



Upper motor neuron lesions & Lower motor neuron lesions

Objectives:

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

Done by:

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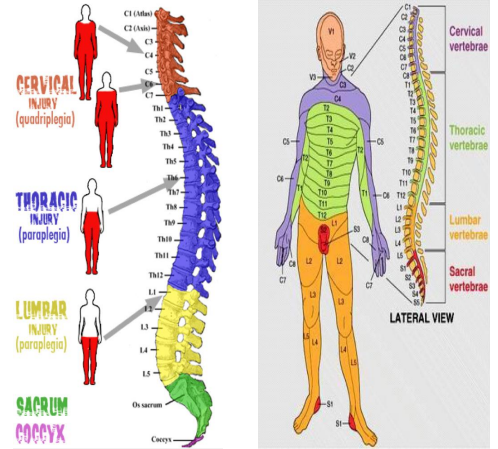
Colour index:

- important
- Numbers
- Extra

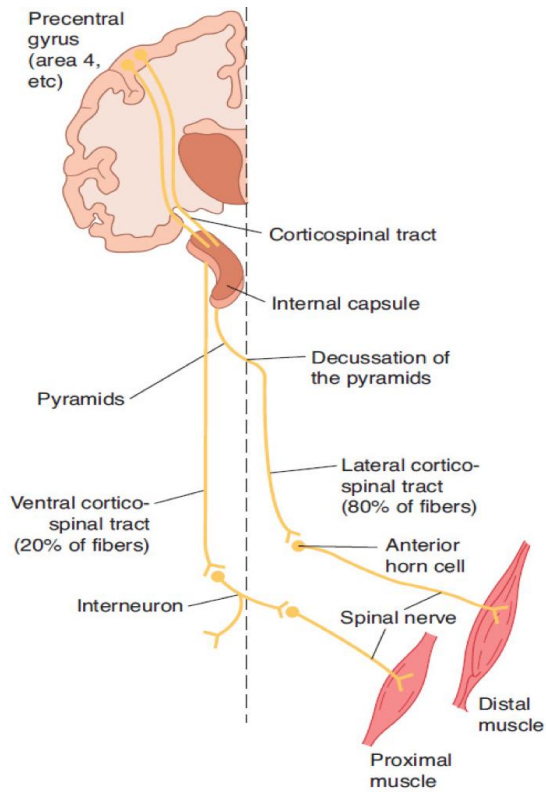
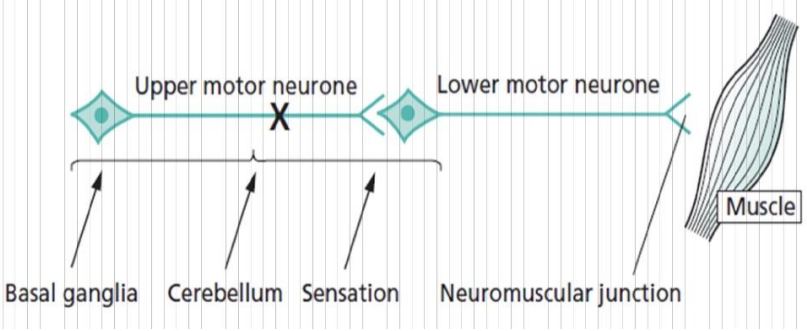
Spinal Cord

- Embryological development — growth of cord lags behind— mature spinal cords **ends at L1**
- It has **31 segments**

