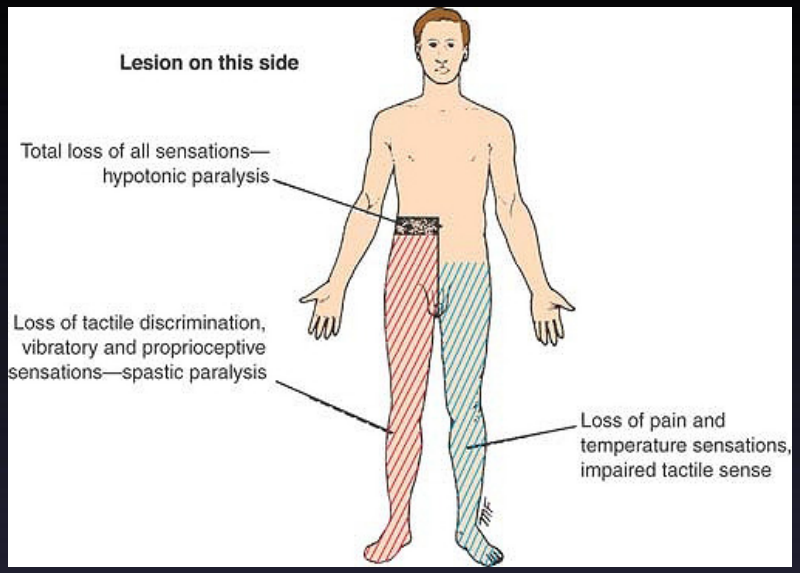
UPPER AND LOWER MOTOR NEURON LESIONS



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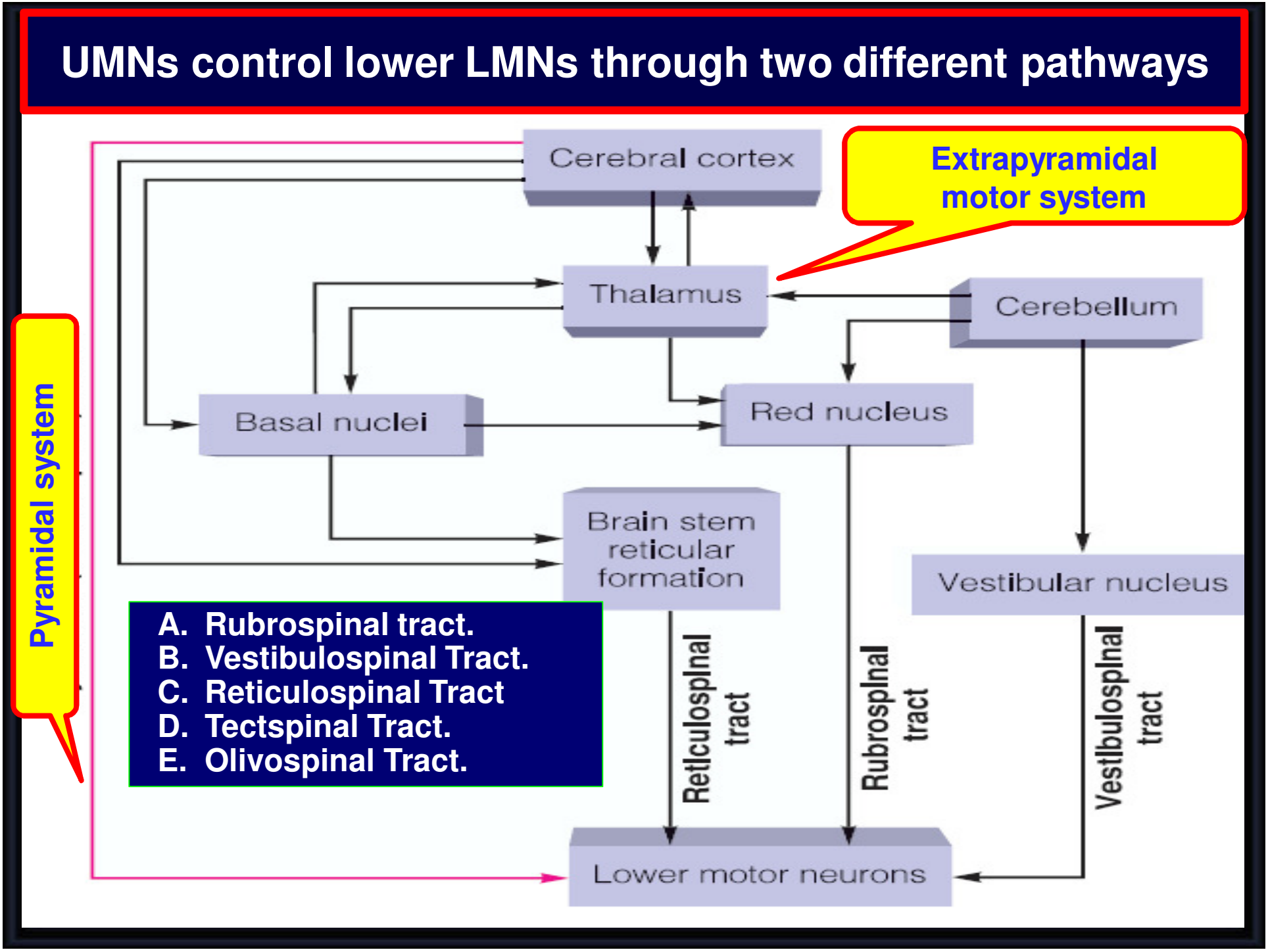
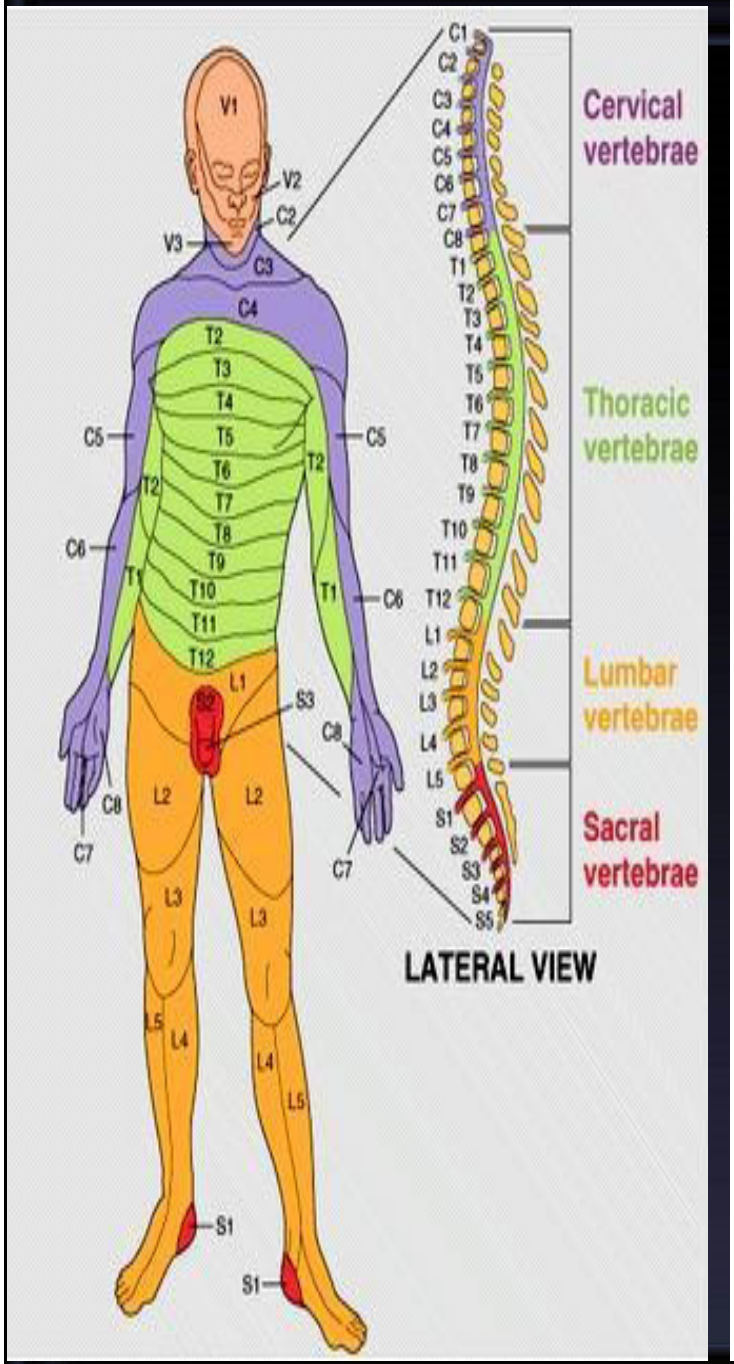
OBJECTIVES

