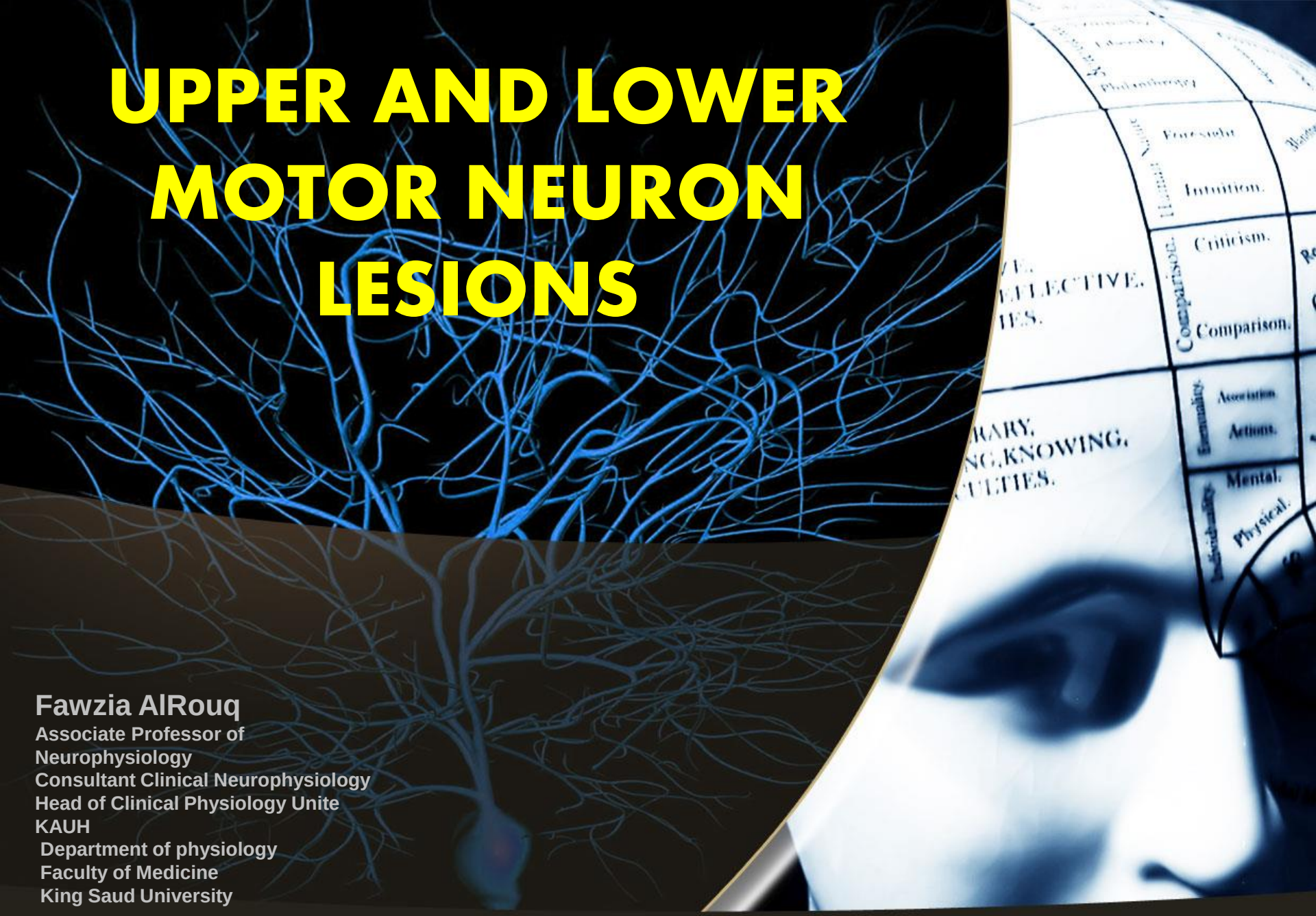
UPPER AND LOWER MOTOR NEURON LESIONS



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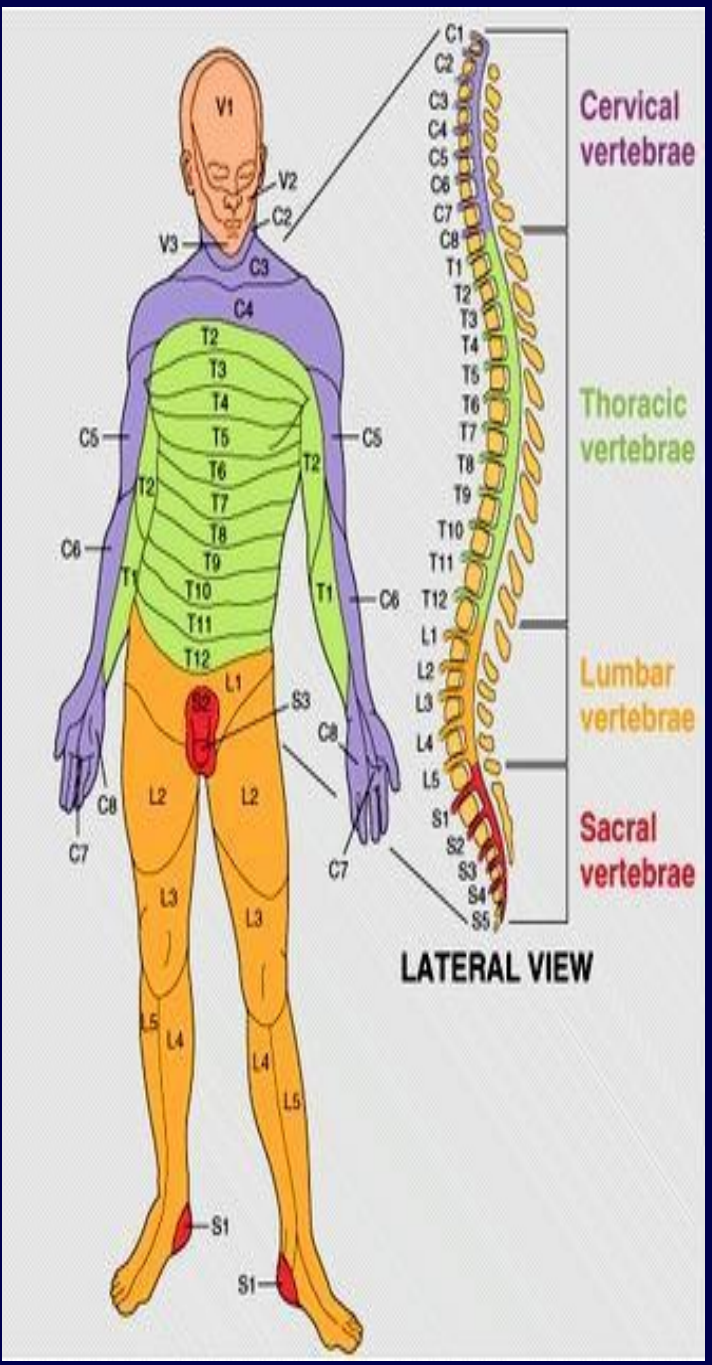
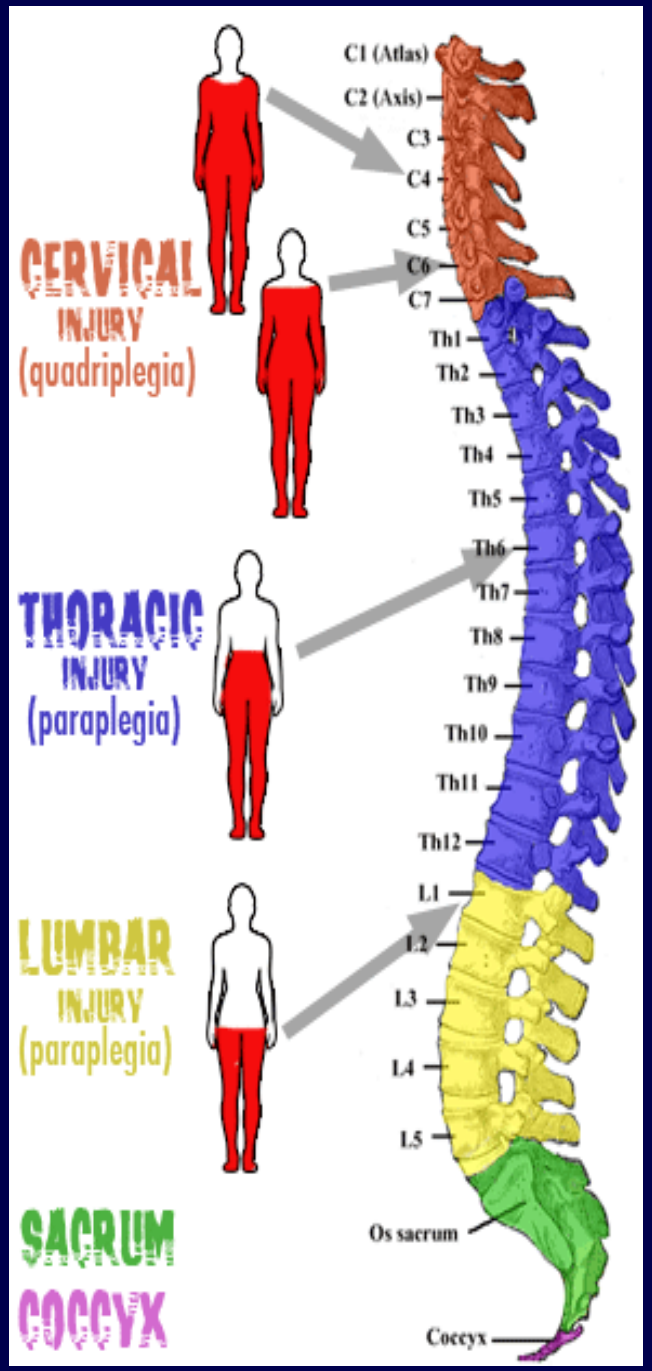
OBJECTIVES

