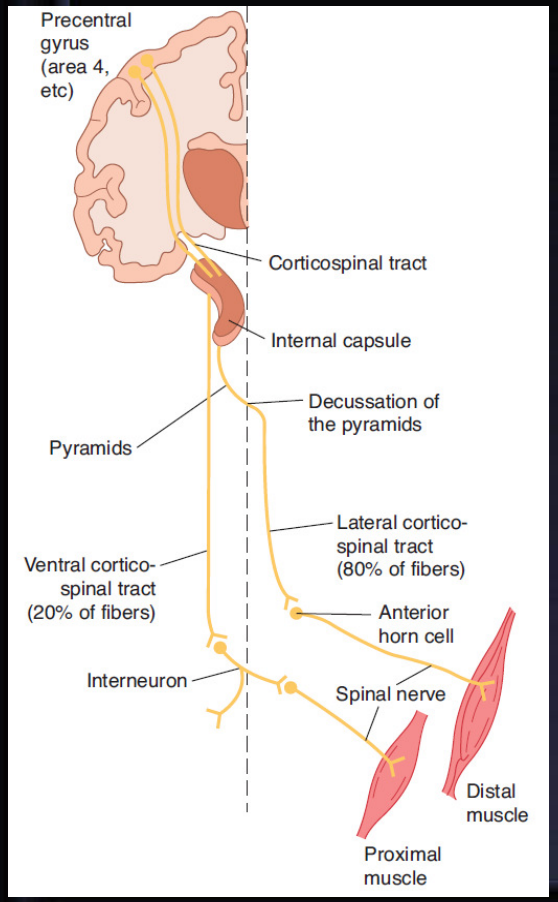
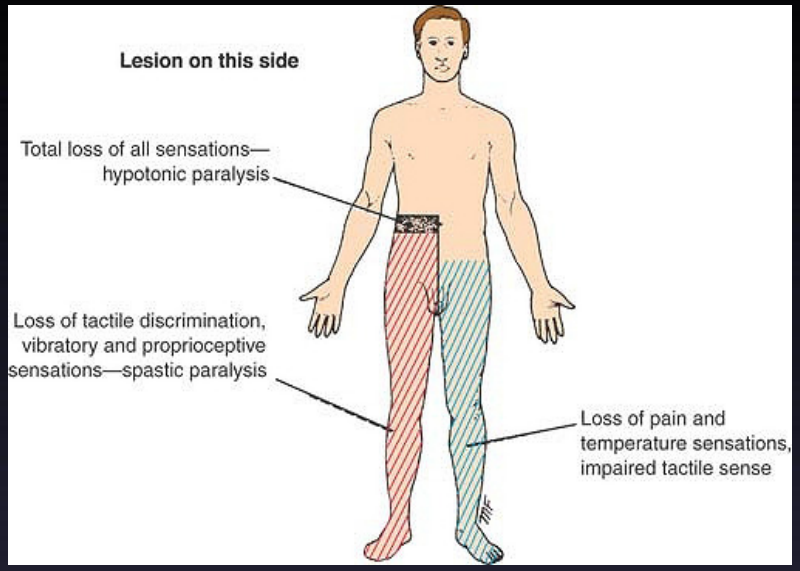
UPPER AND LOWER MOTOR NEURON LESIONS





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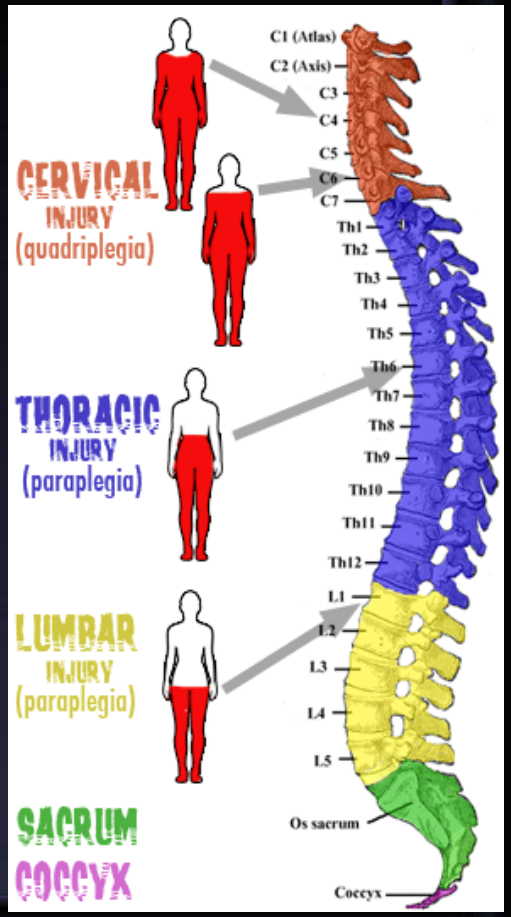
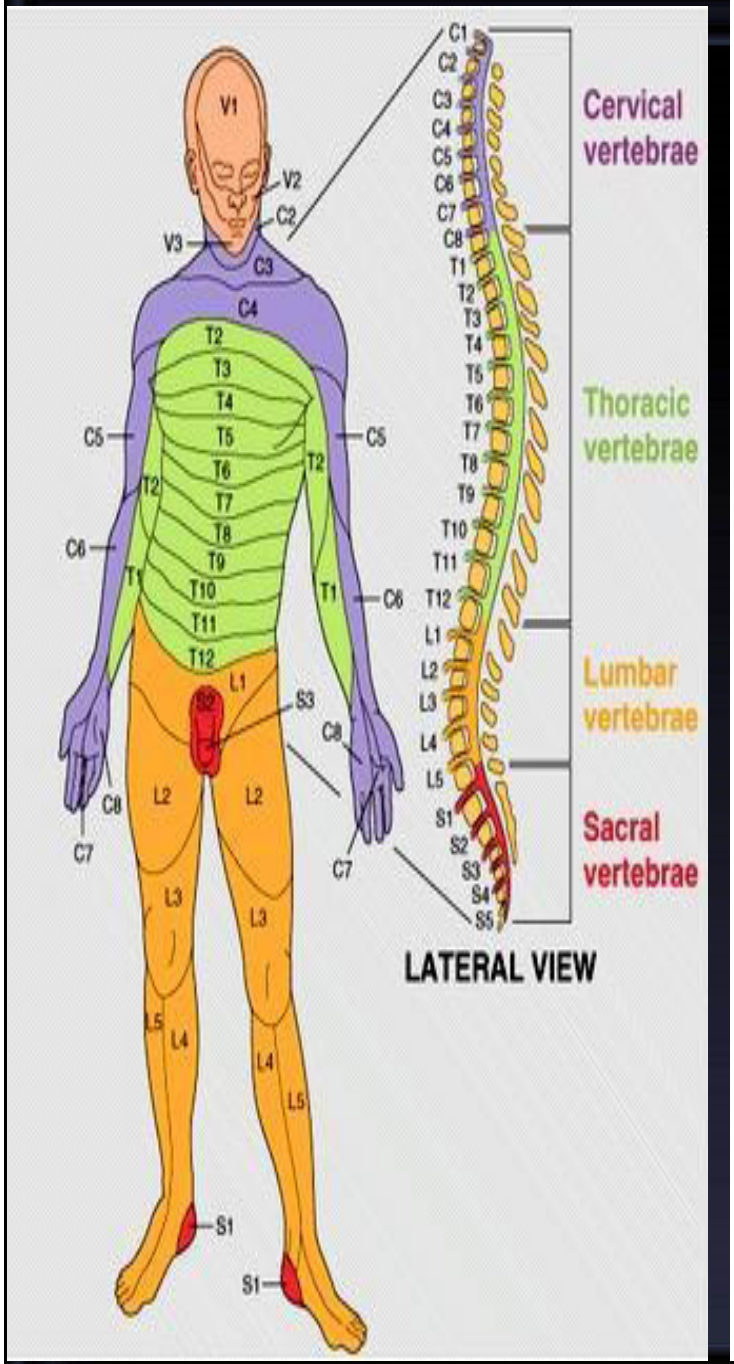
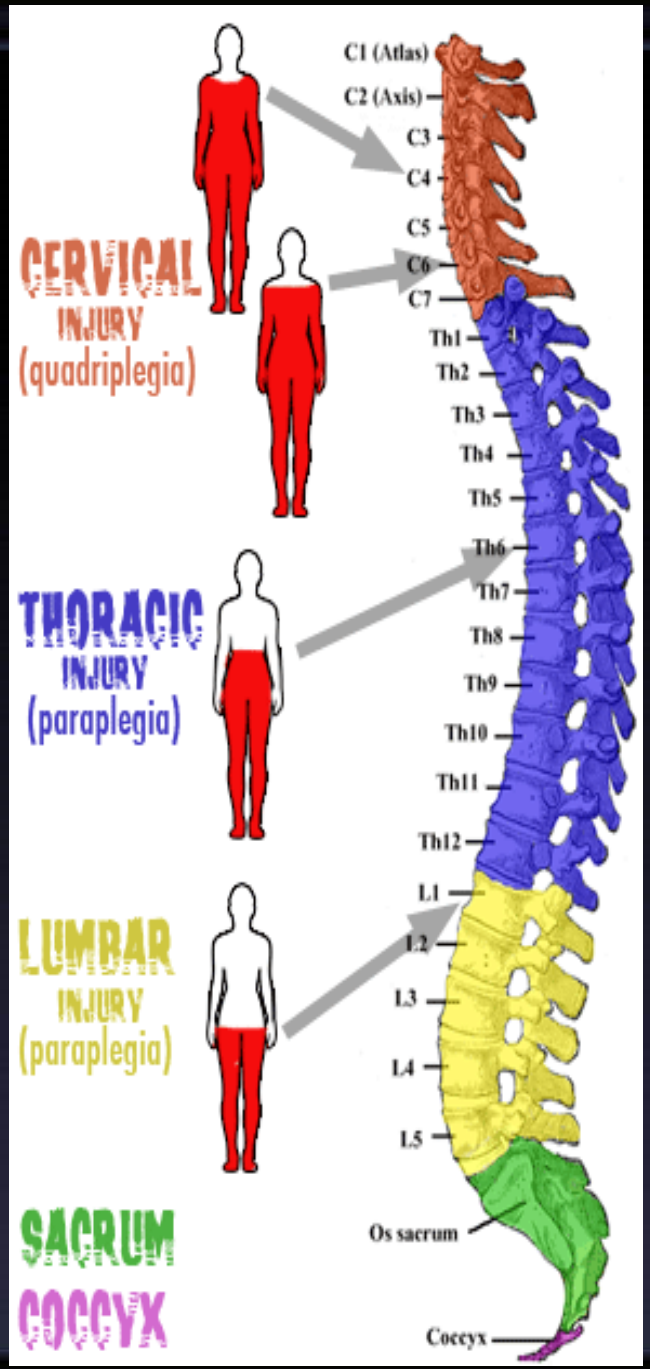