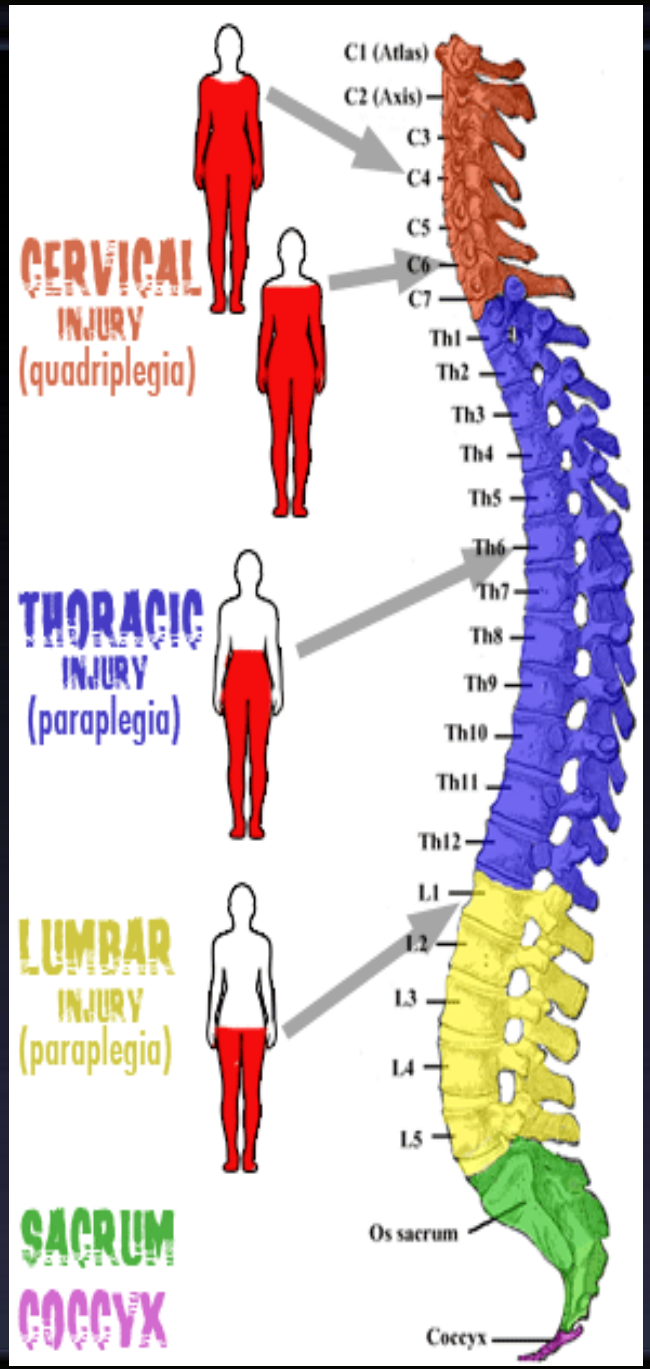
At the end of this lecture you should be able to

- 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

UMNs control lower LMNs through two different pathways

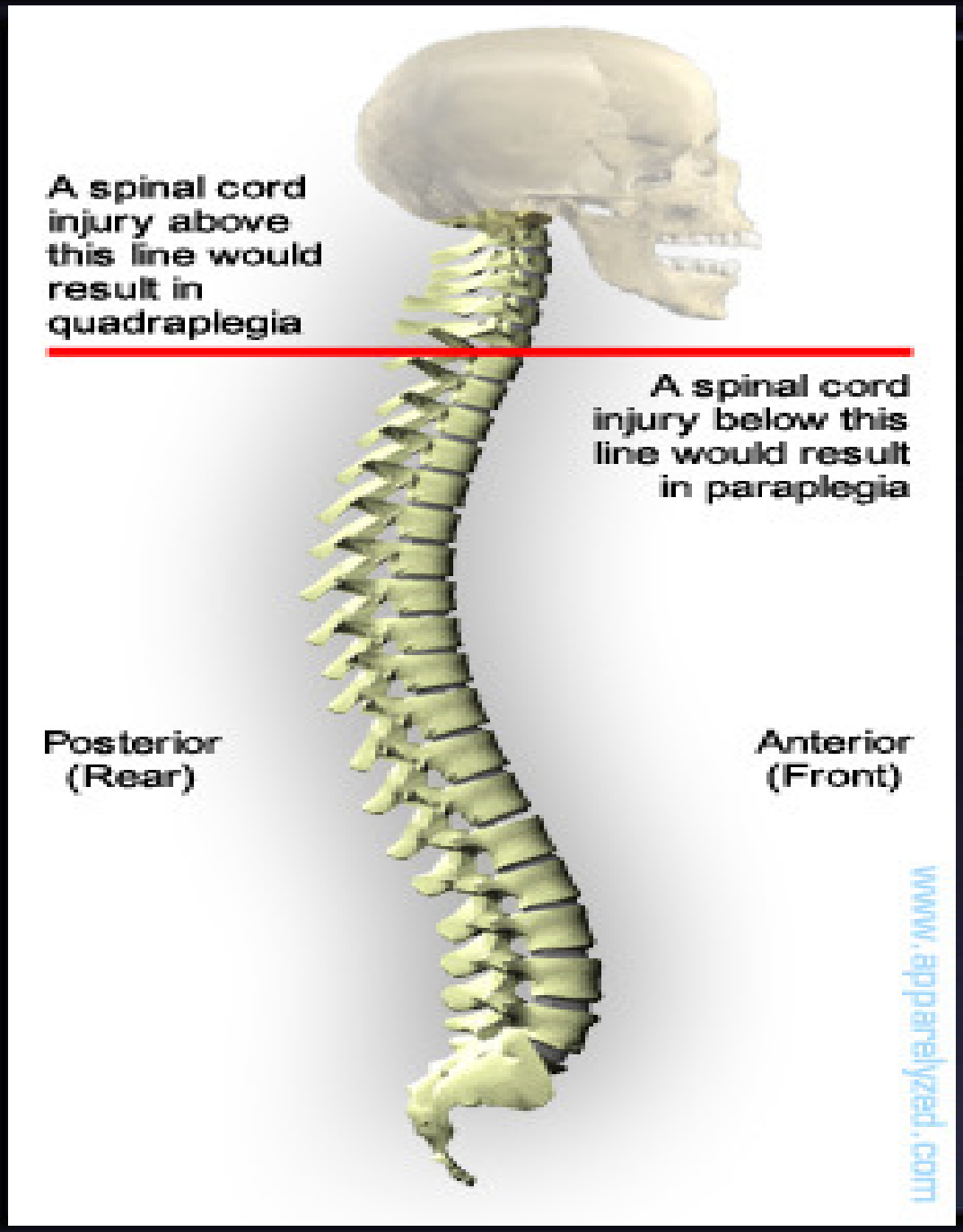






31 segments

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



Upper cervical cord lesions produce quadriplegia and weakness of the Diaphragm
Lower lesions spare diaphragm with good prognosis