At the end of this lecture you should be able to

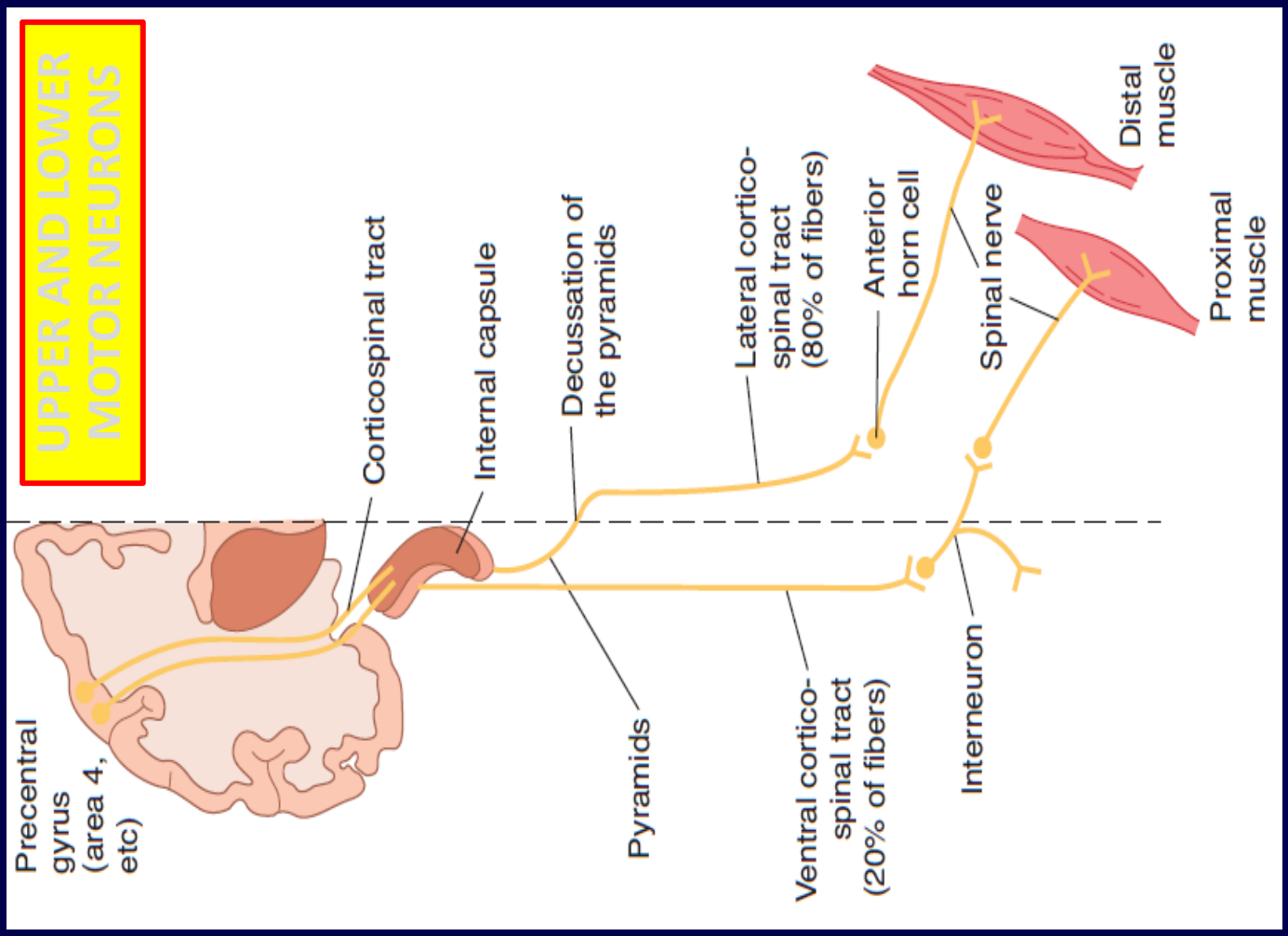
- Describe the functional anatomy of upper and lower motor neurons
- Describe and differentiate the features of upper and lower motor neuron lesions
- Explain features of Brown Sequard Syndrome
- Correlate the site of lesion with pattern of loss of sensations
- Describe facial, bulbar and pseudobulbar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