OBJECTIVES

At the end of this lecture you should be able to

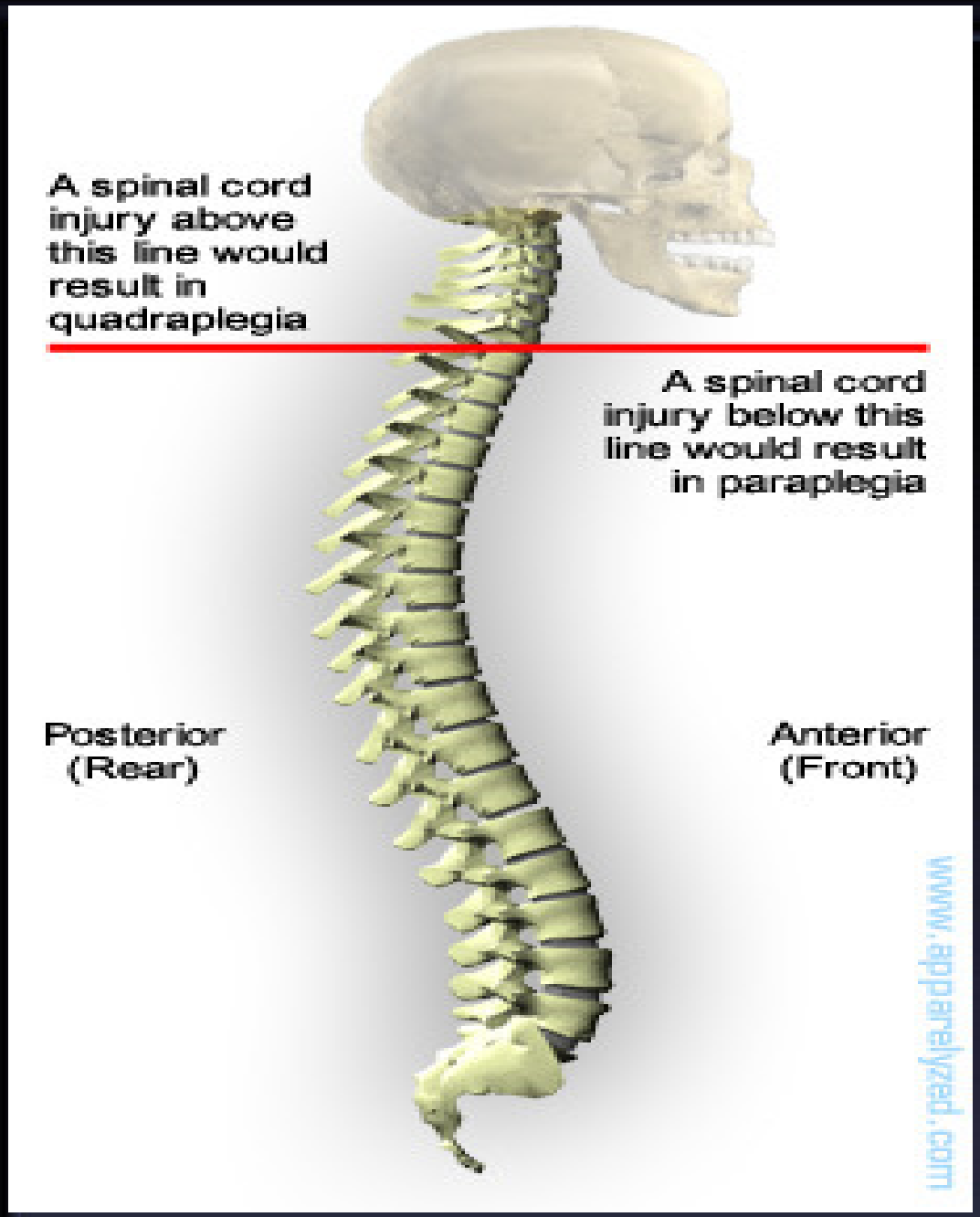
- Describe the functional anatomy of upper and lower motor neurons
- Describe and differentiate the features of upper and lower motor neuron lesions
- Explain features of Brown Sequard Syndrome
- Correlate the site of lesion with pattern of loss of sensations
- Describe facial, bulbar and pseudobulbar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