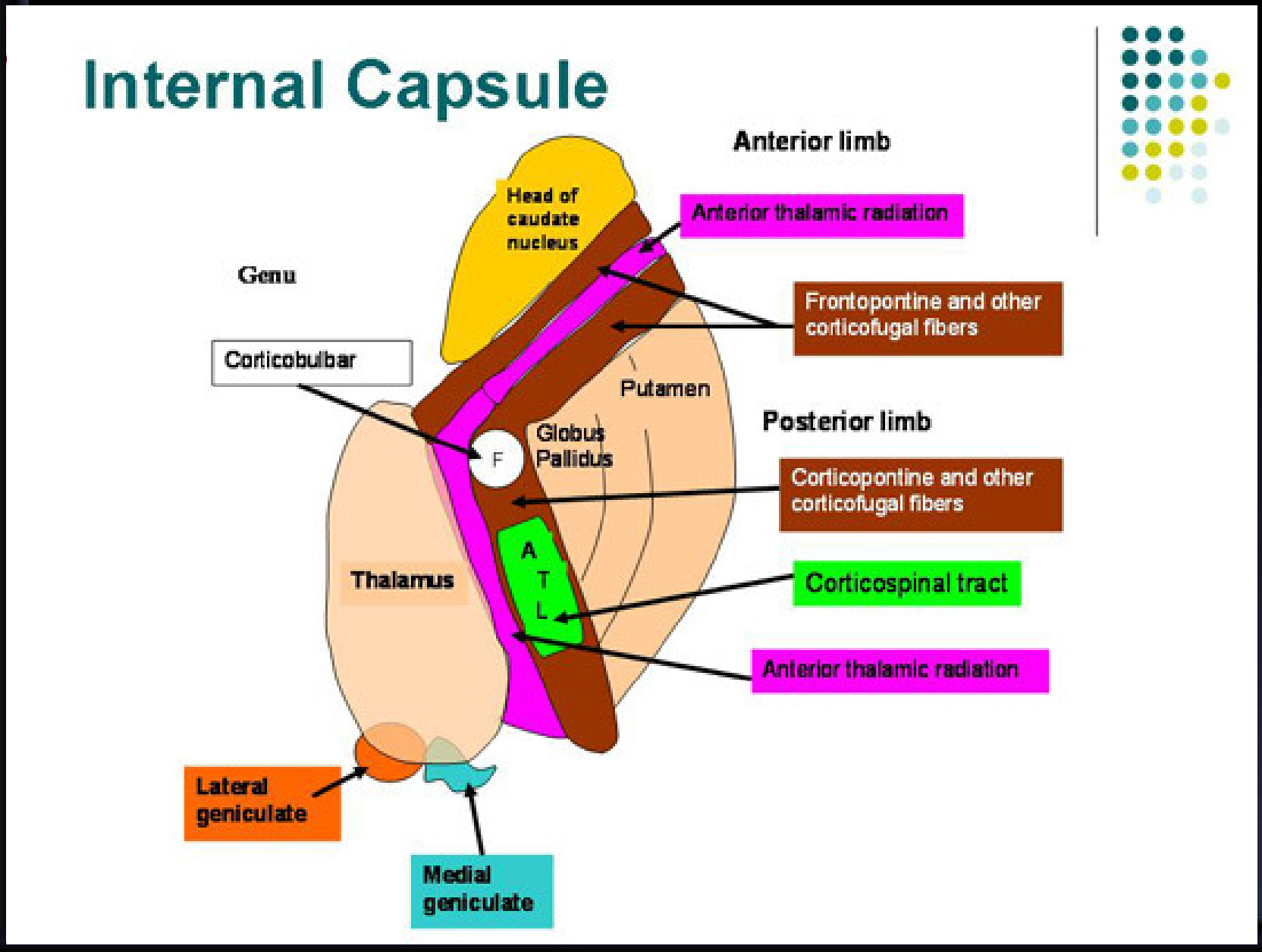
Lesions at C4-C5 produce quadriplegia

Hemiparesis means weakness

Hemiplegia means total paralysis

limerick “C3, 4 and 5 keep the **diaphragm** alive”.

INTERNAL CAPSULE



Causes of UMNL & LMNL

UMN Lesion

Can result from

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

LMN Lesion

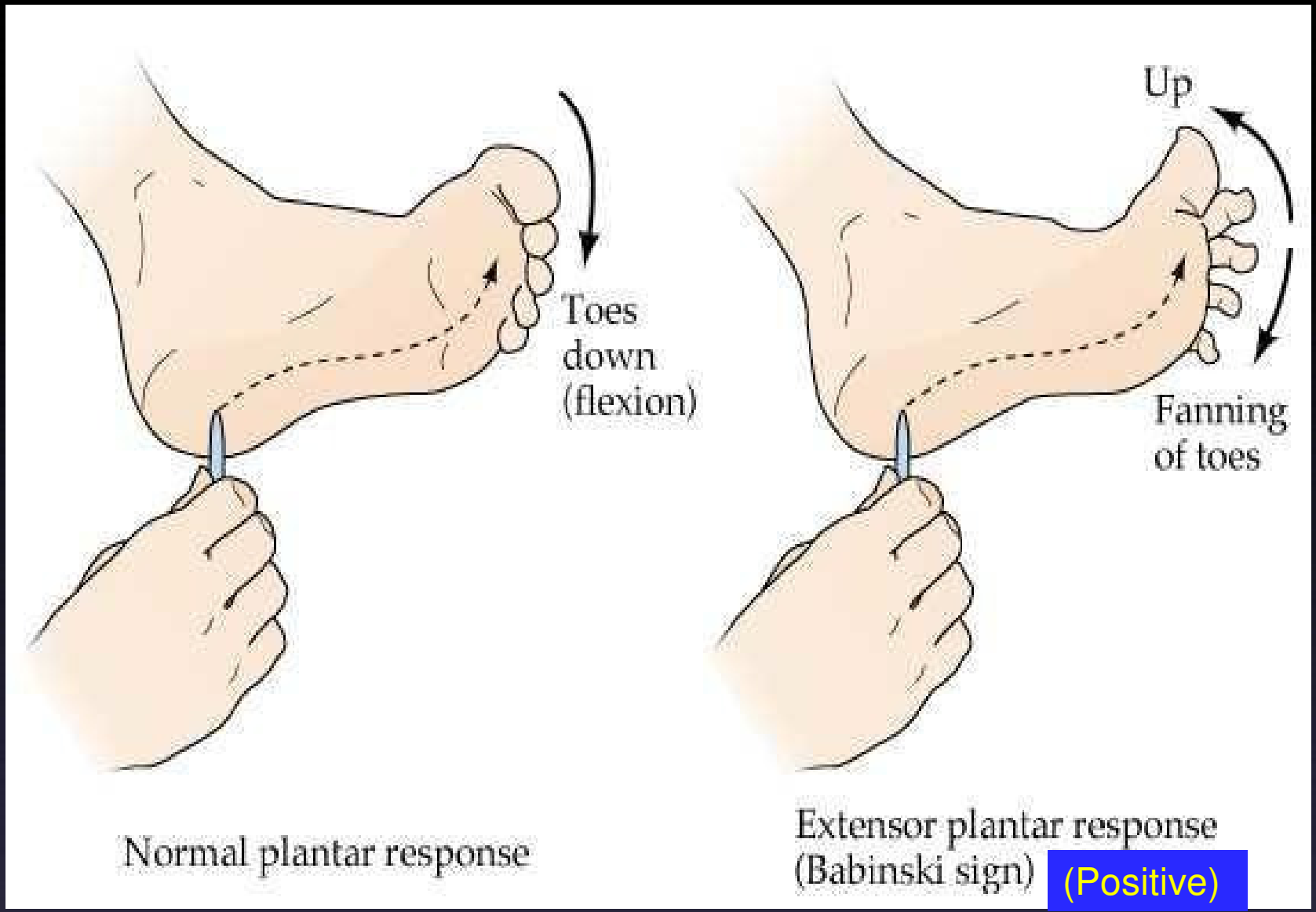
Can result from

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

Note: Pure corticospinal tract lesion cause hypotonia instead of spasticity The reason is that pure pyramidal tract lesion is very very rare, and spasticity is due to loss of inhibitory control of extrapyramidal tracts.