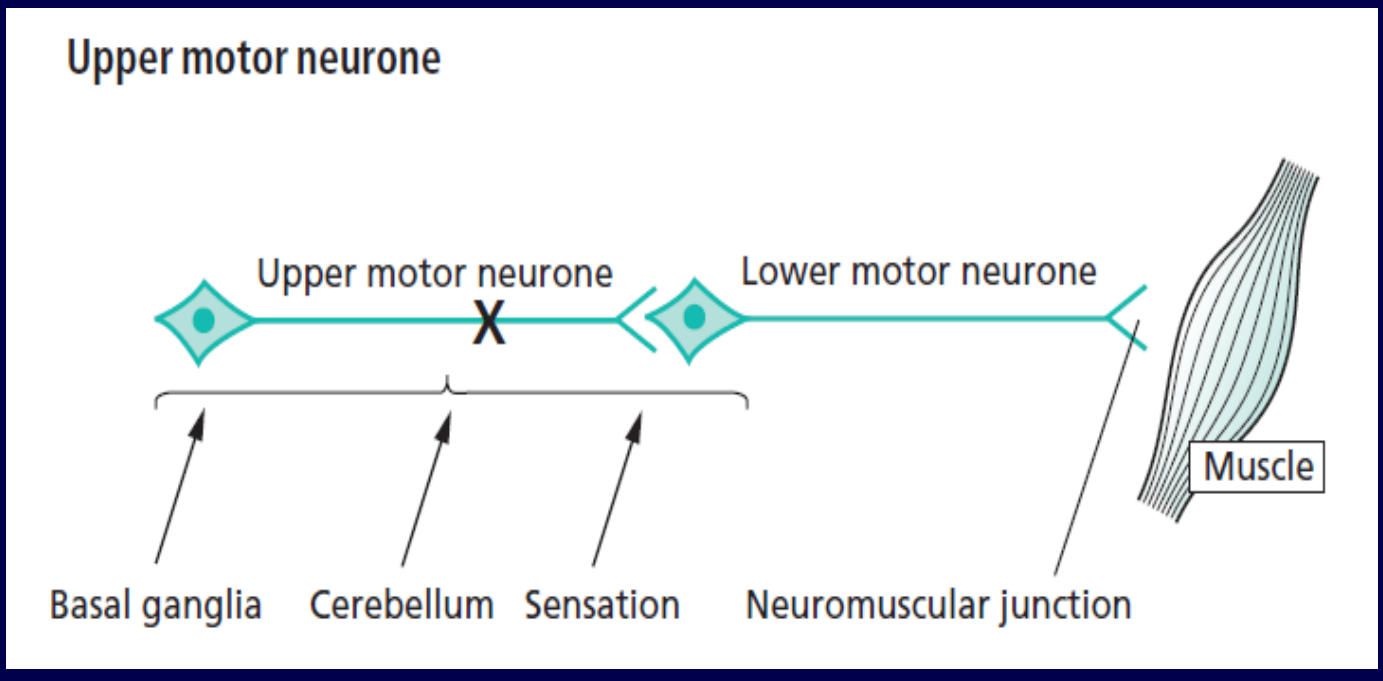




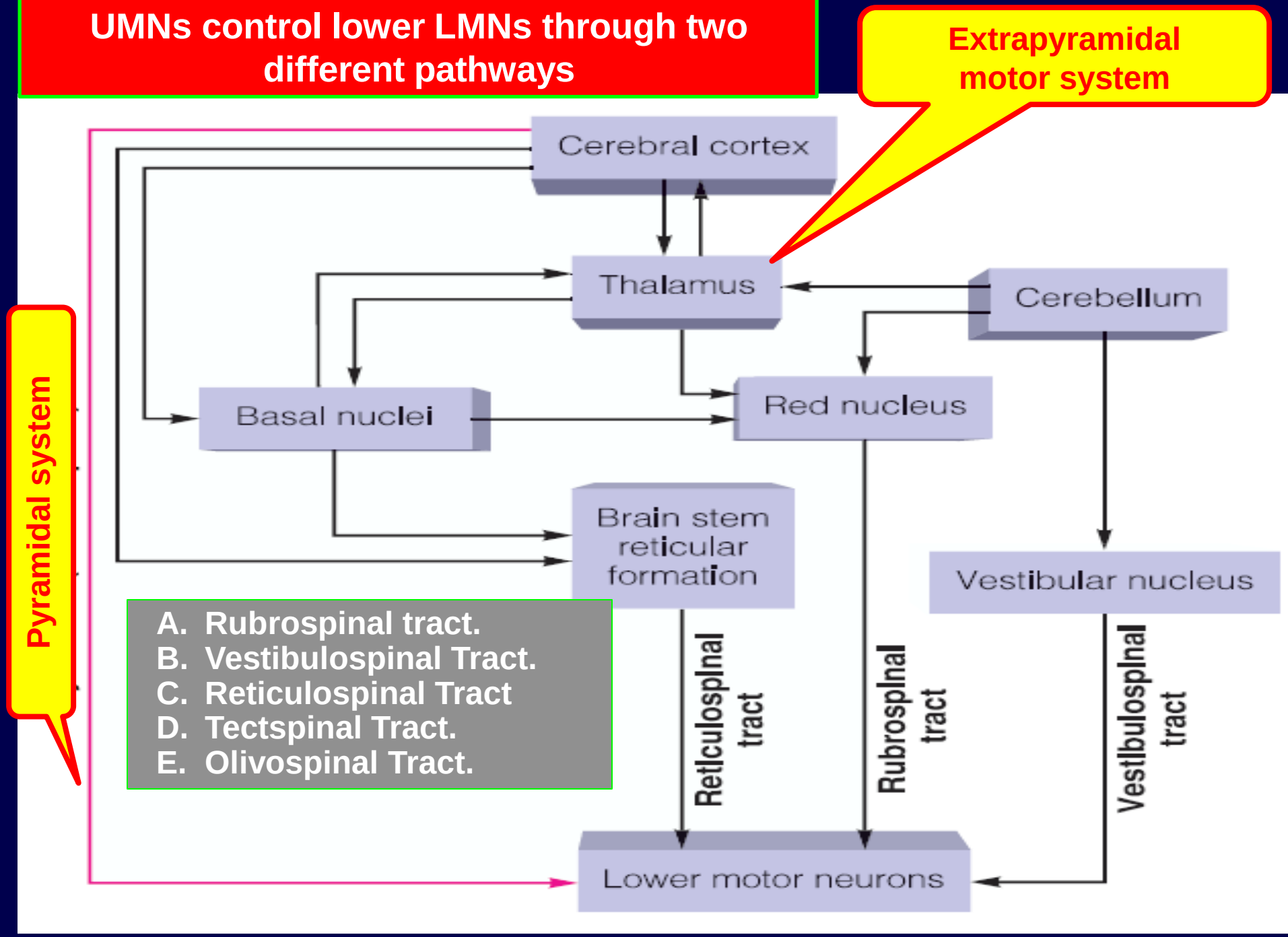
31 segments

Embryological development growth of cord lags behind → mature spinal cord ends at L1

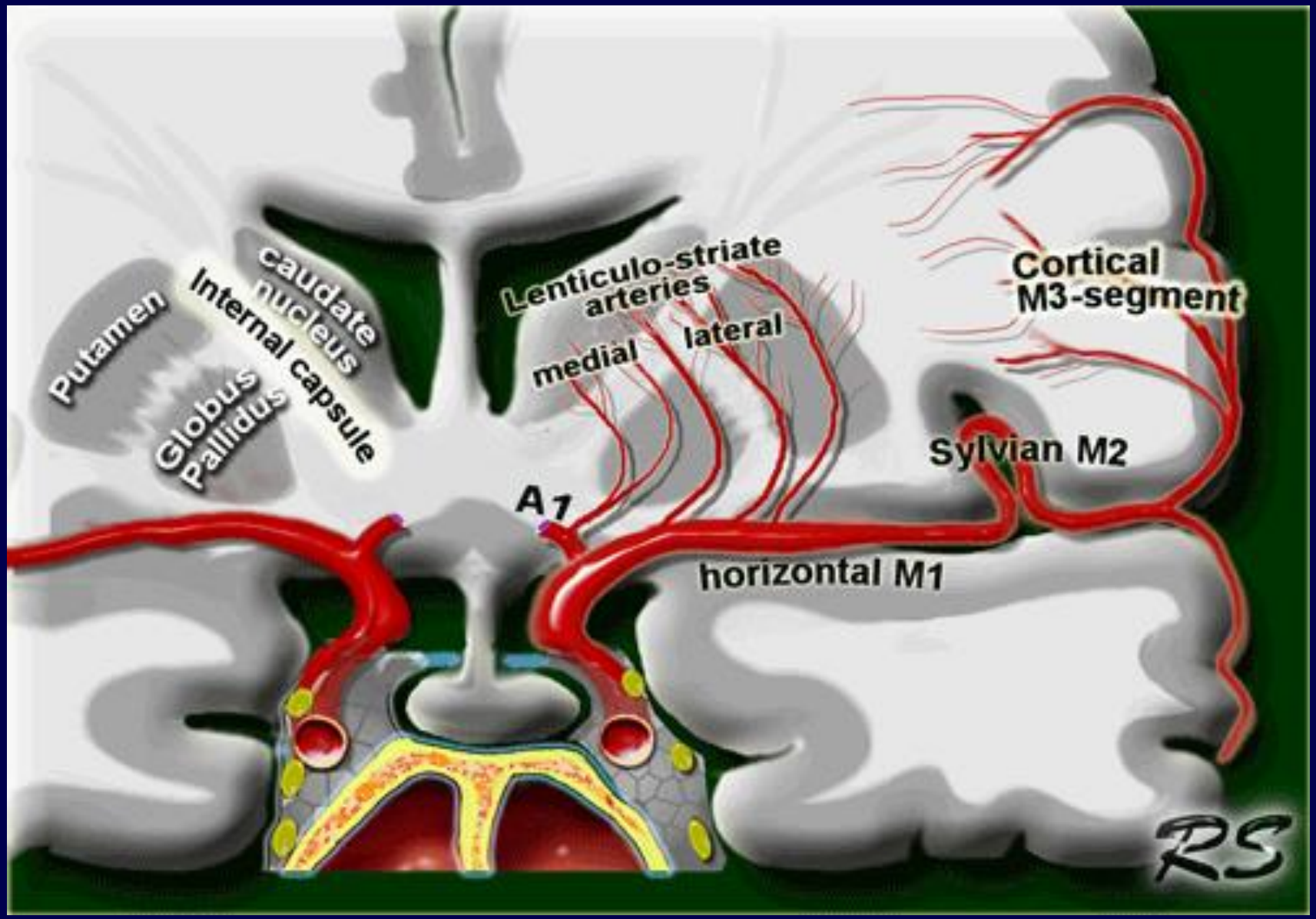

UPPER AND LOWER MOTOR NEURONS
Corticospinal tract
Internal capsule
Decussation of the pyramids
Lateral cortico-spinal tract (80% of fibers)
Anterior horn cell
Distal muscle
Spinal nerve
Proximal muscle
Precentral gyrus (area 4, etc)
Pyramids
Ventral cortico-spinal tract (20% of fibers)
Interneuron


Upper motor neurone
Upper motor neurone
X
Lower motor neurone
Muscle
Basal ganglia
Cerebellum
Sensation
Neuromuscular junction

UMNs control lower LMNs through two different pathways



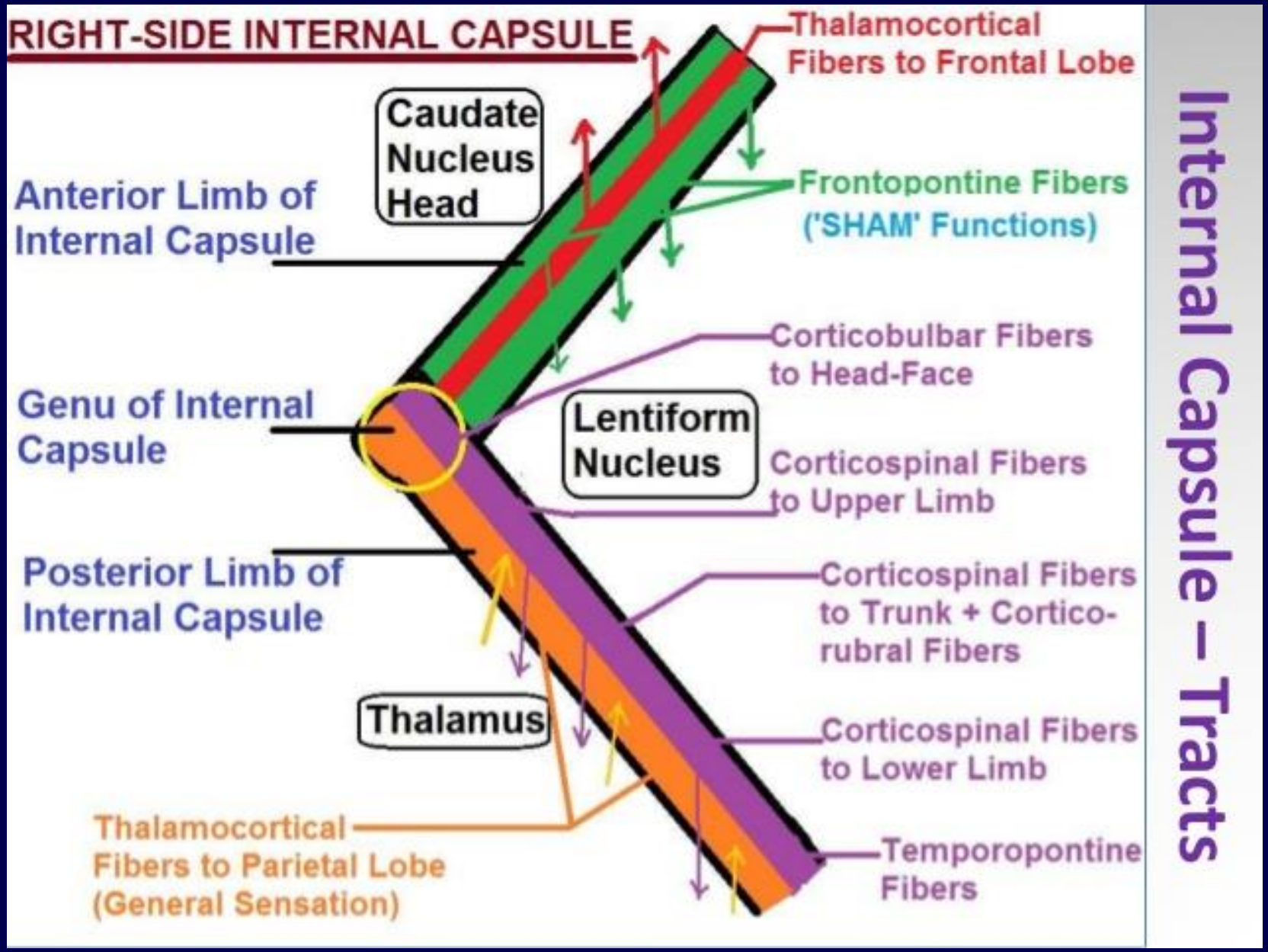
Lenticulostriate Arteries from the middle cerebral artery