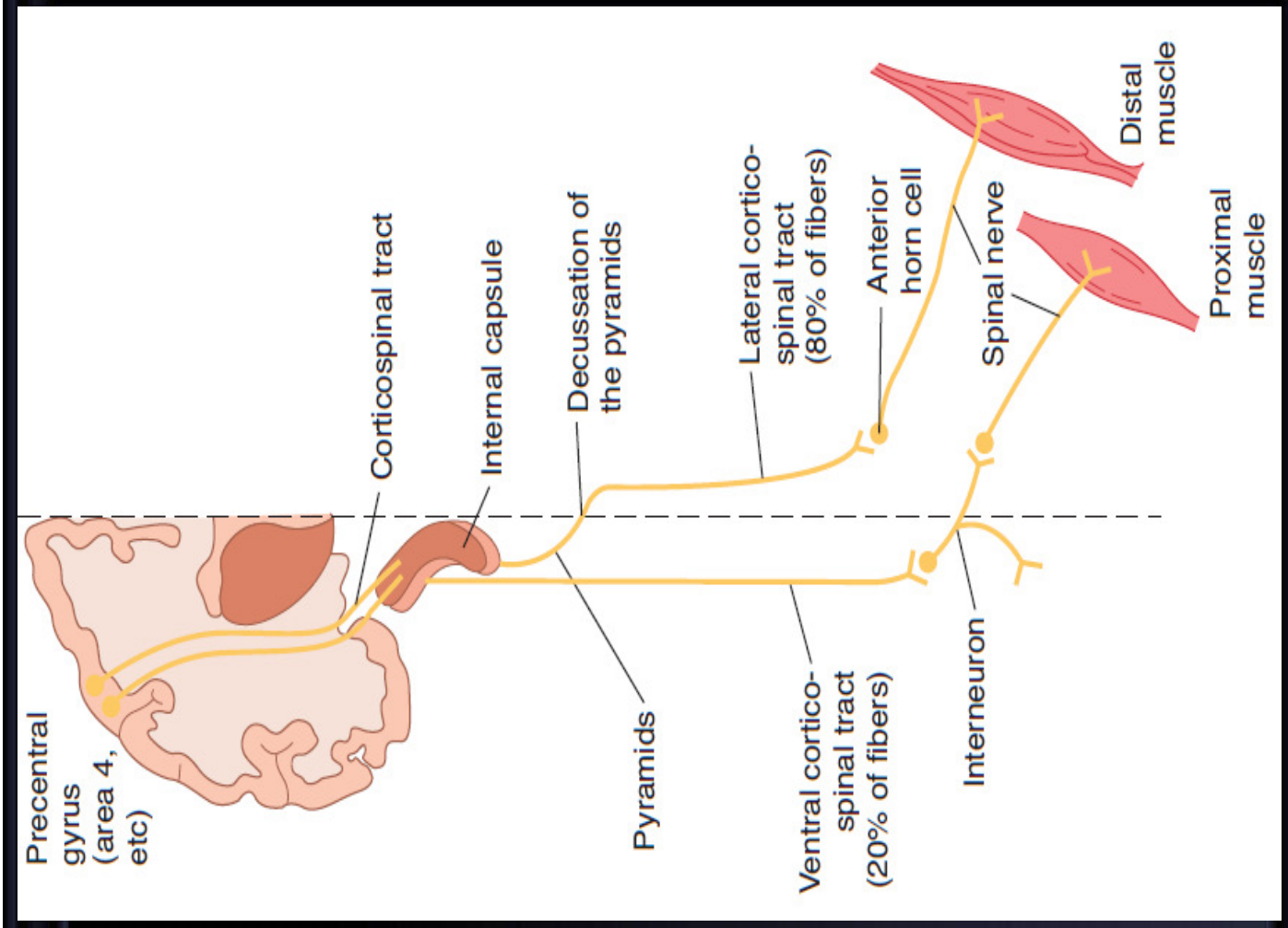
31 segments

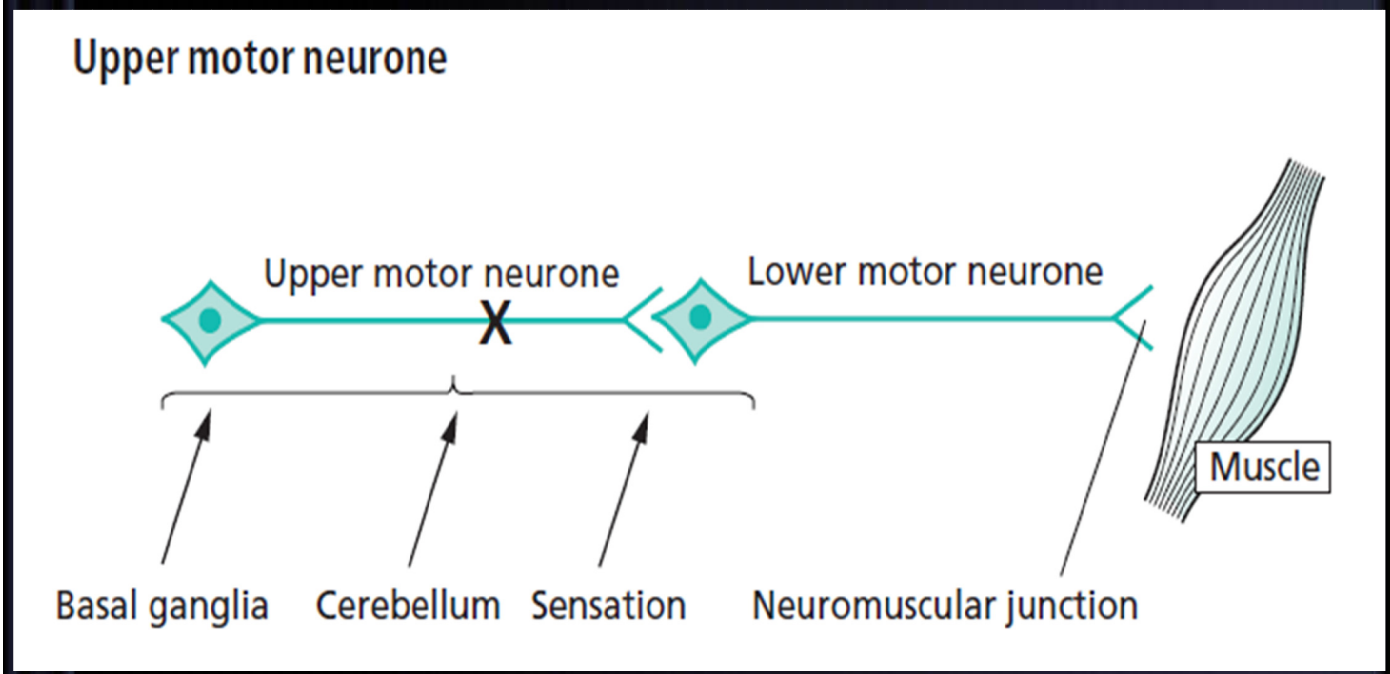
Embryological development→ growth of cord lags behind → mature spinal cord ends at L1



Upper cervical cord lesions produce quadriplegia and weakness of the diaphragm

Lesions at C4-C5 produce quadriplegia


Distal muscle
Proximal muscle
Anterior horn cell
Spinal nerve
Lateral cortico-spinal tract (80% of fibers)
Decussation of the pyramids
Internal capsule
Corticospinal tract
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

COMAPRISON BETWEEN UPPER & LOWER MOTOR NEURON LESIONS

UMN LESION

- Paralysis affect movements
- Wasting not pronounced.
- Spasticity Muscles hypertonic (Clasp Knife).
- Tendon reflexes increased.
- Superficial reflexes diminished
- Babinski's sign +ve,
- NCV- normal
- No denervation potentials in EMG

LMN LESION

- Individual muscle or group of muscles are affected.
- Wasting pronounced.
- Flaccidity. Muscles hypotonic.
- Tendon reflexes diminished or absent.
- NCV- abnormal
- Denervation potentials in EMG (fibrillations)
- Muscle contractures
- Trophic changes in skin and nails