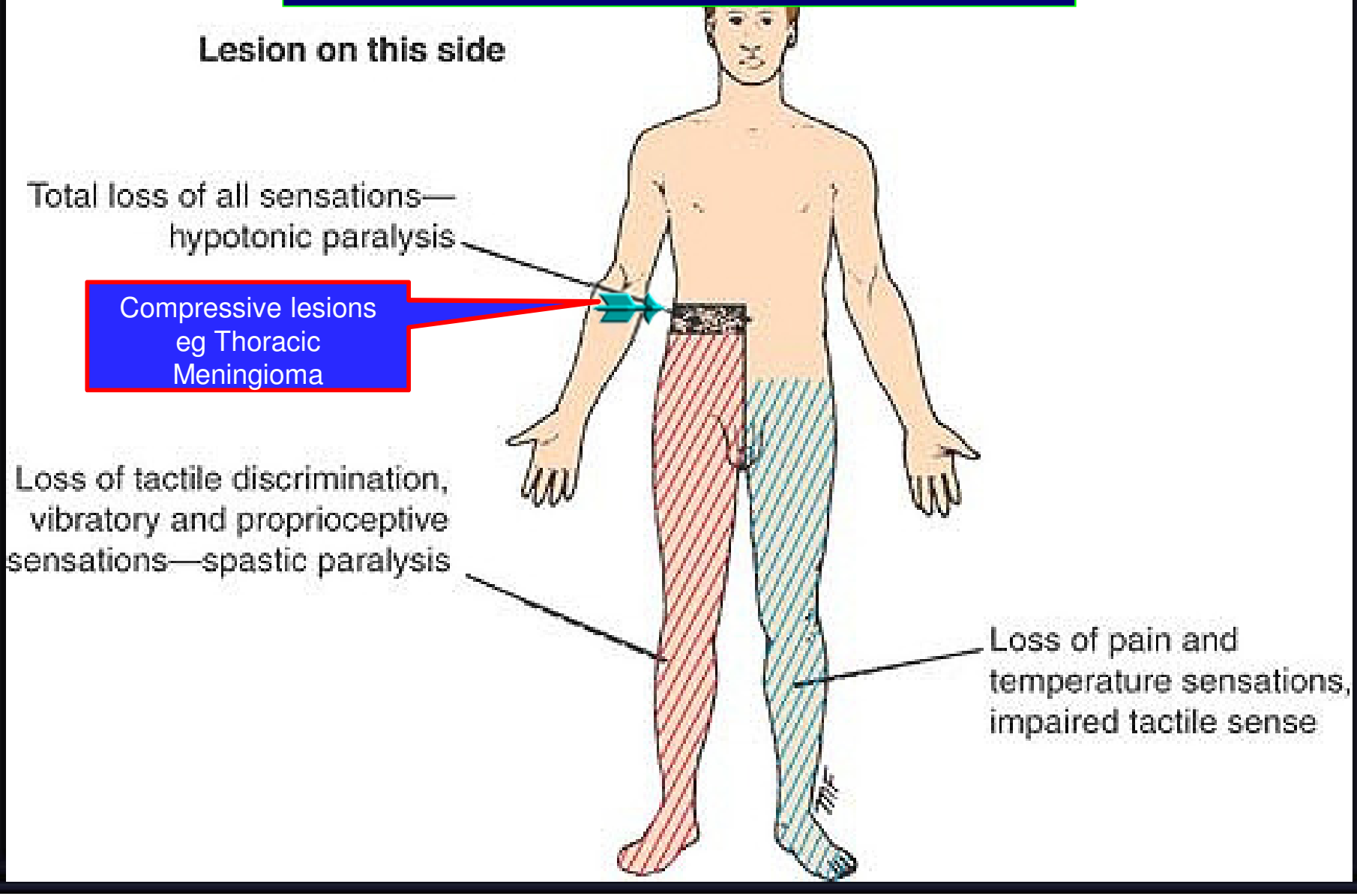
COMAPRISON UPPER & LOWER MOTOR NEURON LESIONS

	UMN LESION	LMN LESION
Pattern	Paralysis affect movements	Individual muscle or group of muscles are affected.
Wasting	Not pronounced (About 20-30% wasting)	Pronounced (About 70-80% wasting)
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


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

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)

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.

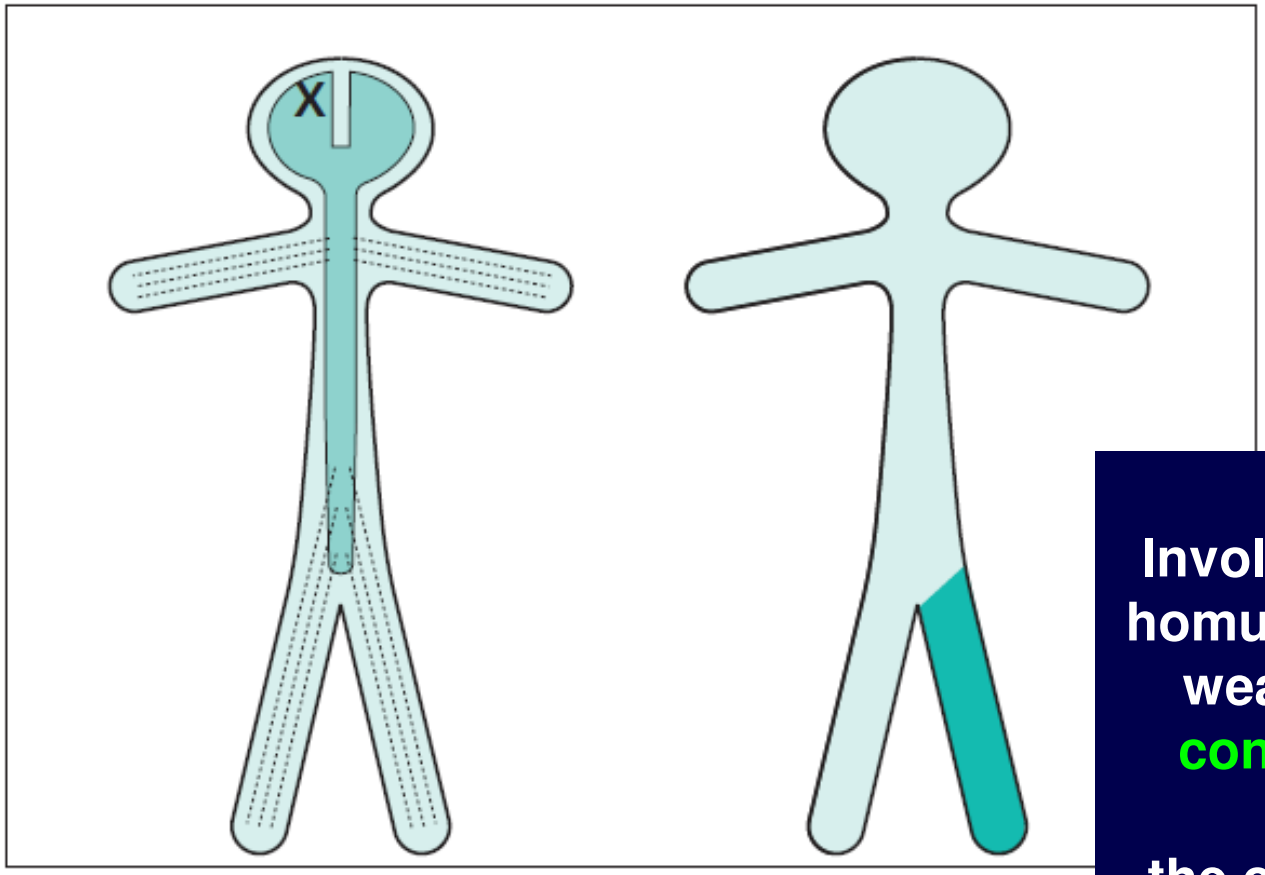
2. Ipsilateral spastic paralysis below the level of the lesion. An ipsilateral Babinski sign is present, and depending on the segment of the cord damaged, an ipsilateral loss of the superficial abdominal reflexes and cremasteric reflex occurs. All these signs are due to loss of the corticospinal tracts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

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.