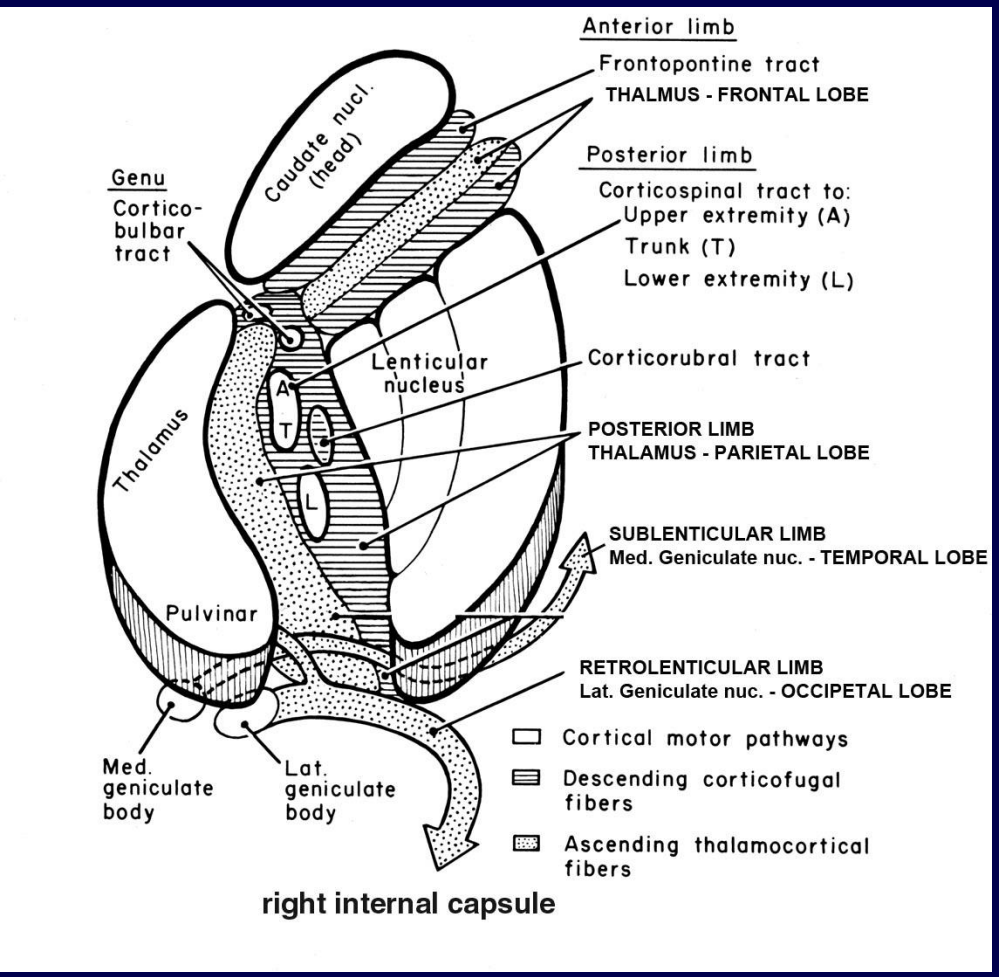
lenticulostriate arteries

The superior parts of both the anterior and posterior limbs and the genu of the **internal capsule** eht yb deilppus era **lenticulostriate arteries** era hcihw , M eht fo sehcnarb1 segment of the middle cerebral **artery**.

INTERNAL CAPSULE



The Right Internal Capsule



Reciprocal connections between thalamus and cortex are found in four limbs of the internal capsule:

- anterior limb with frontal lobe
- posterior limb with parietal lobe
- retrolenticular limb with occipital lobe
- sublenticular limb with temporal lobe

The genu contains the corticobulbar axons.
Corticospinal axons are in the posterior limb.
Corticopontine axons are in both the anterior and posterior limbs