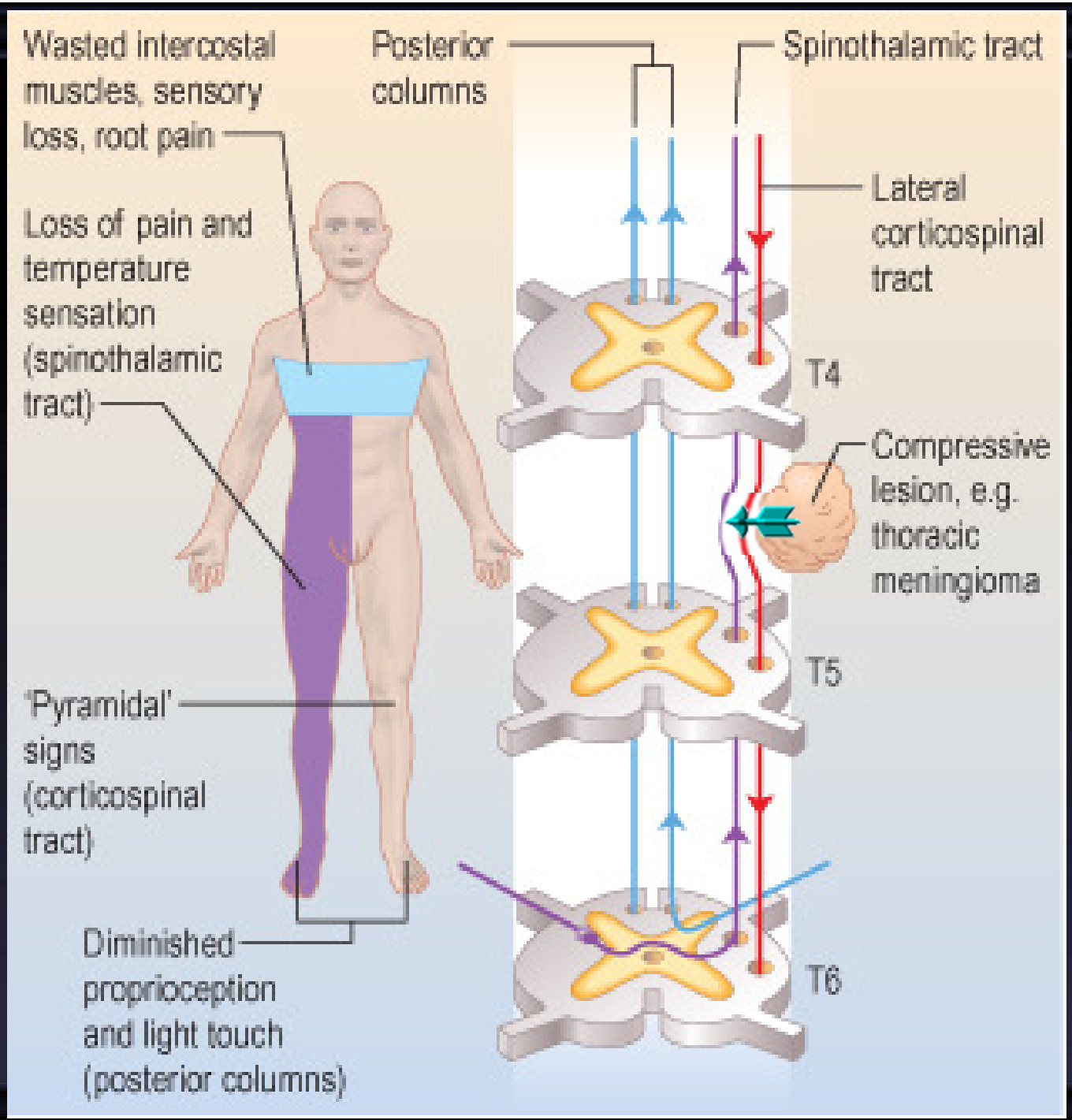
COMAPRISON BETWEEN UPPER & LOWER MOTOR NEURON LESIONS

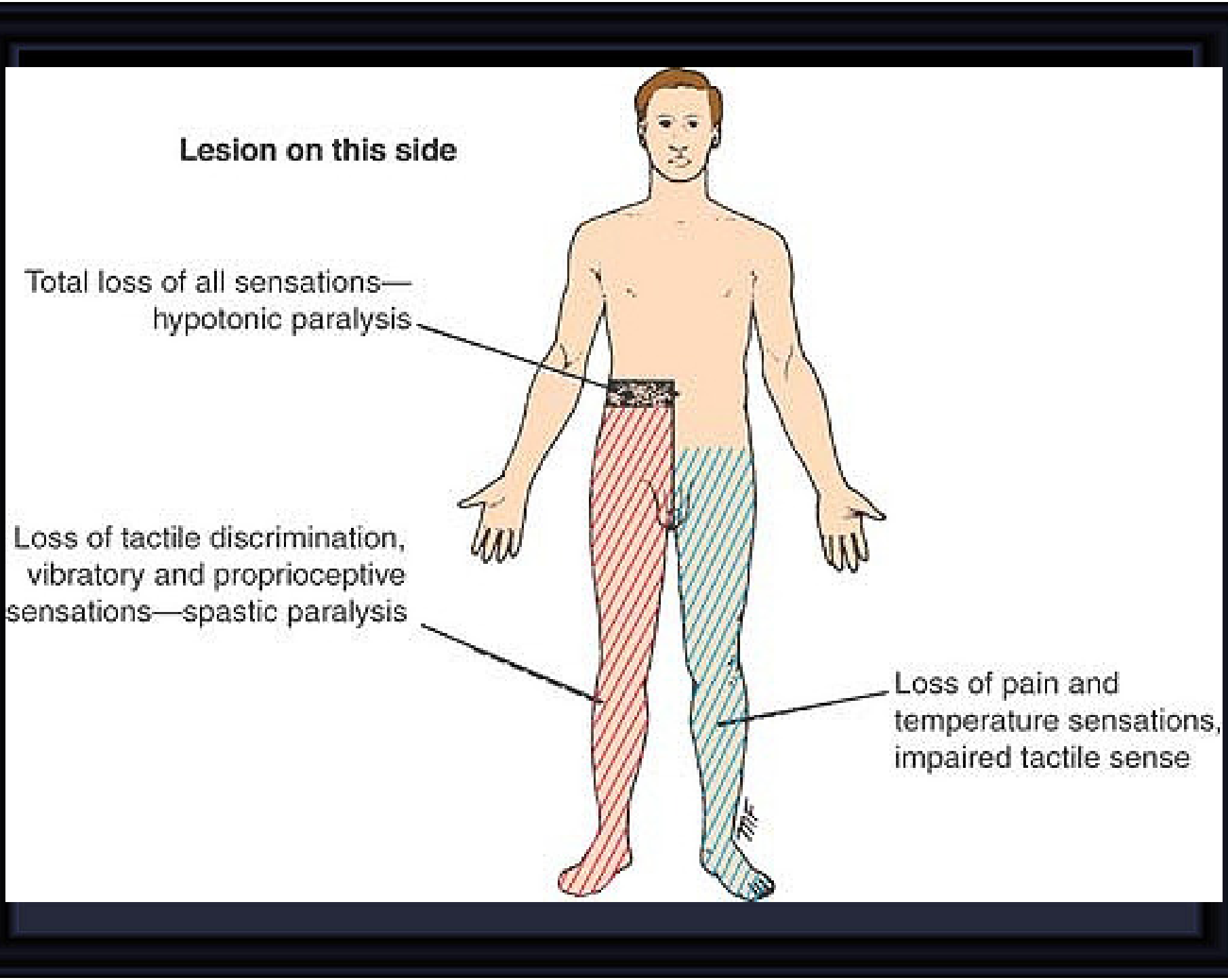
Characteristic of upper motor neurone lesions:

- no wasting;
- Loss of skilled finger/toe movements
- increased tone of clasp-knife type;
- weakness most evident in anti-gravity muscles;
- increased reflexes and clonus;
- extensor plantar responses.

Characteristics of lower motor neurone lesions:

- wasting;
- fasciculation (tapping produce it)
- decreased tone (i.e. flaccidity);
- weakness;
- decreased or absent reflexes;
- flexor or absent plantar responses.


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


Lesion on this side
Total loss of all sensations—hypotonic paralysis
Loss of tactile discrimination, vibratory and proprioceptive sensations—spastic paralysis
Loss of pain and temperature sensations, impaired tactile sense

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.

