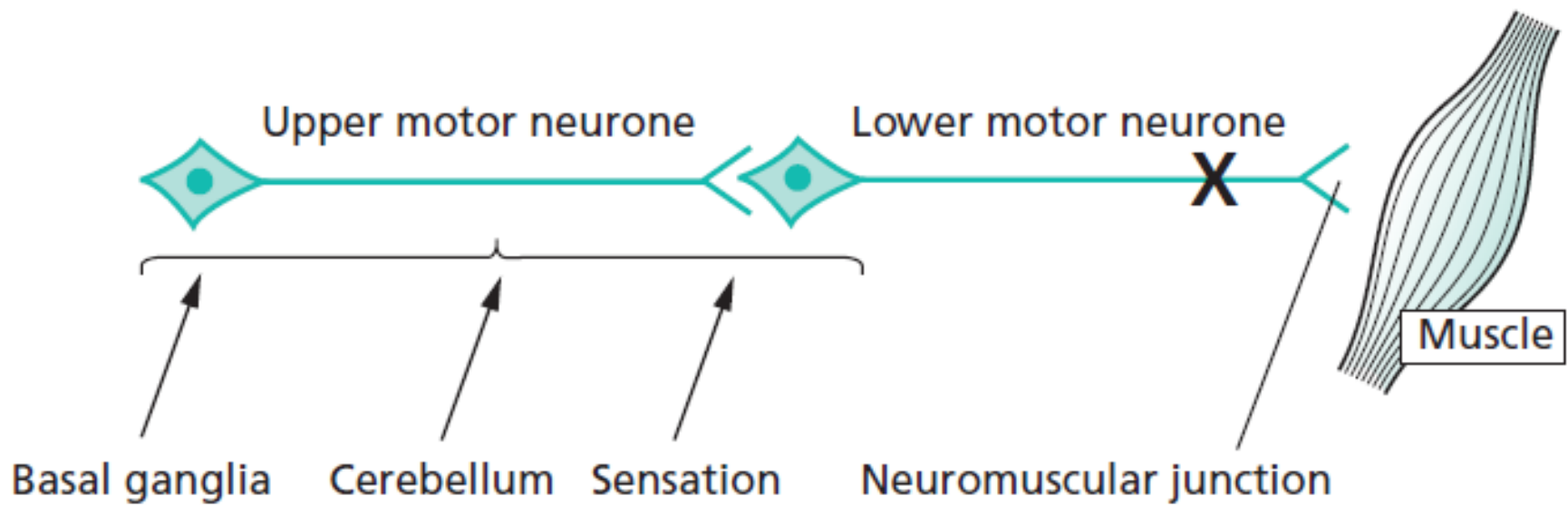
A spinal cord lesion more usually causes upper motor neurone signs in both legs, often asymmetrically since the pathology rarely affects both sides of the spinal cord equally.