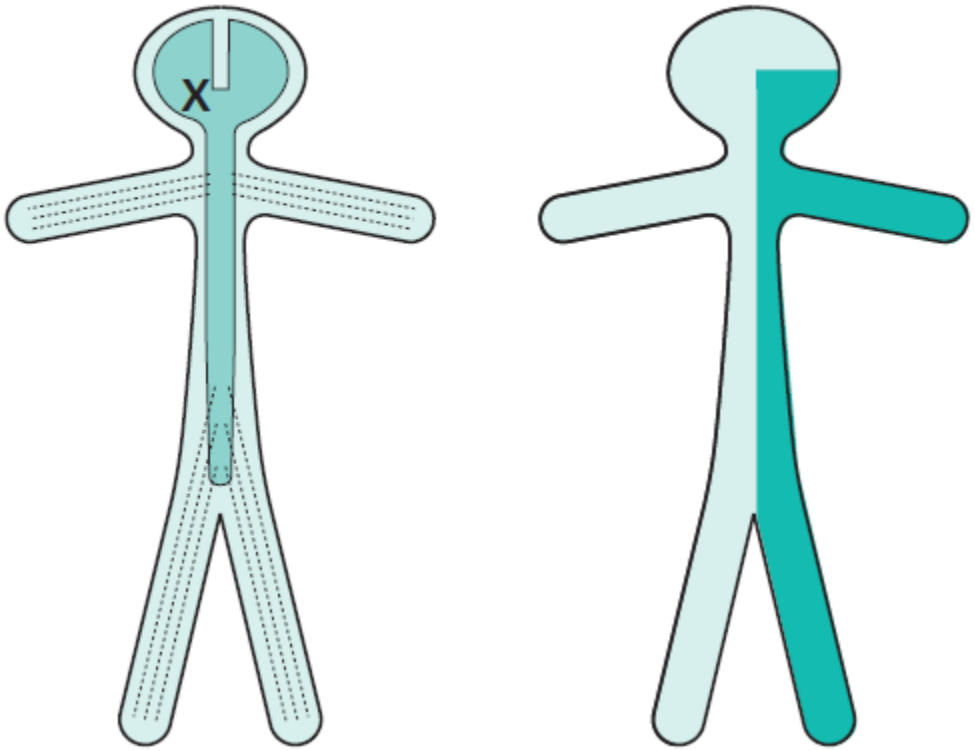
6. Contralateral but not complete loss of tactile sensation below the level of the lesion. This condition is brought about by destruction of the crossed anterior spinothalamic tracts on the side of the lesion. Sensory impairment occurs two or three segments below the level of the lesion distally.



Lesion situated peripherally in the cerebral hemisphere

CONTRALATERAL MONOPARESIS

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.



Internal Capsule

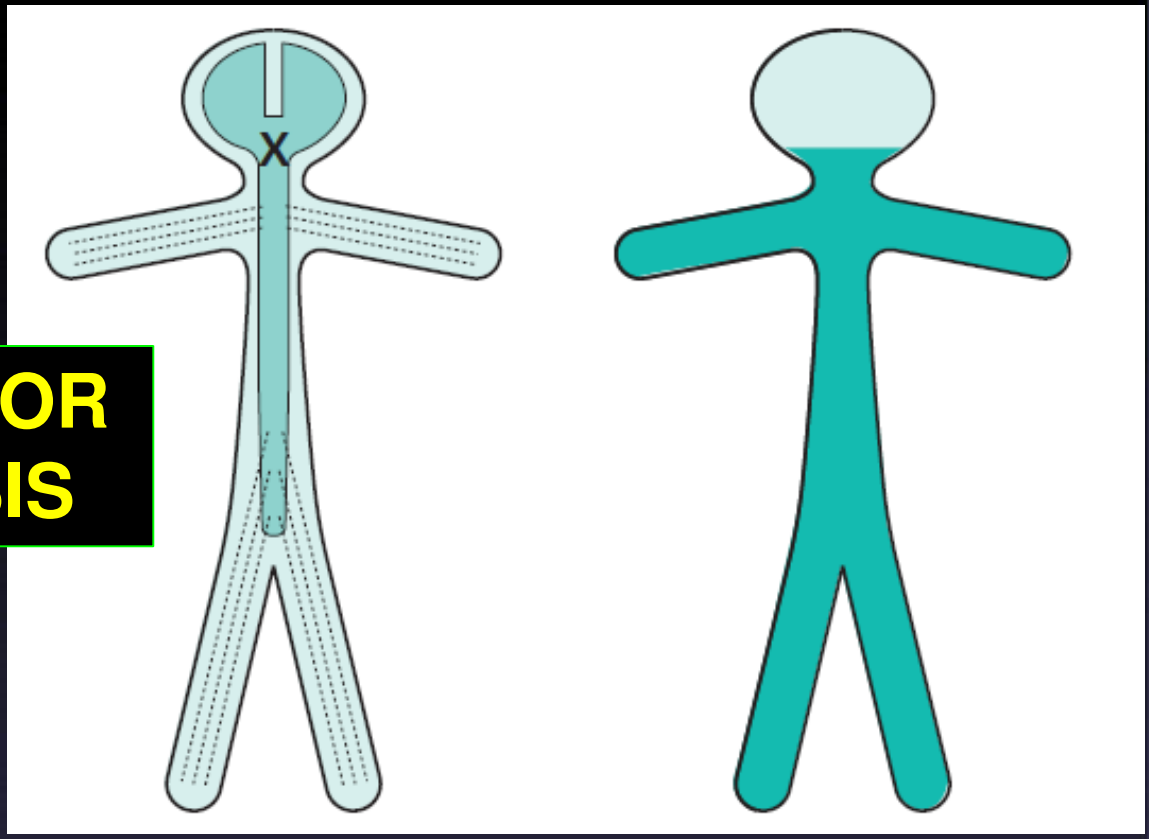
CONTRALATERAL HEMIPARESIS

Lesions situated deep are much more likely to produce weakness of the whole of the contralateral side of the body, face, arm and leg.

Such lesions commonly produce significant contralateral sensory loss (hemianaesthesia) and visual loss (homonymous hemianopia), in addition to the hemiparesis.

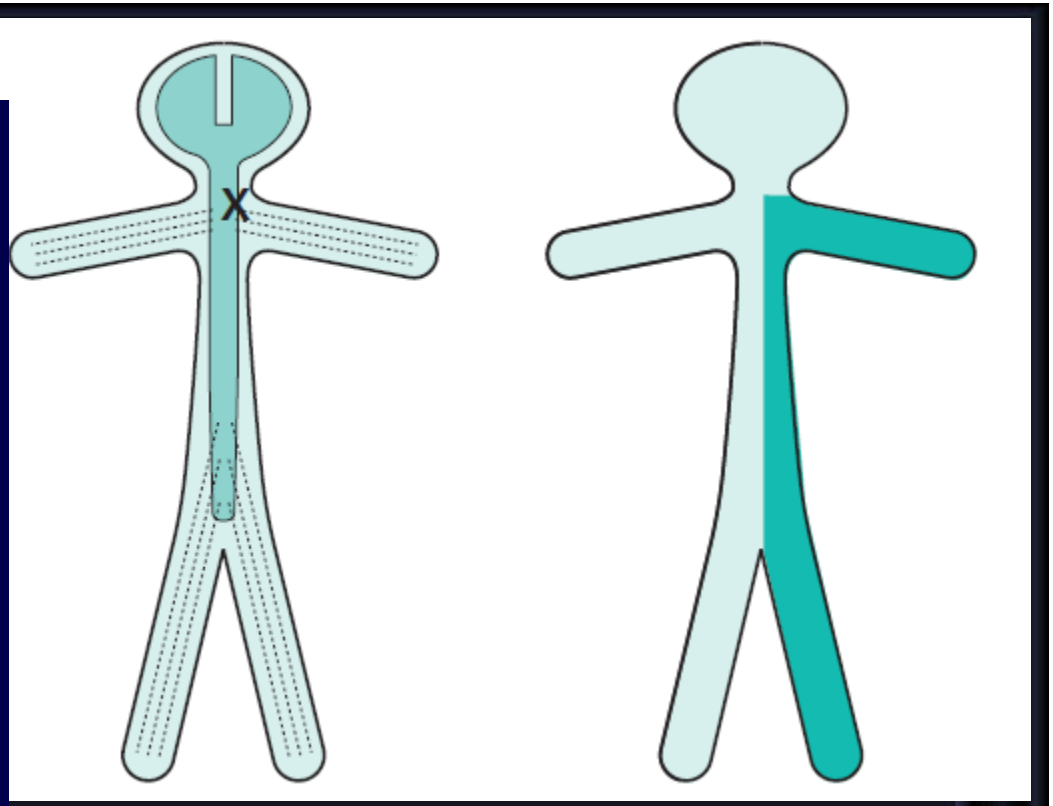
A spinal cord lesion more usually causes upper motor neuron signs in both legs, often asymmetrically since the pathology rarely affects both sides of the spinal cord equally.