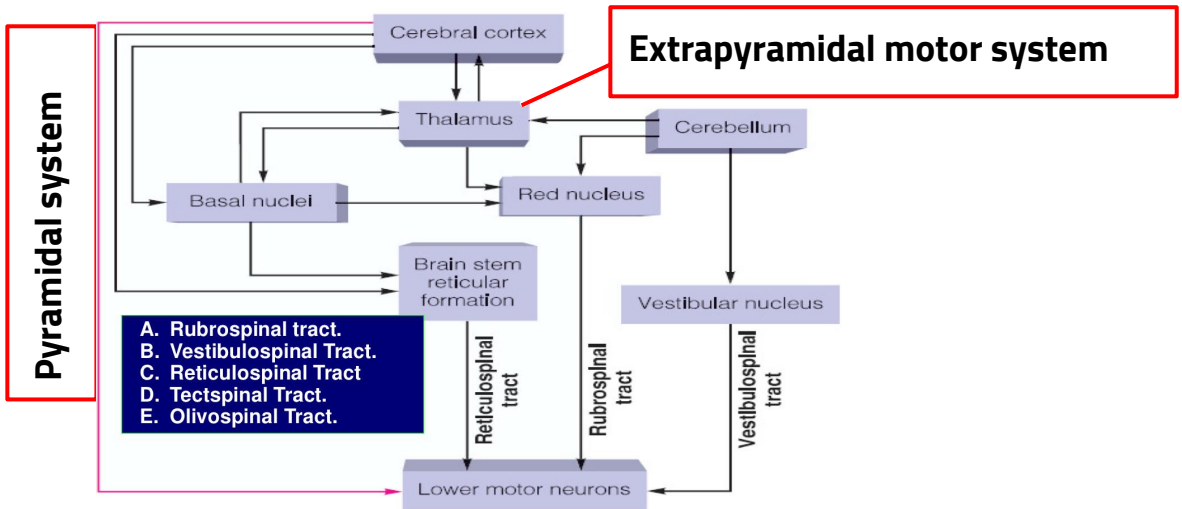
Upper and lower motor neuron



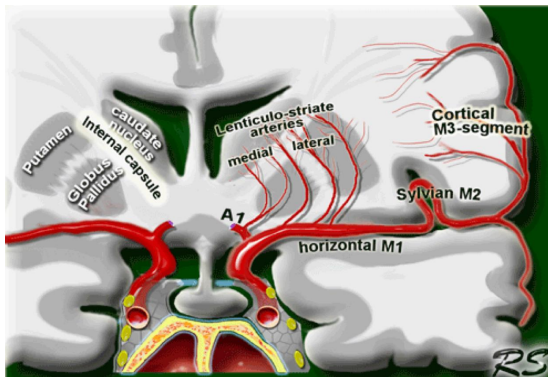
Upper motor neurone



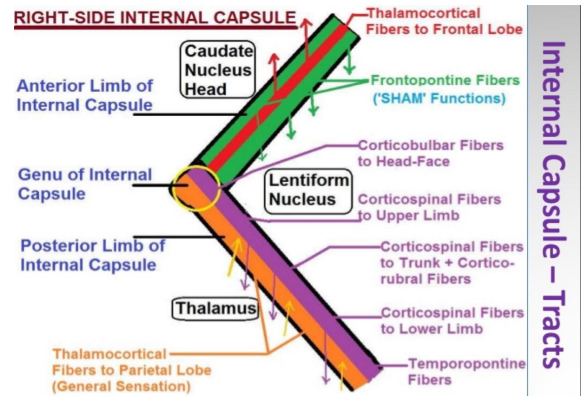
UMNs control lower LMNs through two different pathways