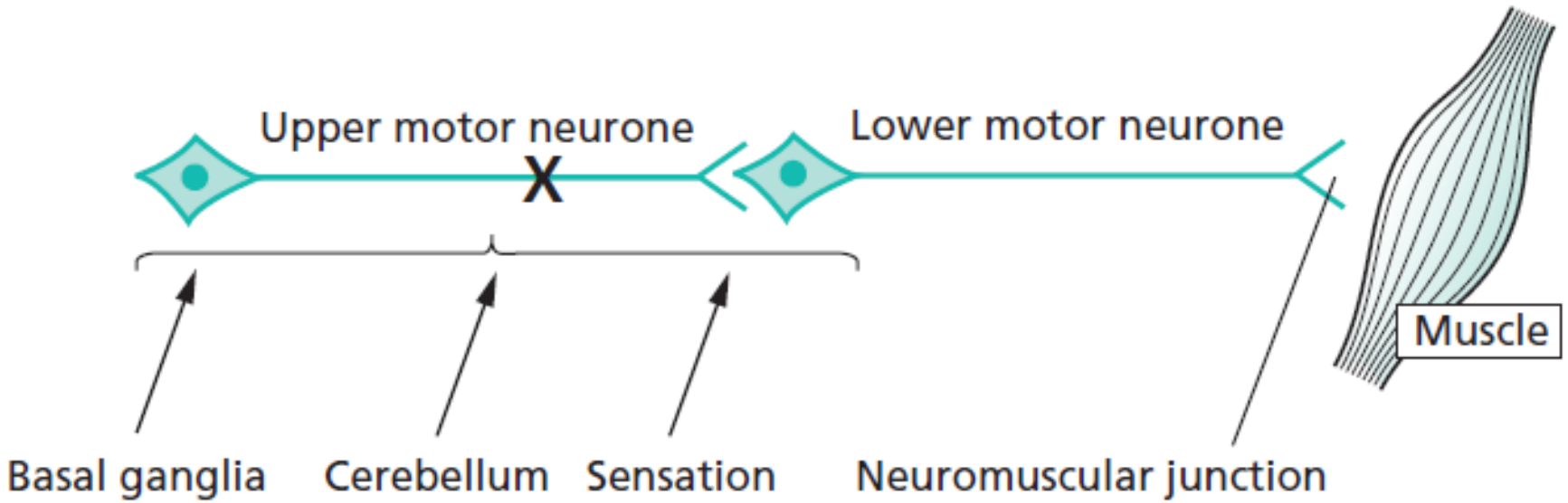
3. Ipsilateral band of cutaneous anesthesia in the segment of the lesion. This results from the destruction of the posterior root and its entrance into the spinal cord at the level of the lesion.

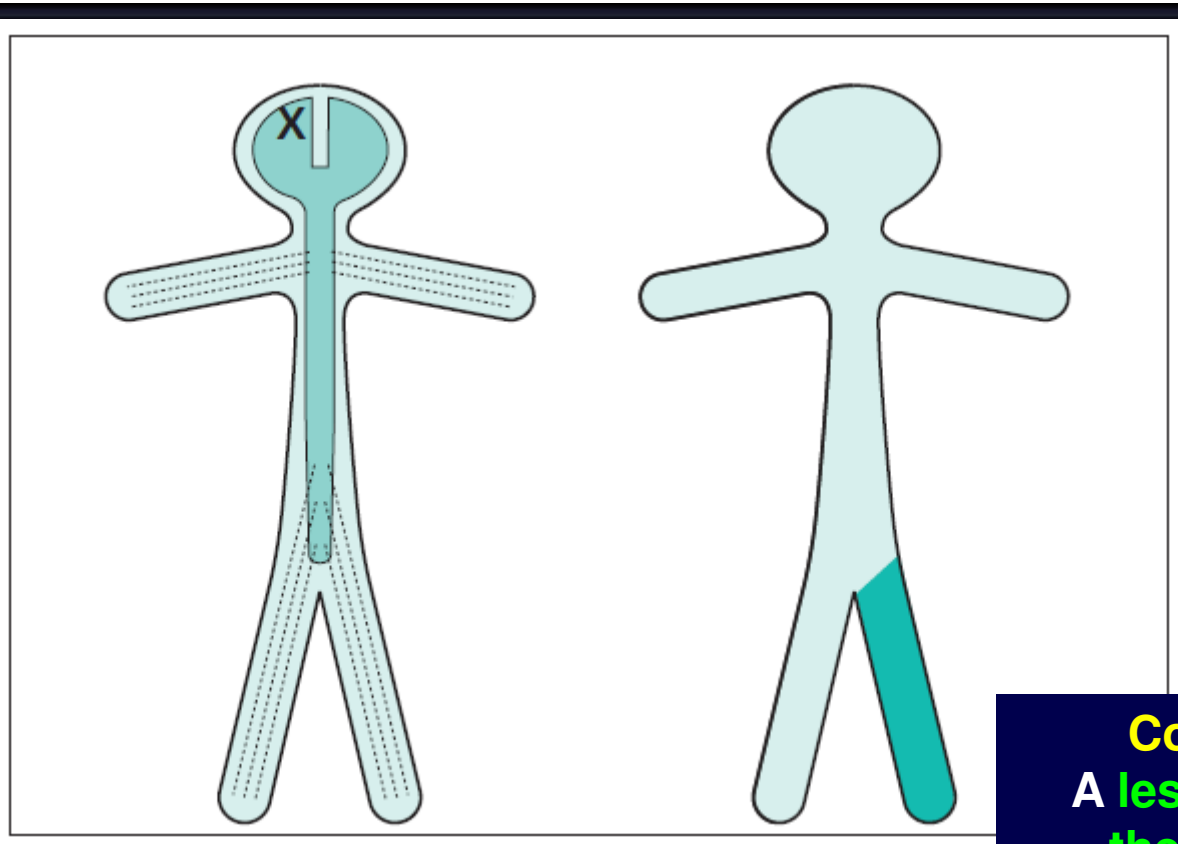
4. Ipsilateral loss of tactile discrimination and of vibratory and proprioceptive sensations below the level of the lesion. These signs are caused by destruction of the ascending tracts in the posterior white column on the same side of the lesion.

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.