TETRAPARESIS OR QUADRIPARESIS



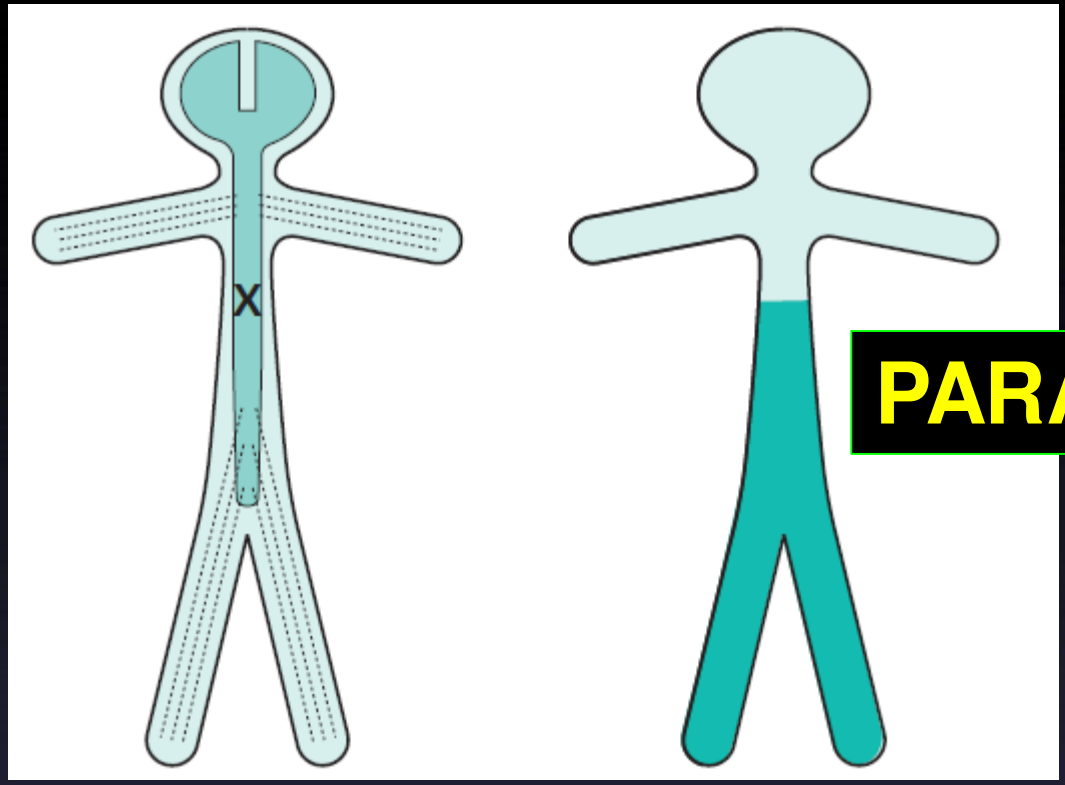
Tetraparesis or quadriparesis, if the lesion is in the upper cervical cord or brainstem.

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 may be found below the level of the lesion.



IPSILATERAL HEMIPARESIS

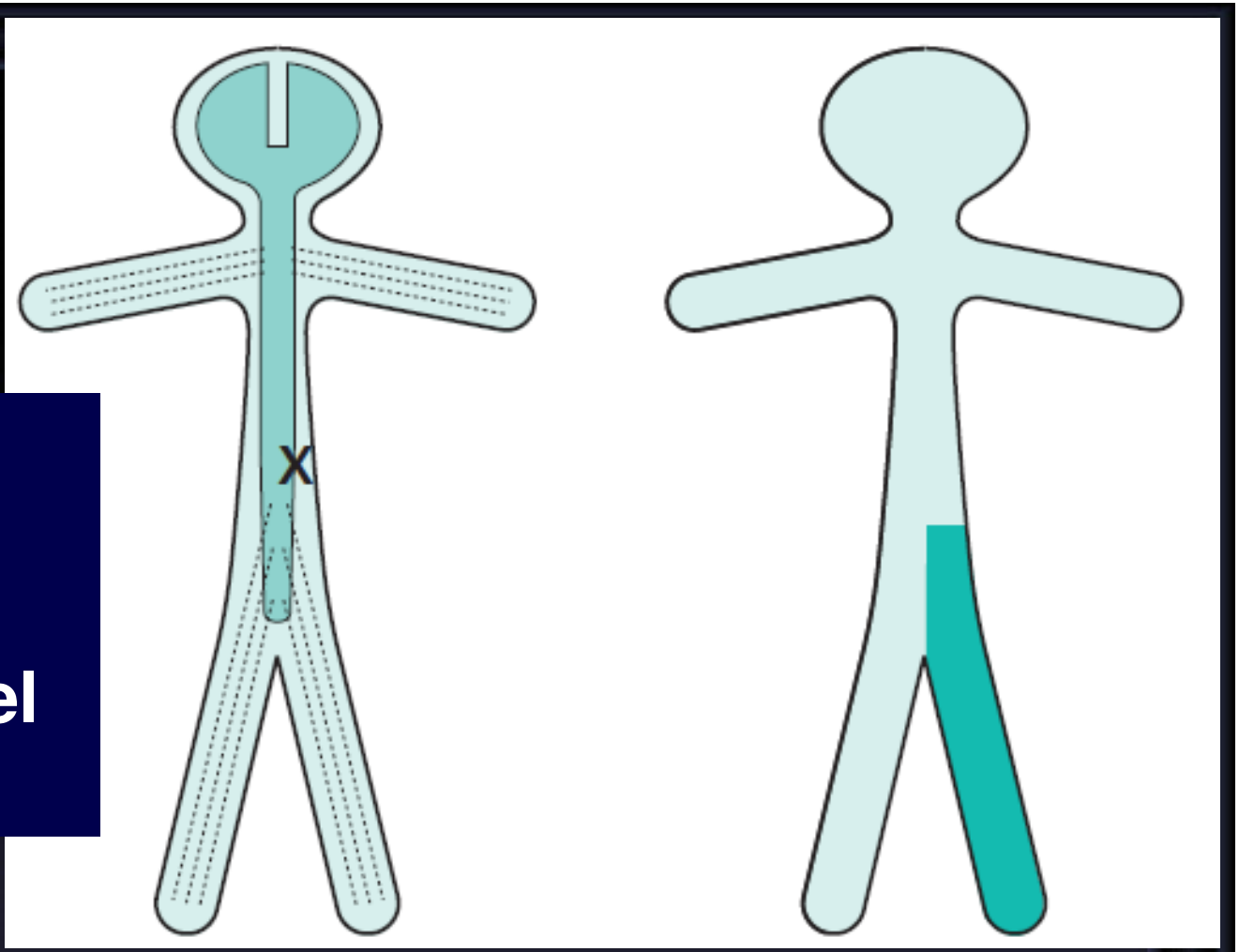
A spinal cord lesion more usually causes upper motor neuron signs in both legs, often asymmetrically since the pathology rarely affects both sides of the spinal cord equally.