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

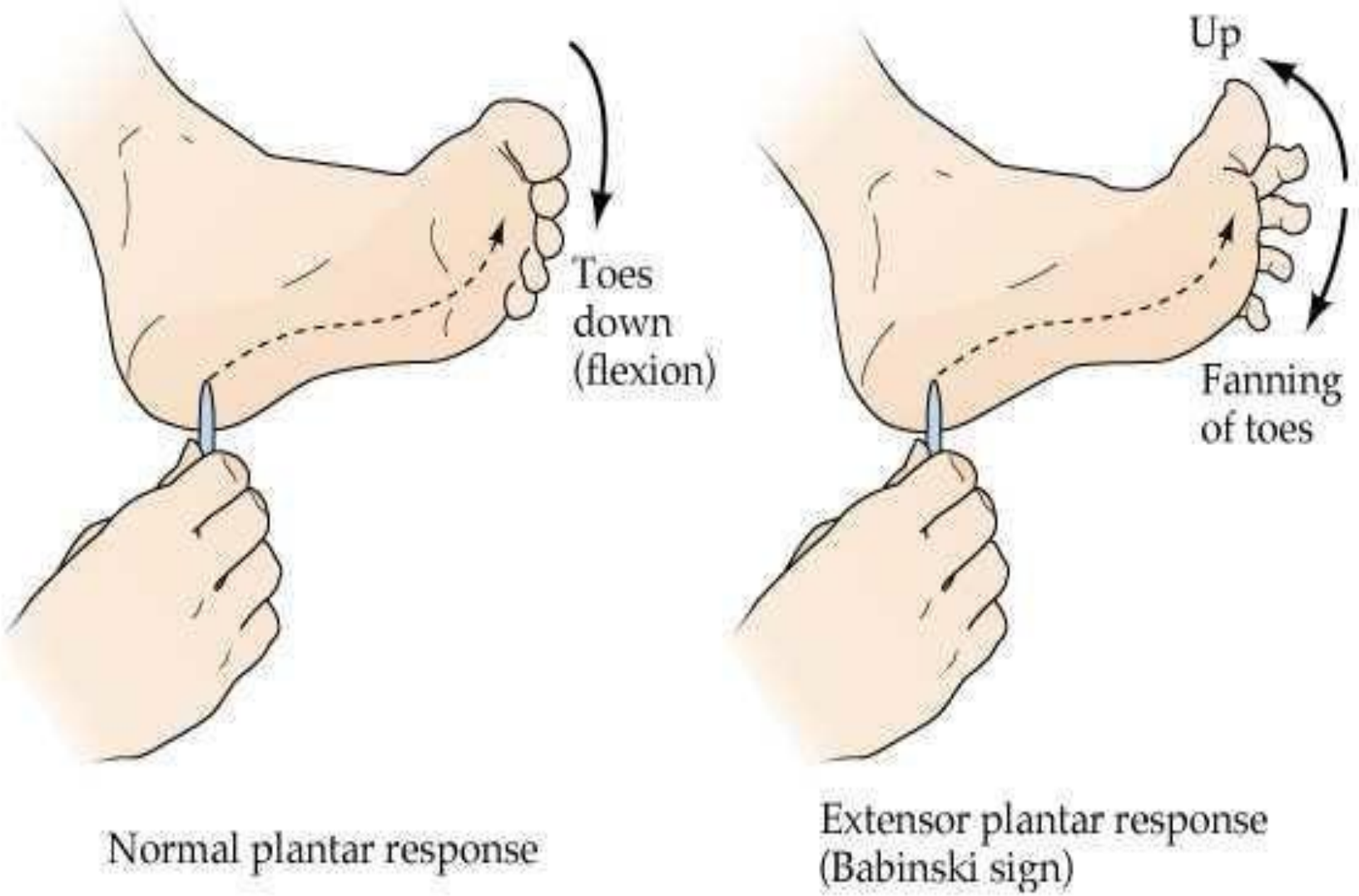
Can result from

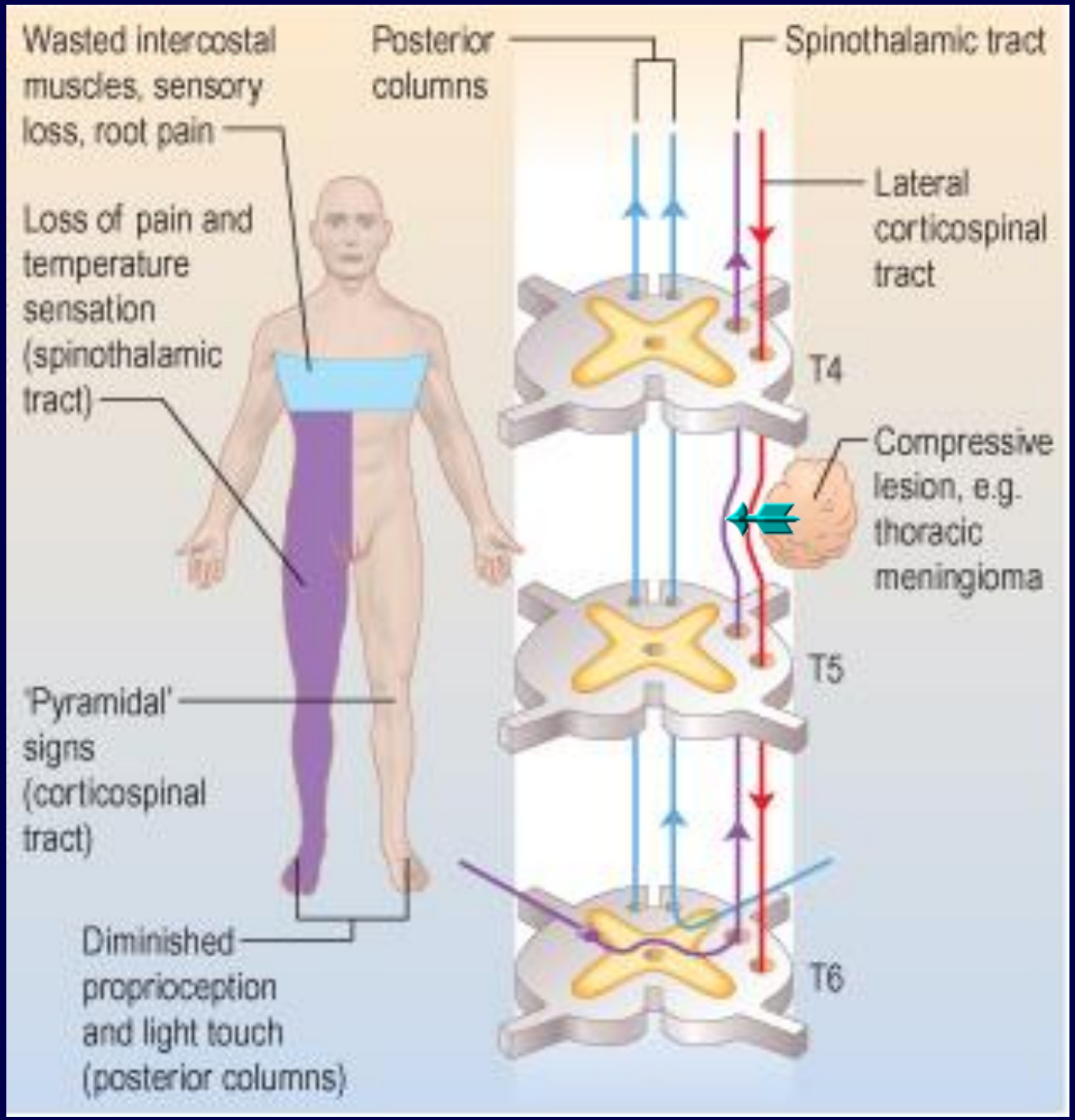
- Anterior horn cell lesions (e.g. poliomyelitis, motor neuron disease
- Spinal root lesions or peripheral nerve lesion (e.g. nerve injury by trauma or compressive lesion)

COMAPRISON UPPER & LOWER MOTOR NEURON LESIONS

	UMN LESION	LMN LESION
Pattern	Paralysis affect movements	Individual muscle or group of muscles are affected.
Wasting	not pronounced	Pronounced
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)	Absent	Present
Fasciculations	Absent	Present
Trophic changes	Less	Pronounced
clonus	Present	Absent
Babinski's sign	Extensor plantar response (Positive)	flexor or absent plantar responses

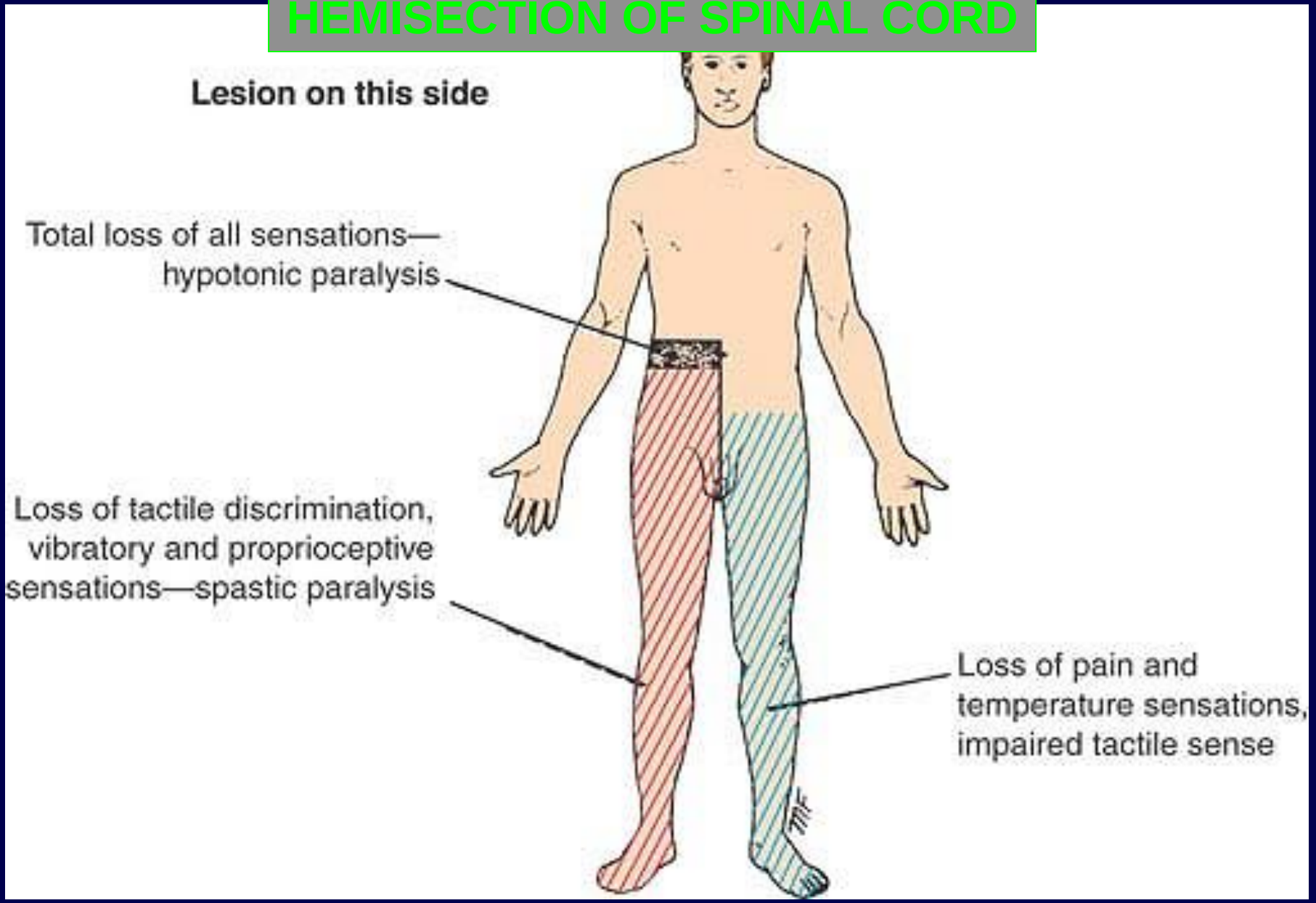
fasciculation
a brief spontaneous contraction affecting a small number of muscle fibres, often causing a flicker of movement under the skin.
fibrillation
a small, local, involuntary, muscular contraction, due to spontaneous activation of single muscle cells or muscle fibers


Toes
down
(flexion)
Normal plantar response
Up
Fanning
of toes
Extensor plantar response
(Babinski sign)


Wasted intercostal muscles, sensory loss, root pain
Posterior columns
Spinothalamic tract
Lateral corticospinal tract
Loss of pain and temperature sensation (spinothalamic tract)
T4
Compressive lesion, e.g. thoracic meningioma
T5
'Pyramidal' signs (corticospinal tract)
Diminished proprioception and light touch (posterior columns)
T6