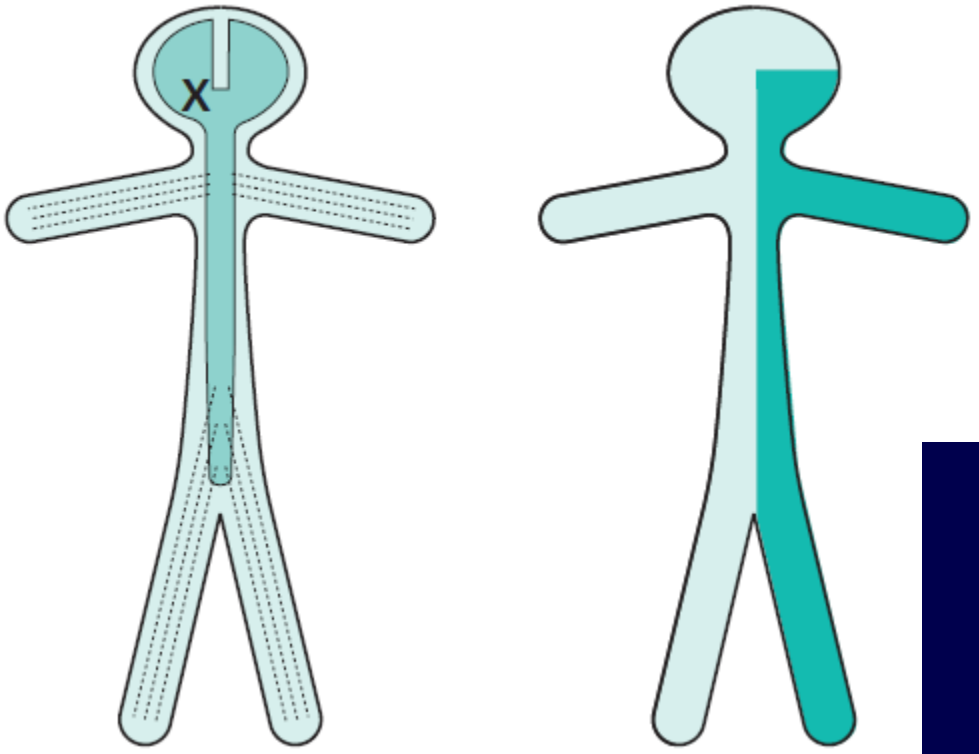
Upper motor neurone





Contralateral monoparesis

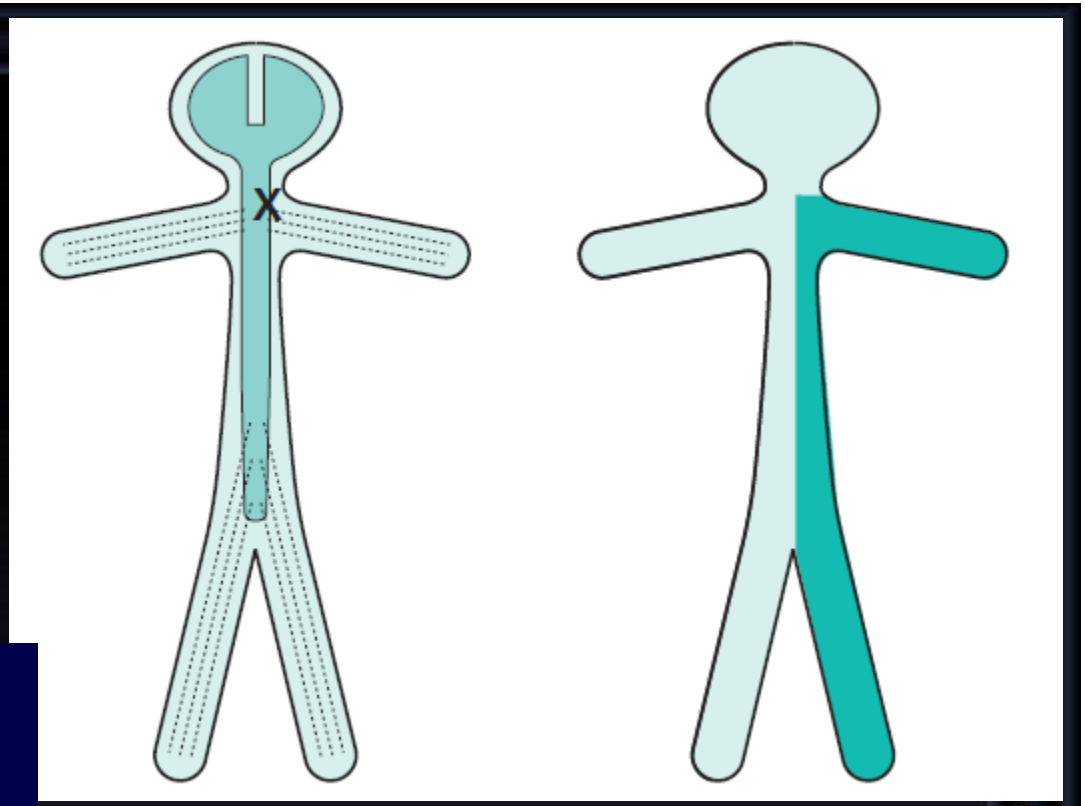
Contralateral monoparesis
A **lesion situated peripherally in the cerebral hemisphere**, i.e. involving part of the motor homunculus only, produces weakness of **part of the contralateral side of the body**, e.g. the contralateral leg. If the lesion also involves the adjacent sensory homunculus in the 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.

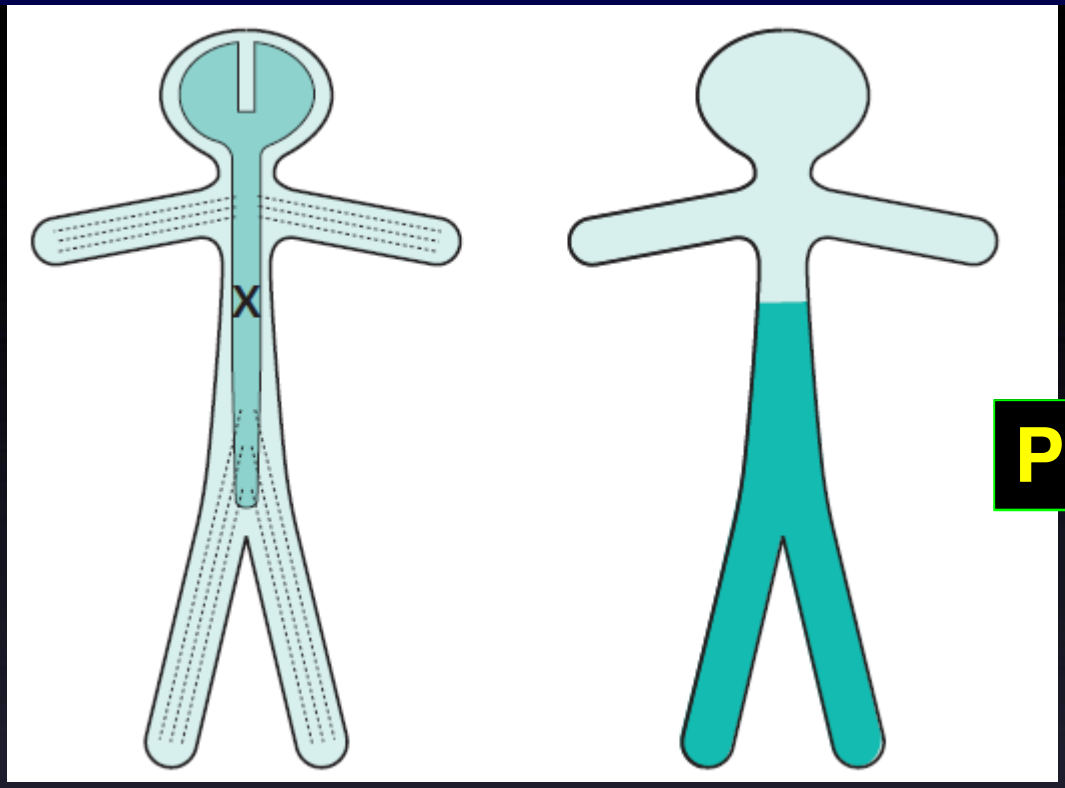


Ipsilateral hemiparesis

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.

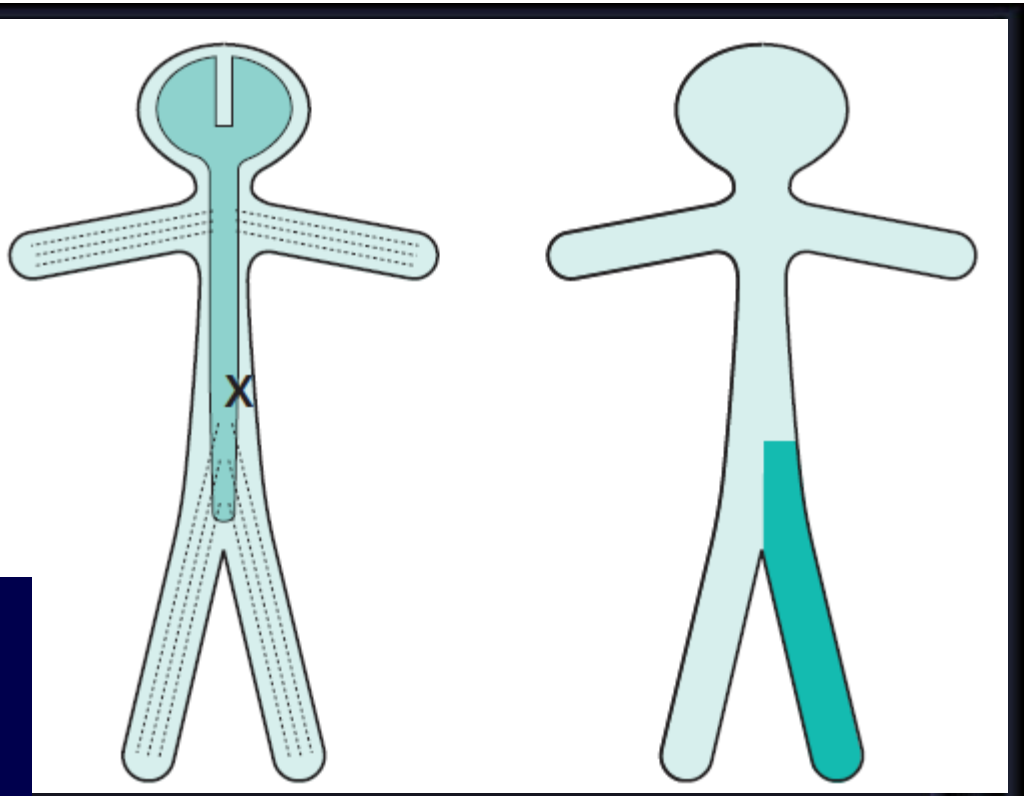
Ipsilateral hemiparesis

A spinal cord lesion more usually causes upper motor neurone 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.