PARAPARESIS

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

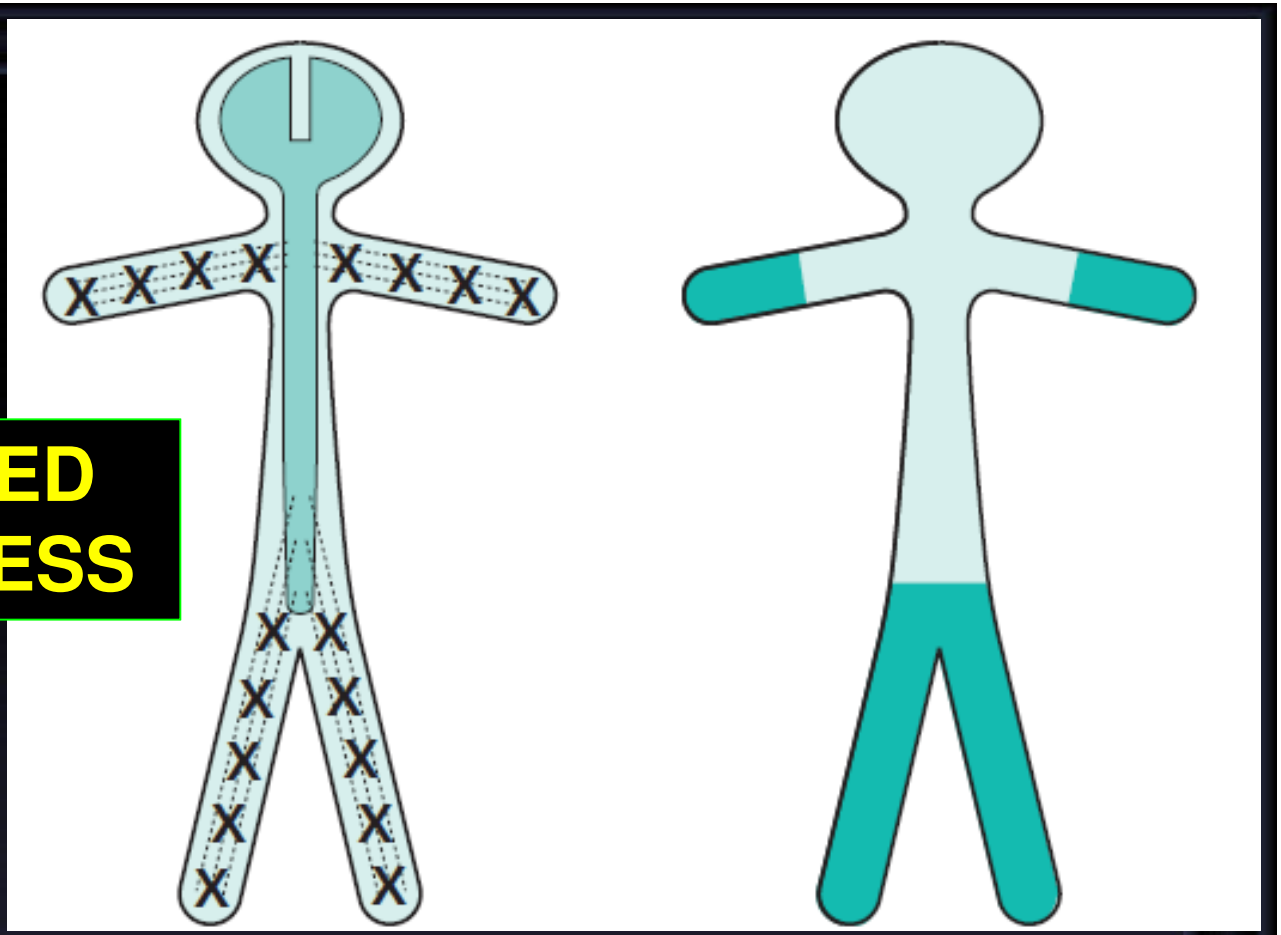
A unilateral lesion in the spinal cord below the level of the neck