Brown Sequard syndrome

HEMISECTION OF SPINAL CORD



Brown Sequard syndrome

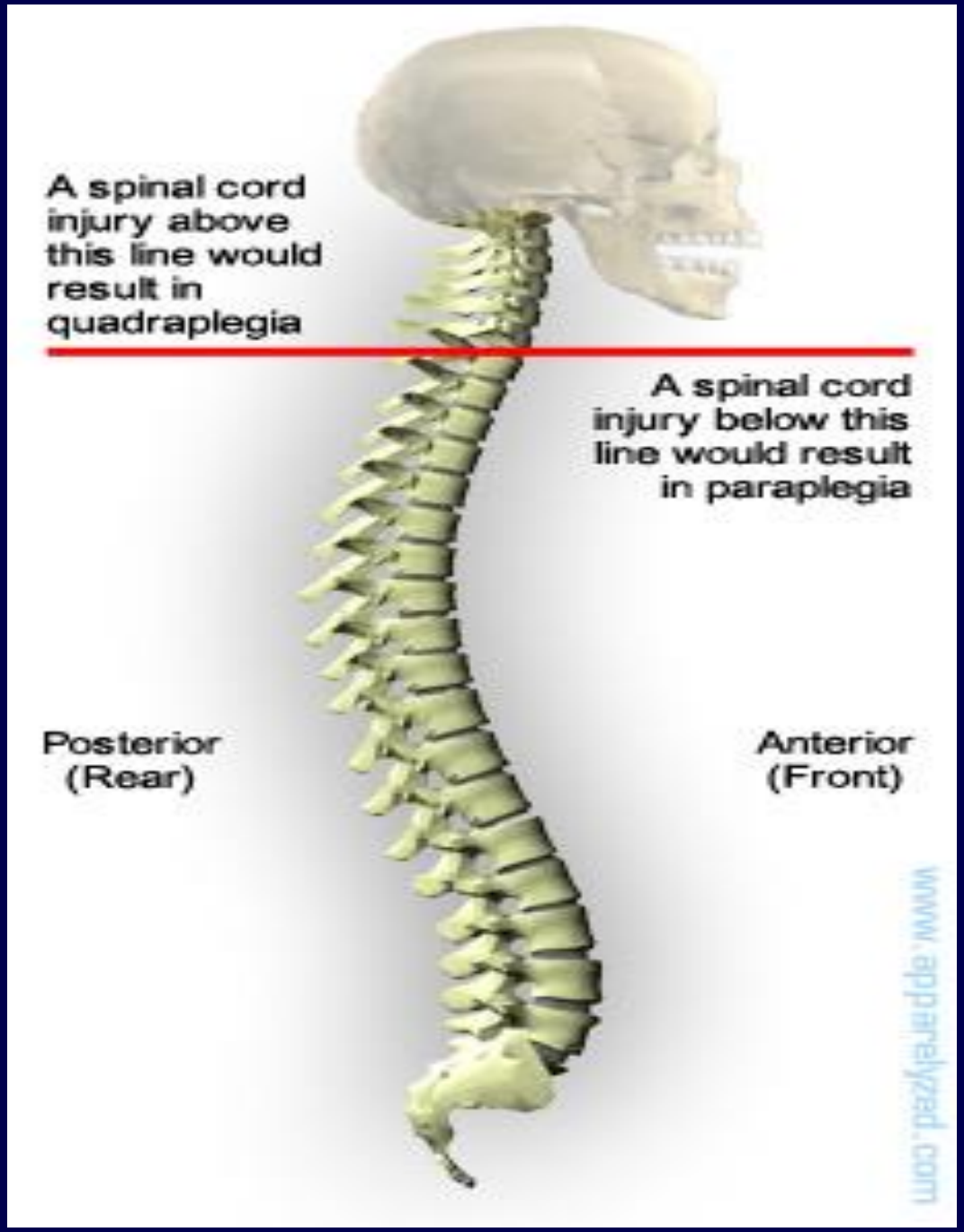
HEMISECTION OF SPINAL CORD

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)



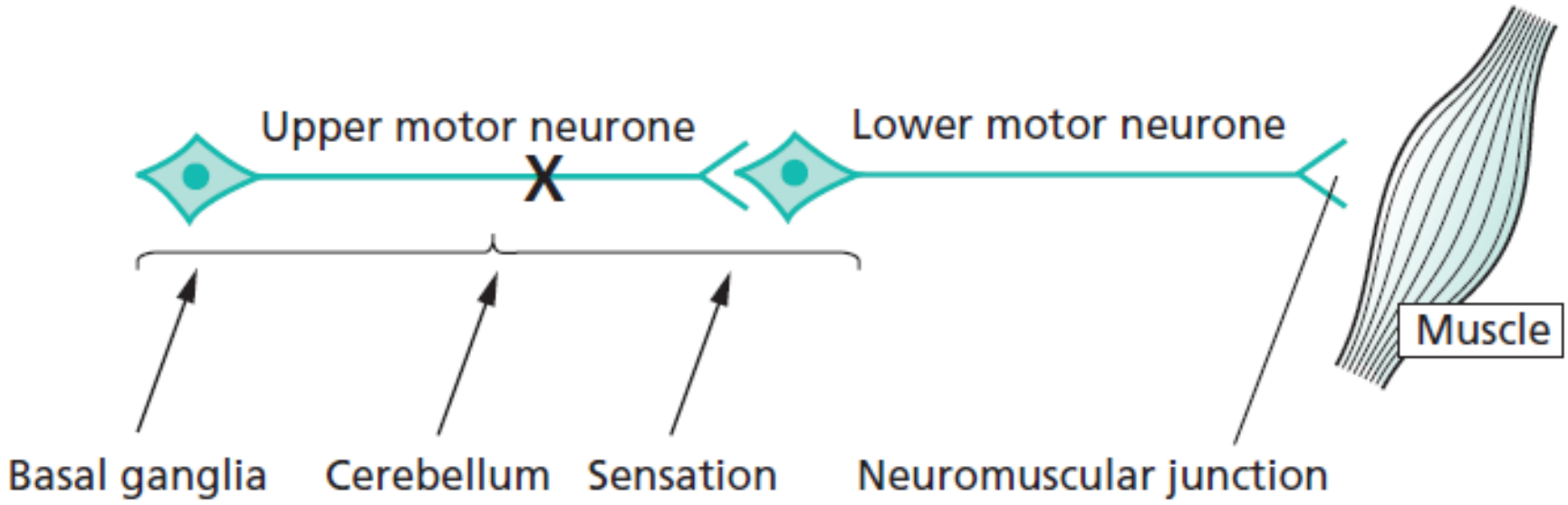
Upper cervical cord lesions produce quadriplegia and weakness of the diaphragm