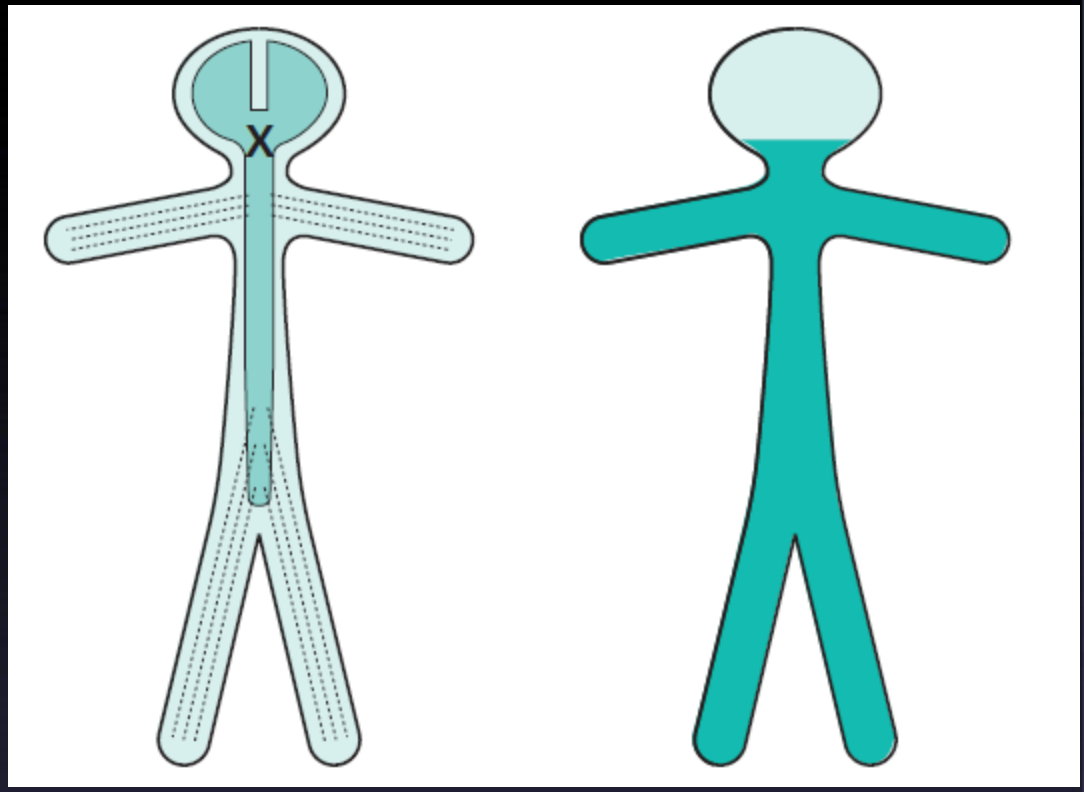
Ipsilateral monoparesis

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.

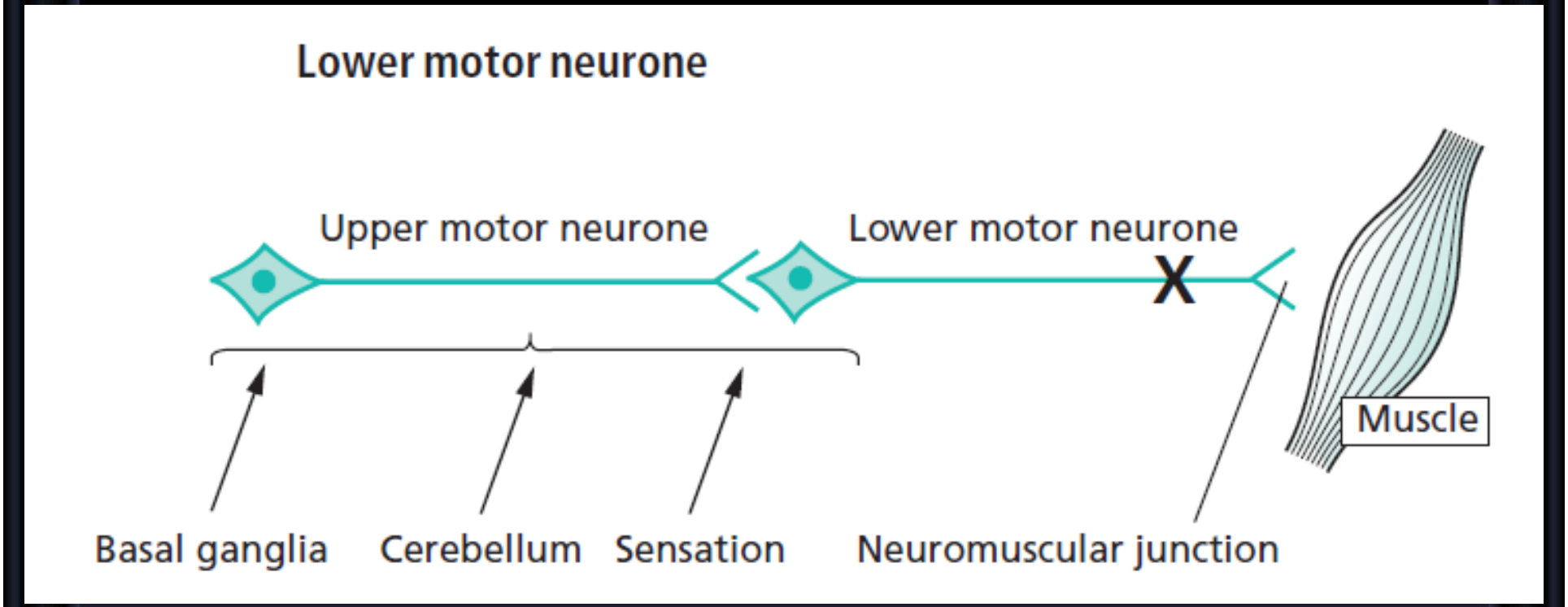
Ipsilateral monoparesis

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.