Brown-Séquard syndrome

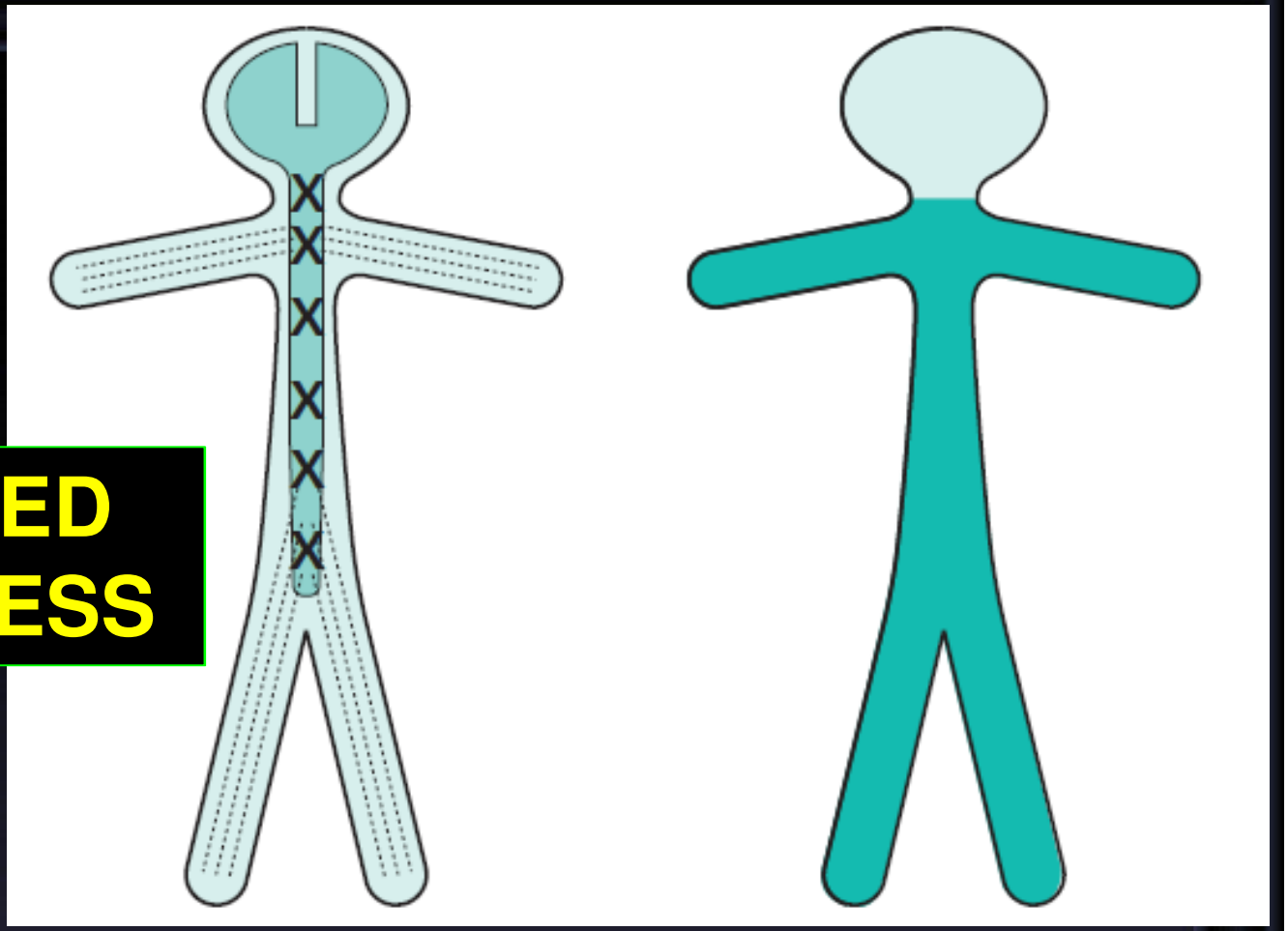
IPSILATERAL MONOPARESIS

GENERALIZED LMN WEAKNESS



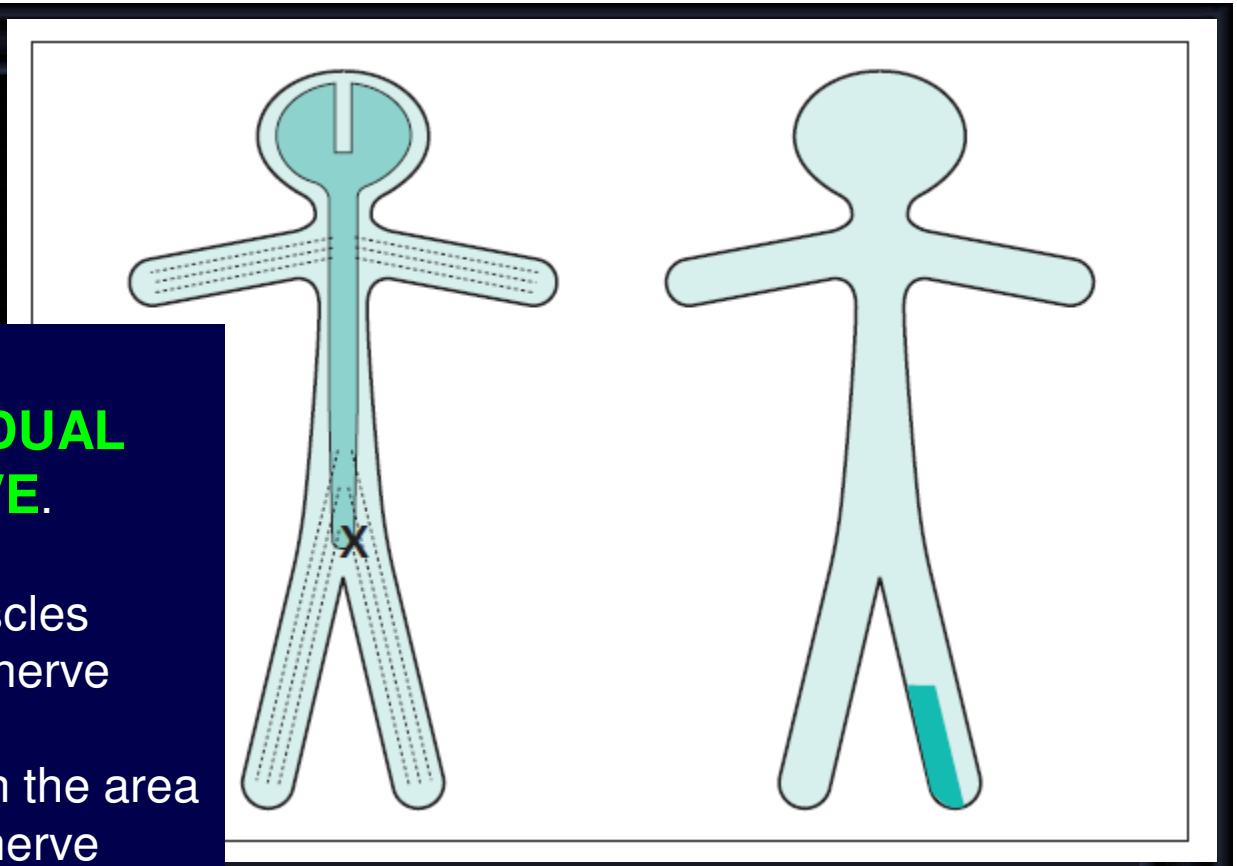
May result from widespread damage to the axons of LMNs.
This is the nature of peripheral neuropathy (polyneuropathy).
The axons of the dorsal root sensory neurons are usually simultaneously involved.
The LMN weakness and sensory loss tend to be most marked distally in the limbs.

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.



ONE SPINAL ROOT OR ONE INDIVIDUAL PERIPHERAL NERVE.
the LMN signs
are found only in the muscles
supplied by the particular nerve
root or peripheral
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** (neck of the fibula).
Median (CTS)

LMN WEAKNESS OF ONE SPINAL ROOT

Bulbar vs Pseudobulbar palsy

Bulbar (Nuclear)

- **Bilateral affection of LMN** defect of IX-XII CN or their nuclei in Medulla Oblongata
- LMN lesion Peripheral Palsy
- Dysphagia (liquid>solid), nasal regurgitation, slurred speech
- Nasal speech, wasted tongue with fasciculation, absent gag reflex
- Cause: polyradiculoneuritis (GBS): brainstem lesions, tumors, meningoencephalitis, MND

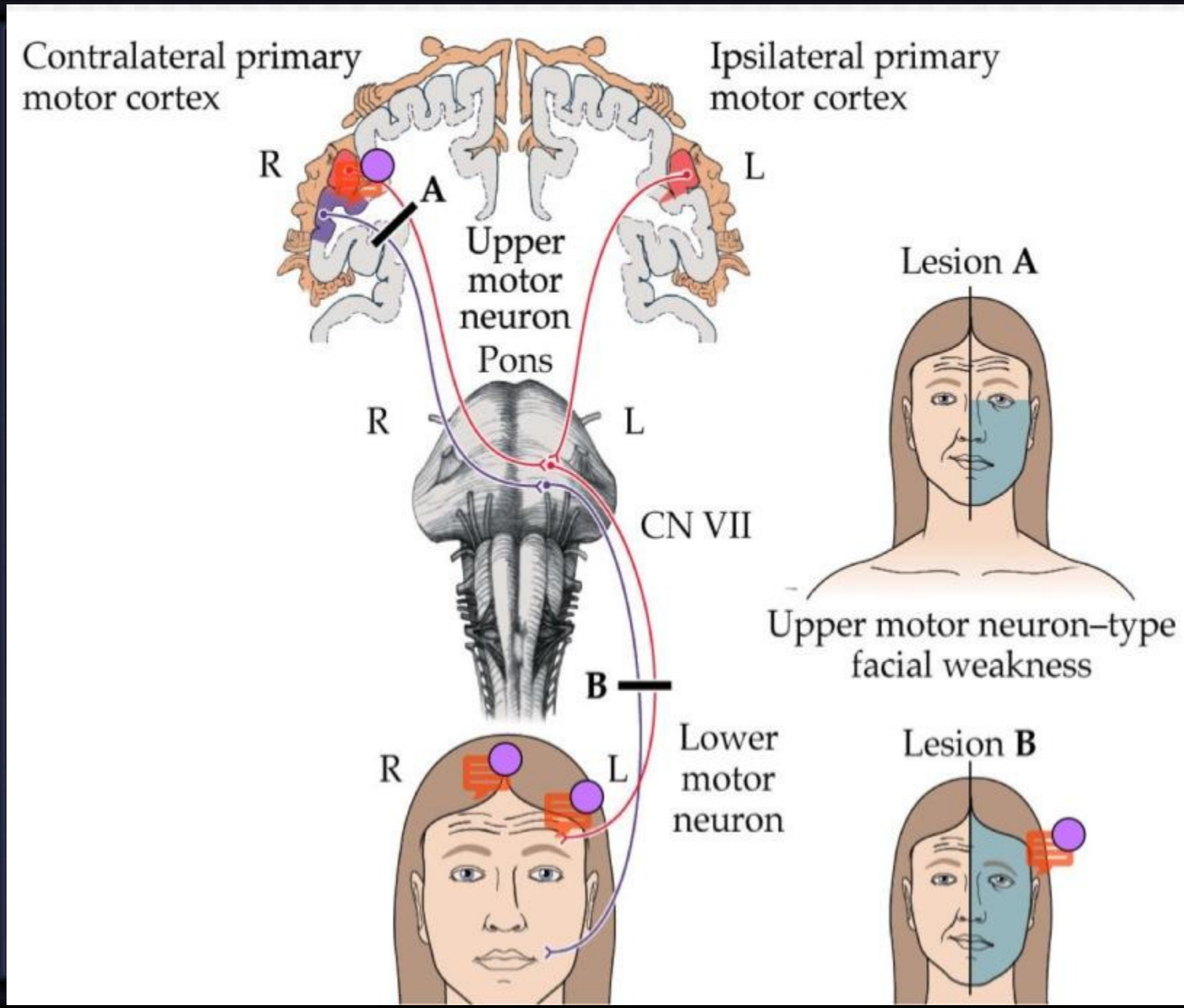
Pseudobulbar (Supranuclear)

- **Bilateral lesion of corticobulbar tract**
- **UMN defect** of central palsy
- IX-XII cranial nerves
- Dysphagia, dysarthria, emotional lability (unprovoked crying or laughing)
- Slow indistinct speech, spastic tongue, brisk jaw jerk (masseter reflex)
- Frontal release signs
- Cause: CVA, arteriosclerosis.

VIIth Cranial Nerve lesion

UMN VIIth CN 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 CN 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

Clinical Features	Site of Lesion
1. Ipsilateral LMN paralysis in the segment	Ant Horn cell
2. Ipsilateral spastic paralysis below the level	**UMN Lesion**
3. Ipsilateral band of cutaneous anesthesia	**Post Root damage**
4. Ipsilateral loss of tactile vibratory and proprioceptive sensations below the level	**Dorsal Column**
5. Contralateral loss of pain and temperature sensations below the level of the lesion.	**Lat Spinothalamic**
6. Contralateral but not complete loss of tactile sensation	**Ant Spinothalamic**
7. Ipsilateral Dystaxia	**Dorsal Spinocerebellar**
8. Contralateral Dystaxia	**Ventral Spinocerebellar**
9. Bilateral Pain & Temp Loss Upper limbs	**Ant Commisure**
10. All sensory lost	**Dorsal Horn**
11. All motor lost	**Ant Horn**