## Tetraparesis or Quadriparesis



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

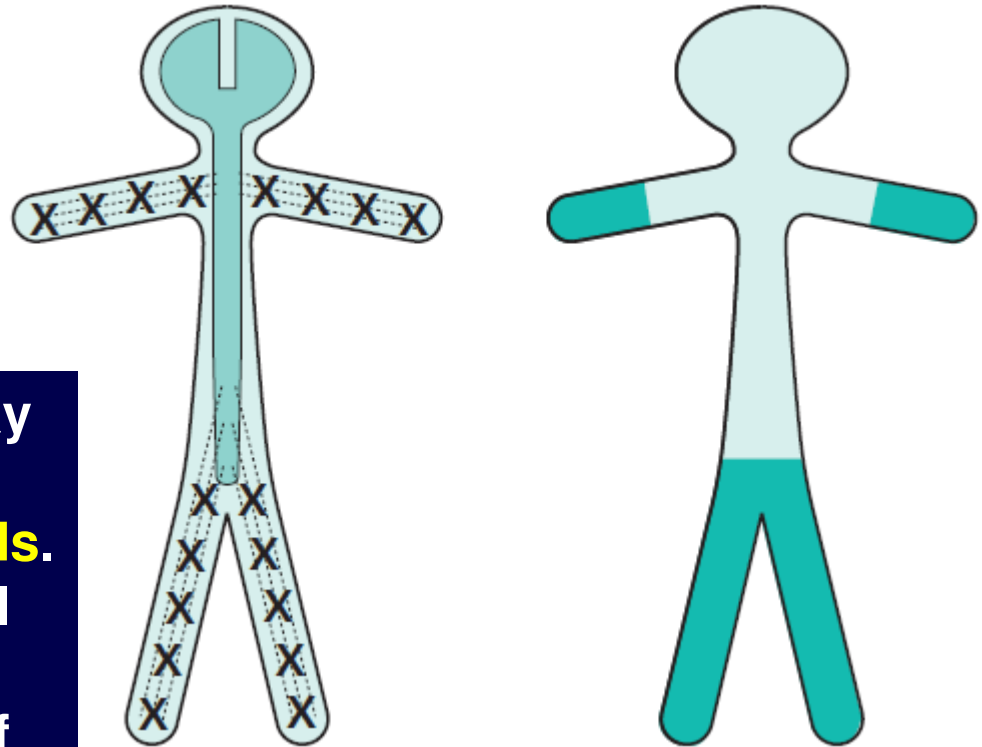
## Lower motor neurone



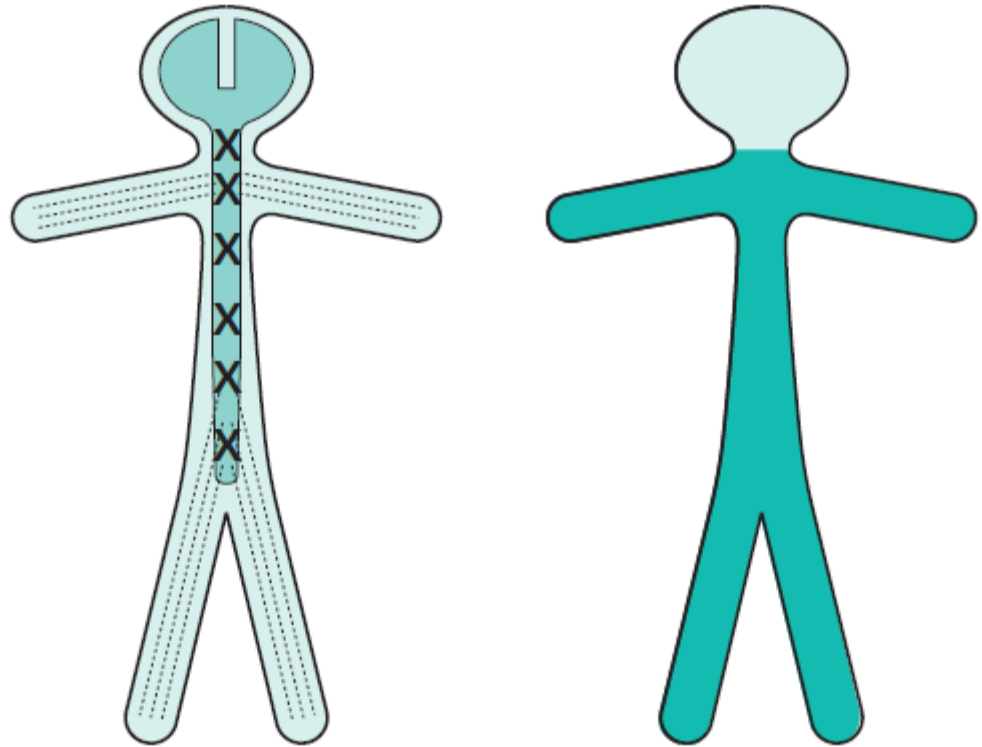


## Generalized LMN weakness

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 neurones are usually simultaneously involved. The LMN weakness and sensory loss tend to be most marked distally in the limbs.