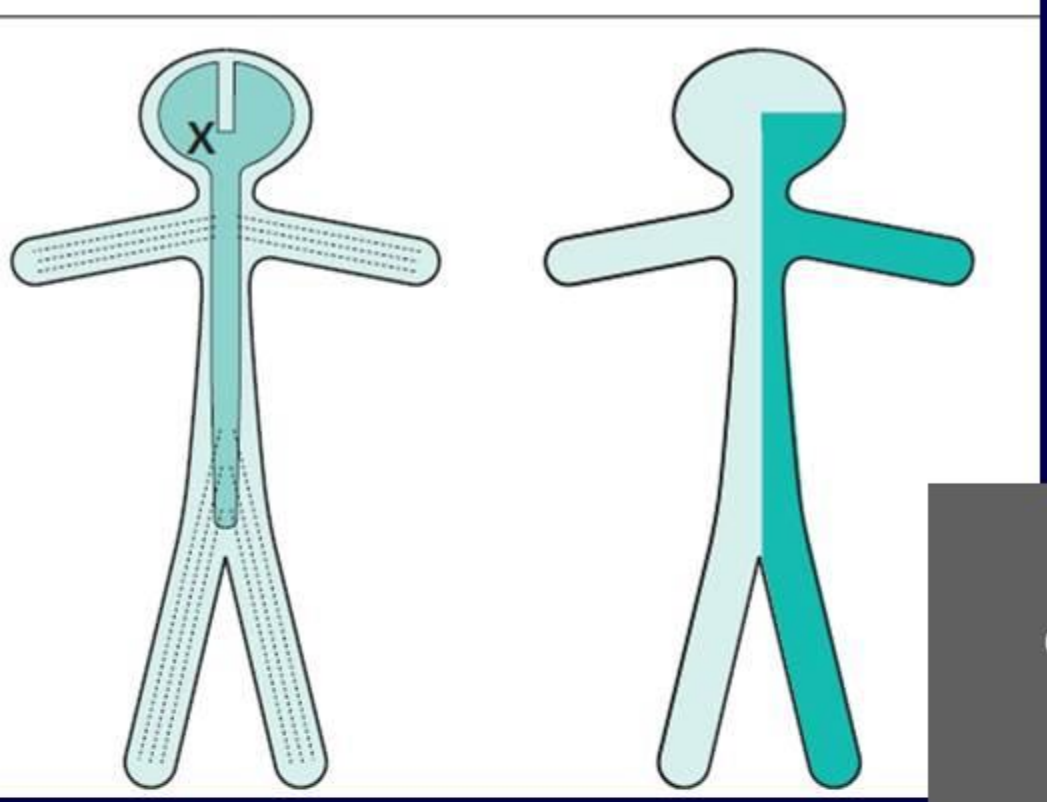
Lesions at C4-C5 produce quadriplegia

Hemiparesis means weakness

Hemiplegia means total paralysis

Upper motor neurone





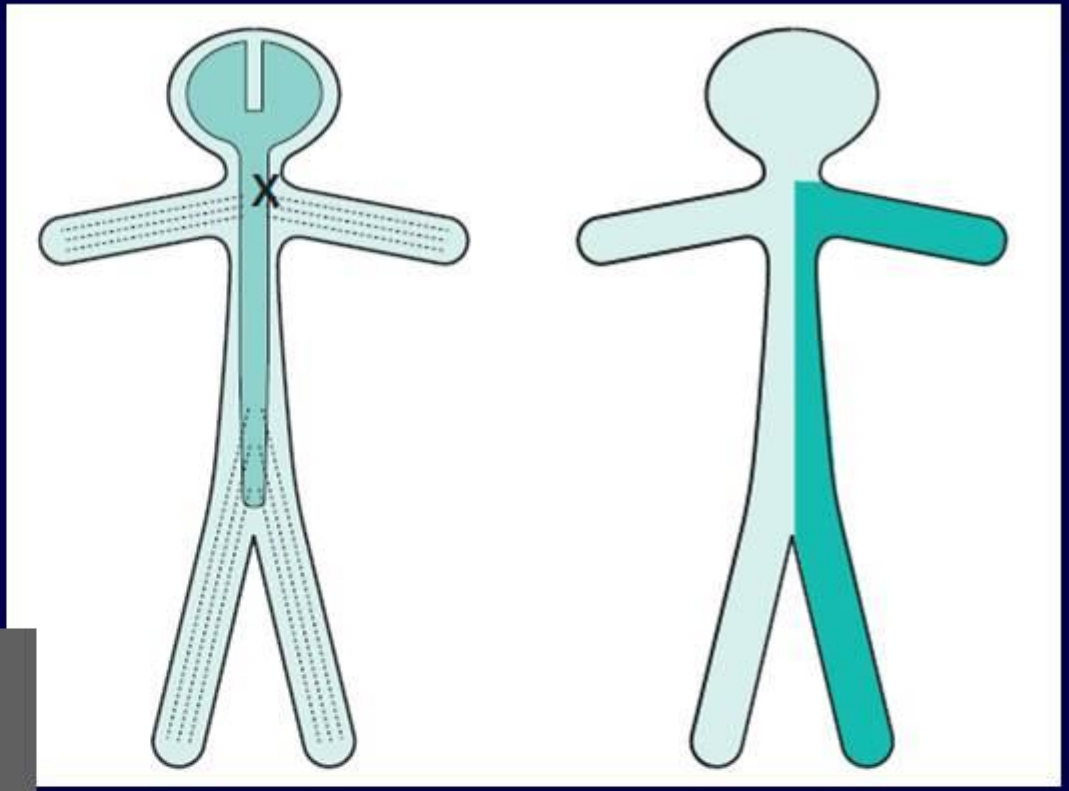
Contralateral Hemiparesis

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

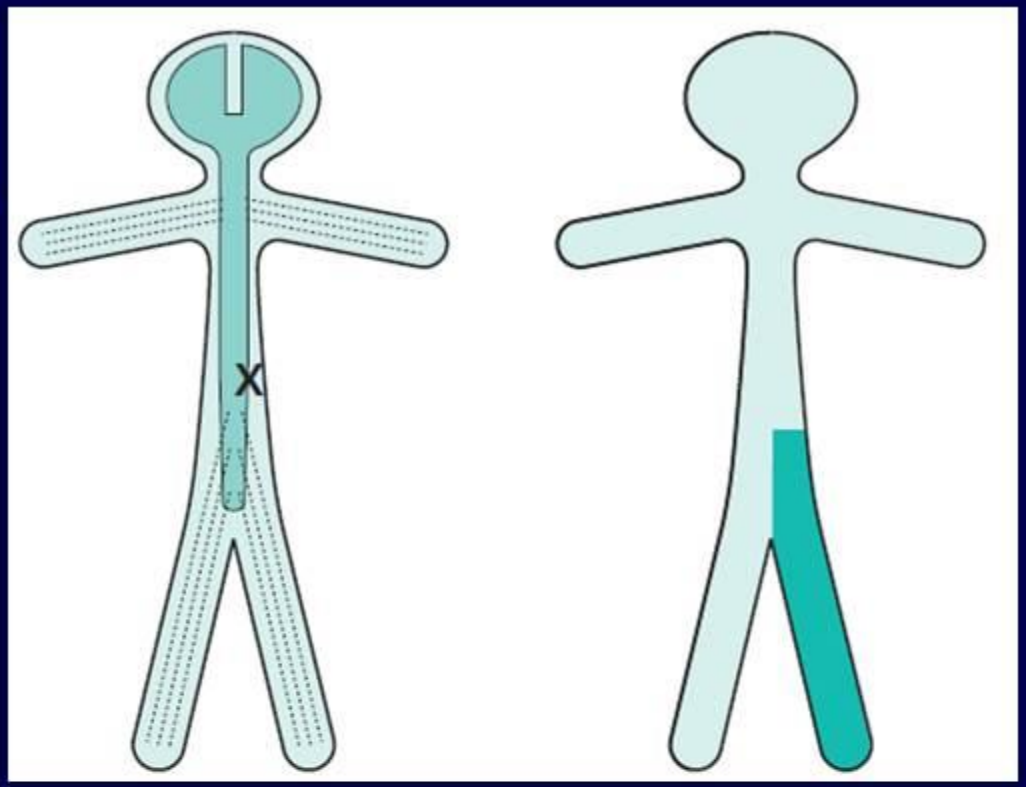
A unilateral high cervical cord lesion will produce a hemiparesis similar to that which is caused by a contralateral cerebral hemisphere lesion, except that the face cannot be involved in the hemiparesis, vision will be normal, and the same dissociation of sensory loss (referred to above) may be found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



Ipsilateral Hemiparesis

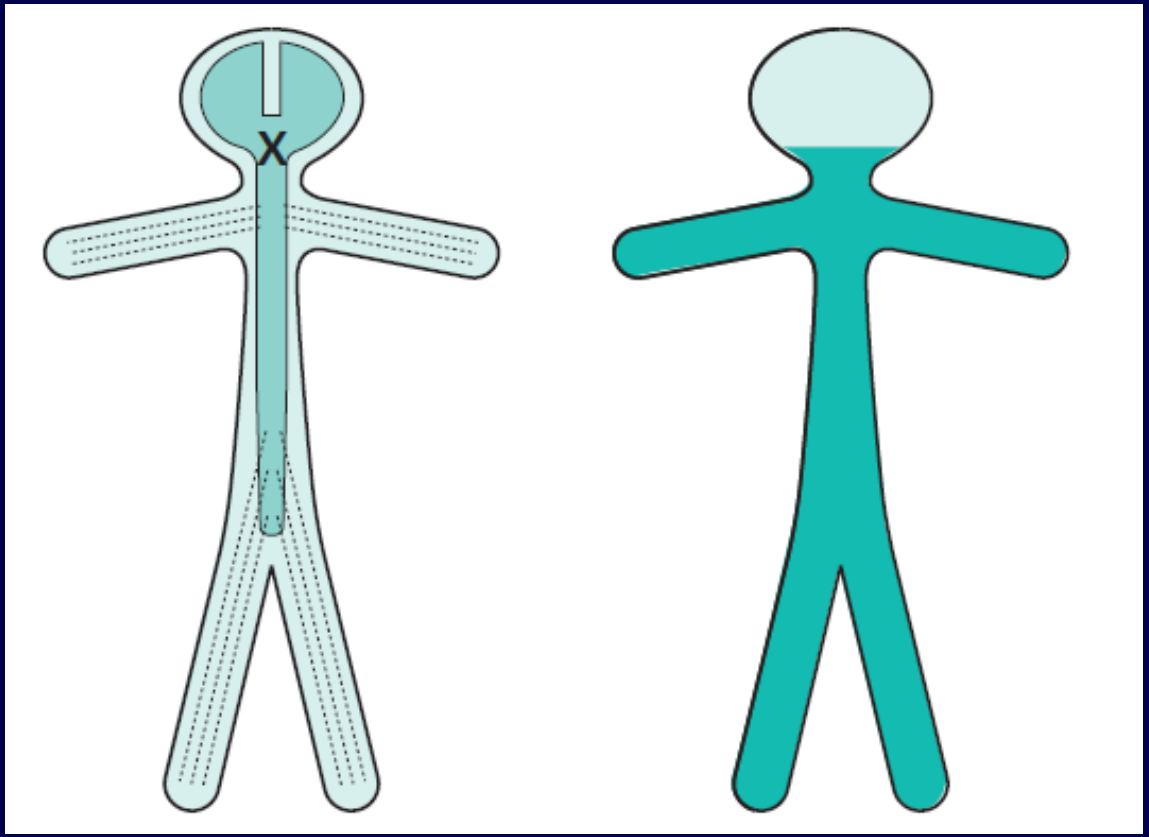
Ipsilateral Monoparesis

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.

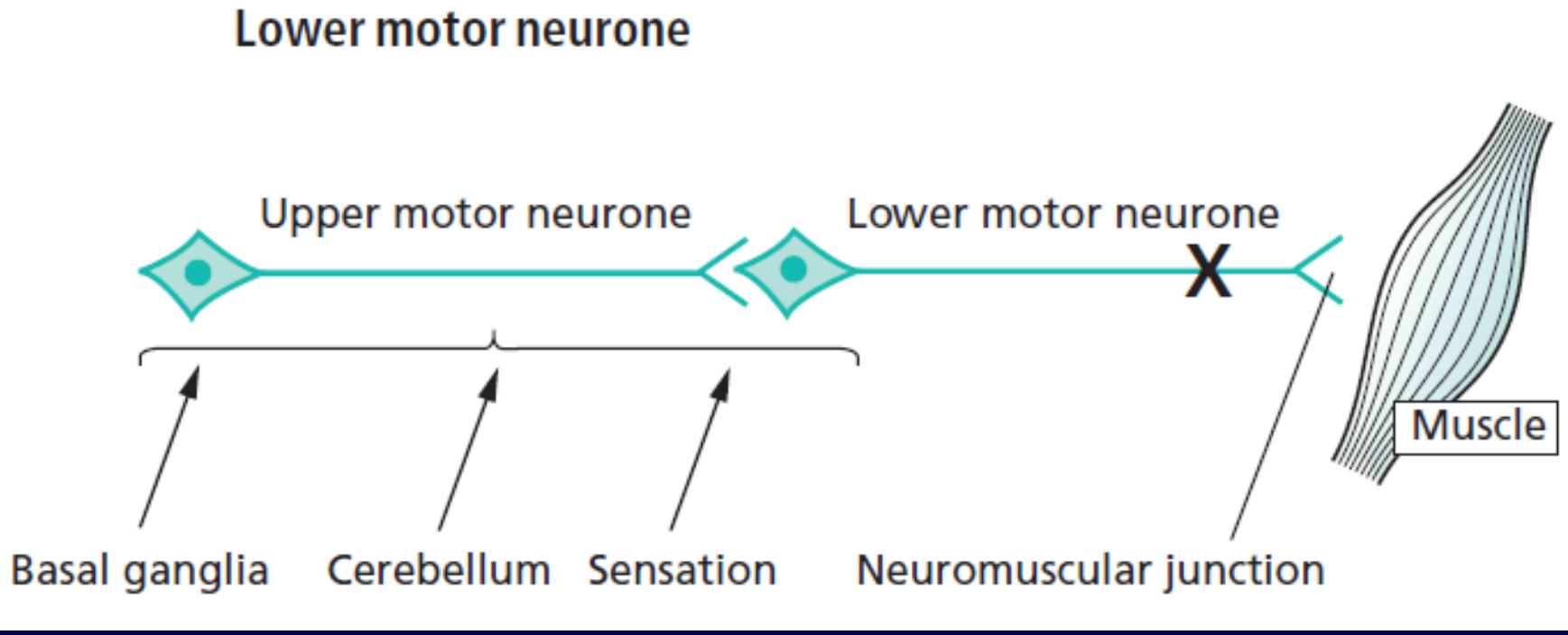


Ipsilateral Monoparesis

Tetraparesis or Quadriparesis



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


Lower motor neurone
Upper motor neurone
Lower motor neurone
X
Muscle
Basal ganglia
Cerebellum
Sensation
Neuromuscular junction