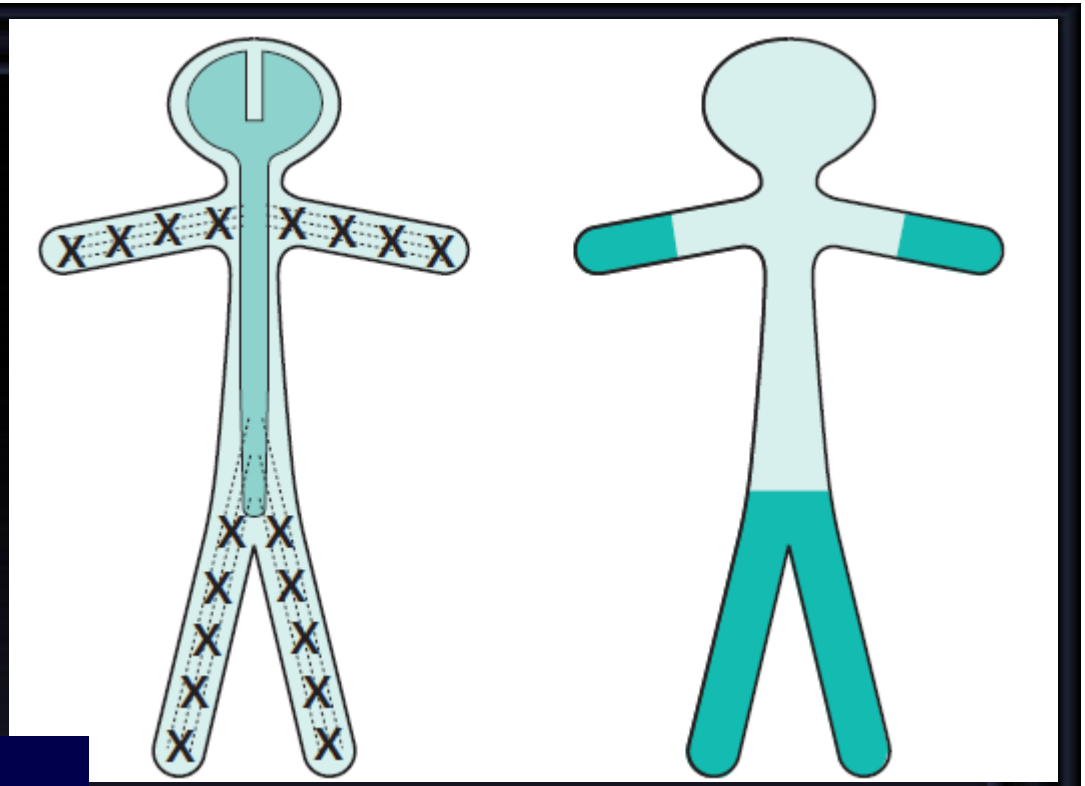
Tetraparesis or quadriparesis



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

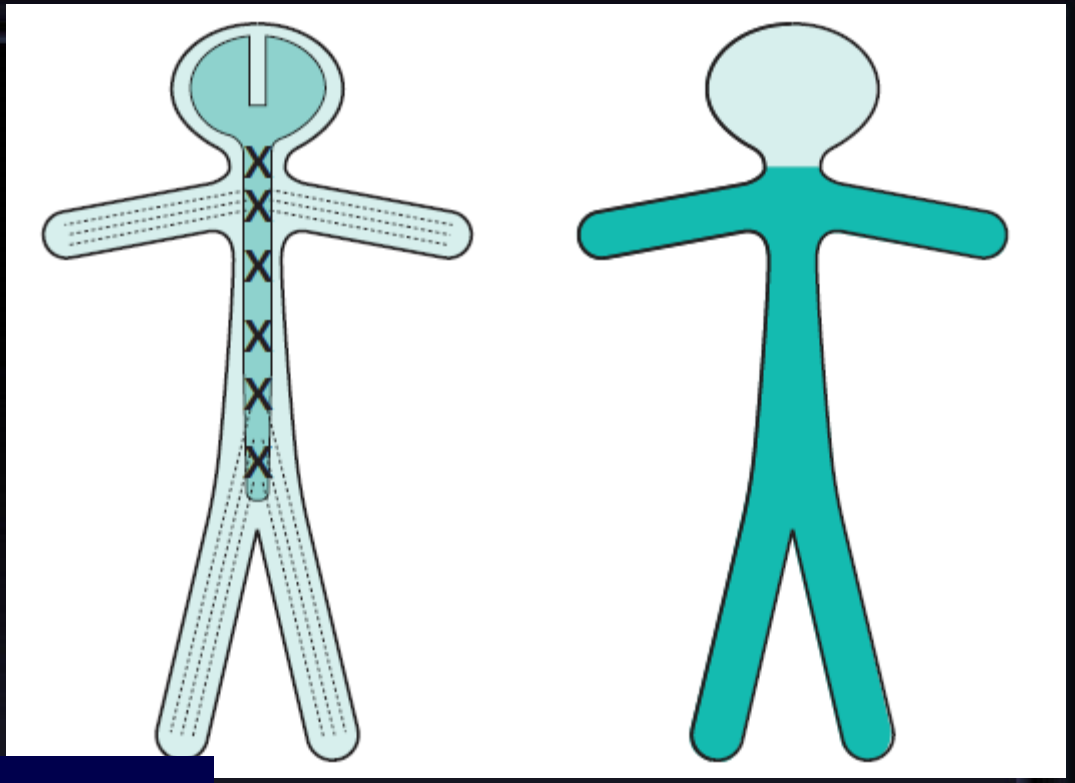

Lower motor neurone
Upper motor neurone
Lower motor neurone
X
Muscle
Basal ganglia
Cerebellum
Sensation
Neuromuscular junction

Generalized LMN weakness

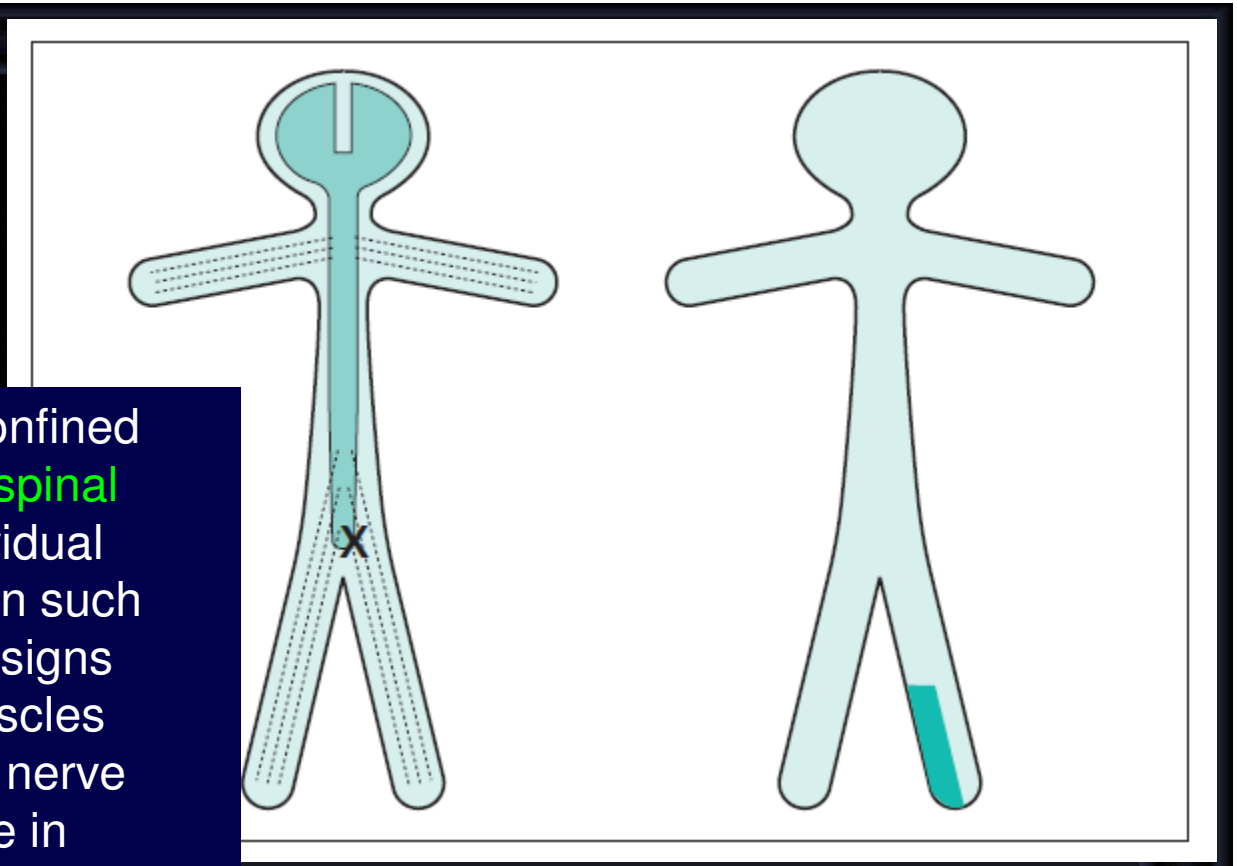


Generalized LMN weakness may also result from widespread damage to the axons of the LMNs. This is the nature of peripheral neuropathy (also called polyneuropathy). The axons of the dorsal root sensory neurones are usually simultaneously involved. The LMN weakness and sensory loss tend to be most marked 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

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

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

Unilateral facial weakness

- **Upper motor neurone (UMN lesions)** 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 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.

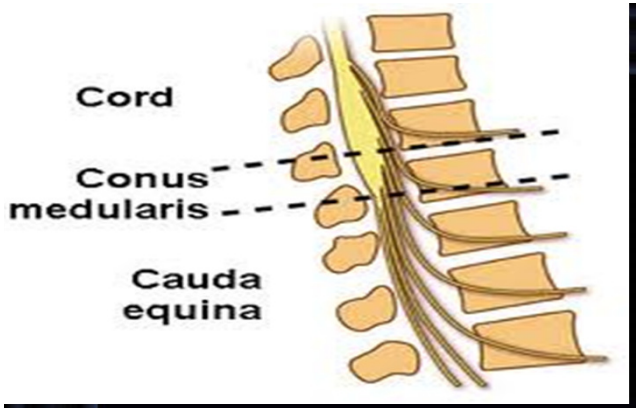
Intramedullary and Extramedullary Syndromes

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



Cauda equina and conus medullaris lesions

CONUS MEDULLARIS	CAUDA EQUINA
B/L saddle anaesthesia	asymmetric leg weakness and sensory loss
Prominent bowel, bladder symptoms, impotence	Relative sparing of bowel-bladder function
Bulbocavernous (S2-s4) and anal reflexes (s4-s5) are absent	Variable areflexia in lower extremities
Muscle strength largely preserved	Low back and radicular pain