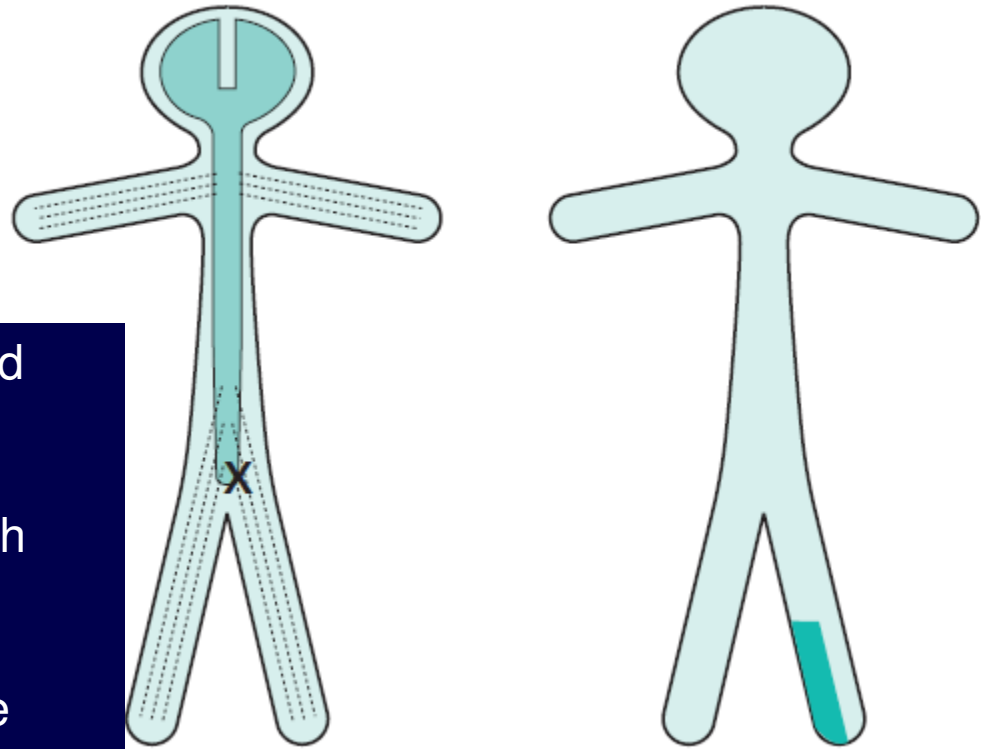
## Generalized LMN weakness



Generalized LMN weakness may result from pathology affecting the LMNs throughout the spinal cord and brainstem, as in motor neurone disease or poliomyelitis.

Generalized limb weakness (proximal and distal), trunk and bulbar weakness characterize this sort of LMN disorder.