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

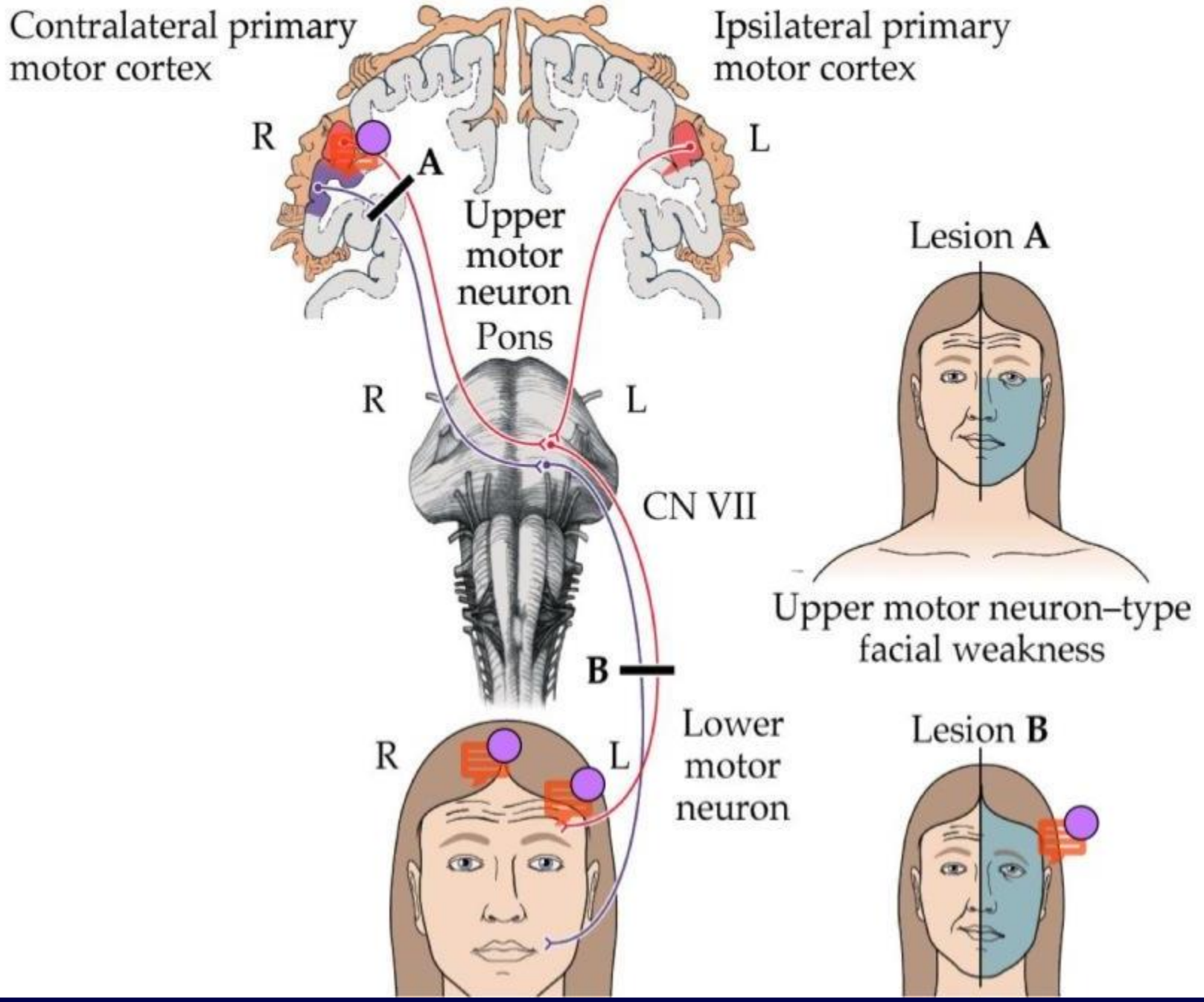
Pseudobulbar palsy-

- **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
L
A
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.**

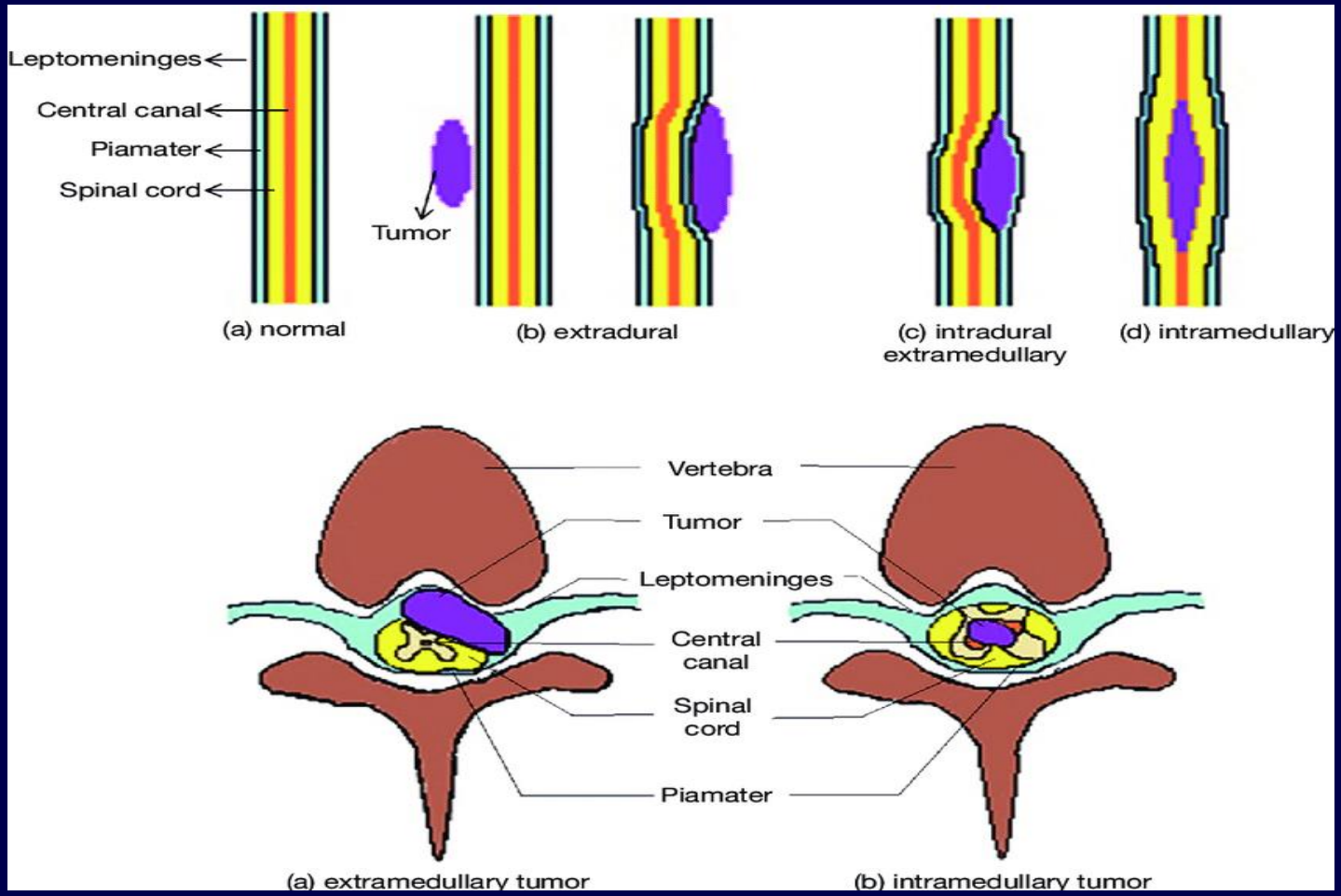
Intramedullary and Extramedullary Syndromes

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


Leptomeninges
Central canal
Piamater
Spinal cord
Tumor
(a) normal
(b) extradural
(c) intradural extramedullary
(d) intramedullary
Vertebra
Tumor
Leptomeninges
Central canal
Spinal cord
Piamater
(a) extramedullary tumor
(b) intramedullary tumor

SYSTEM	FEATURES	EXTRAMEDULLARY	INTRAMEDULLARY
HISTORY	Onset	Asymmetrical	Symmetrical
	Pain	Local or vertebral (extradural) Radicular (intradural)	Funicular or tract pain
MOTOR	UMN signs	early	late
	LMN signs	segmental	diffuse
SENSORY	Sensory involvement	Ascending (sacral involvement)	Descending (sacral sparing)
	Dissociated sensory loss	absent	present
AUTONOMIC	Sphincter involvement	late	early