**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.



**LMN weakness of  
one spinal root**

# 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

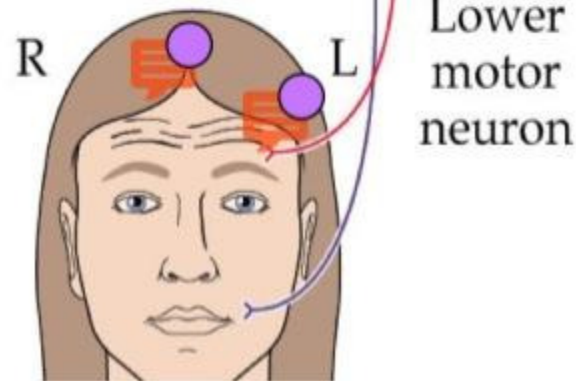
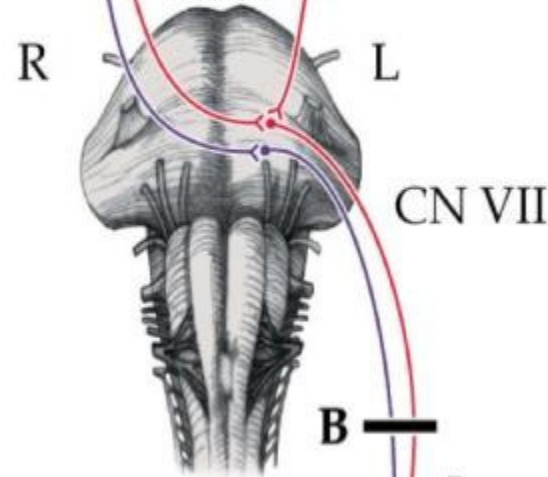
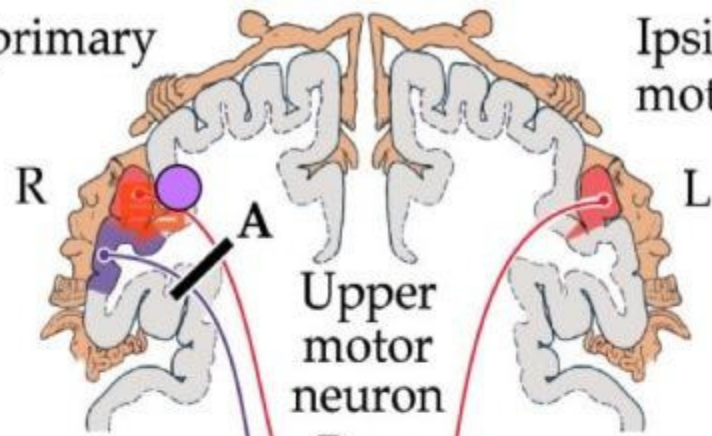
## **VII<sup>th</sup> Cranial Nerve lesion**

**UMN VII<sup>th</sup> 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.

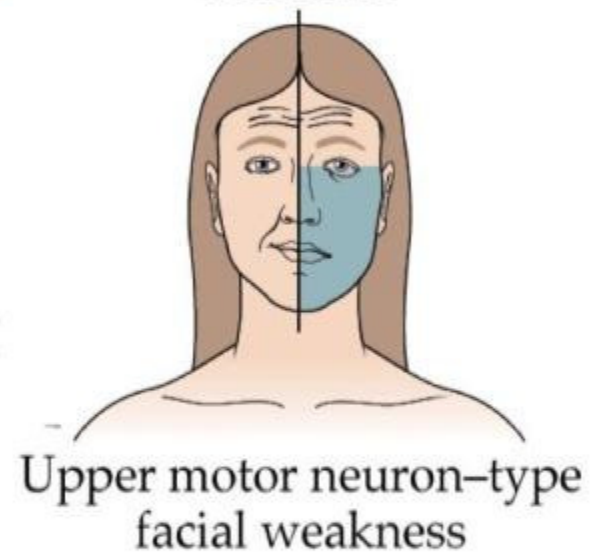
**LMN VII<sup>th</sup> Cranial Nerve lesion** causes weakness (ipsilateral) of all facial expression muscles. The angle of the mouth falls; unilateral dribbling develops. Frowning (frontalis) and eye closure are weak. Corneal exposure and ulceration occur if the eye does not close during sleep.

Contralateral primary motor cortex

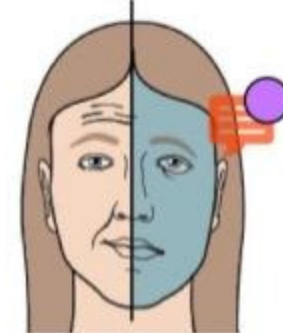
Ipsilateral primary motor cortex



Lesion A



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.

# Intramedullary and Extramedullary Syndromes

Extramedullary lesions, **radicular pain** is often prominent, and there is **early sacral sensory loss** (lateral spinothalamic tract) and **spastic weakness in the legs** (corticospinal tract) due to the superficial location of leg fibers in the corticospinal tract

**Early UMN signs**

Intramedullary lesions tend to produce **poorly localized burning pain** rather than radicular pain and **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**