Lenticulostriate arteries from the middle cerebral artery



Internal capsule



Causes of UMNL and LMNL

Lower motor neuron lesion

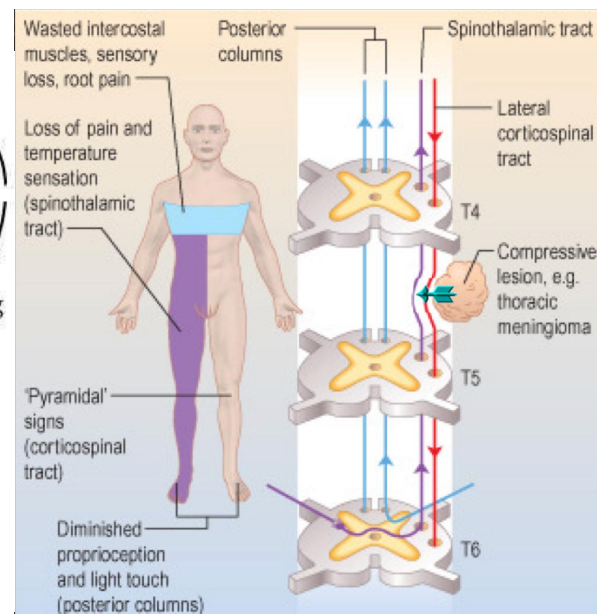
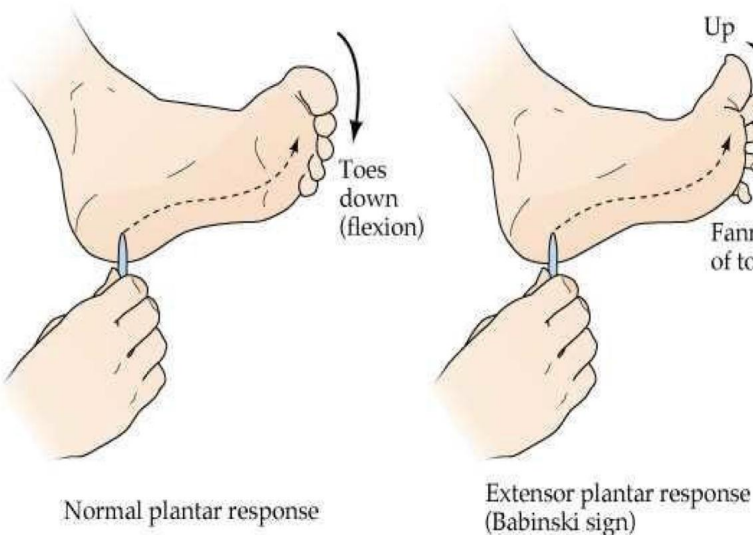
Can result from :

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

Upper motor neuron lesion

Can result from :

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



Comparison upper and lower motor neuron lesions

| | UMN lesion | LMN lesion |
|--|--|---|
| Pattern | Paralysis affect movements (The whole muscles are affected) | Individual muscle or group of muscles are affected |
| Wasting | Not pronounced (70-80) (there is muscle waste due to disuse *but less than LMN) (Tropic) | Pronounced (20-30) (more wasted than UMN because the muscle supply is damaged) (atropic) |
| Tone | <u>Spasticity muscles; hypertonic</u> (clasp knife) (increase <u>disinhibition</u>) ** | Tendon reflexes <u>diminished or absent</u> |
| Tendon reflex | Brisk /increased (due to increases gamma discharge) | Diminished or absent |
| Superficial reflexes | Absent (because it's polysynaptic)*** | Absent |
| NCV (nerve conduction velocity) | Normal | Decrease |
| Denervation potential (fibrillation) [on EMG ONLY] (LMN) | Absent | <u>Present</u> |
| Fasciculation (visible) (LMN) | Absent | <u>Present</u> |
| Trophic changes | Less | Pronounced in skin and nails |
| Clonus (rapid repetitive contraction) (UMN) | <u>Present</u> | Absent |
| Babinski's sign | <u>Extensor</u> plantar response (positive) | <u>Flexor</u> or absent plantar response |

*disuse means that the muscle is active and can respond to reflexes but you can't use or control the muscle that's why the wasting is less in UMN.

**the intensity of a reflex is regulated by the higher centers so, if the UMN is damaged ,the reflex will still occur but it will not be lowered by the UMN

***UMN has a main role in polysynaptic reflexes so, damage in UMN will inhibit the superficial reflex

Brown sequard syndrome "Hemisection of spinal cord"

Ipsilateral loss :

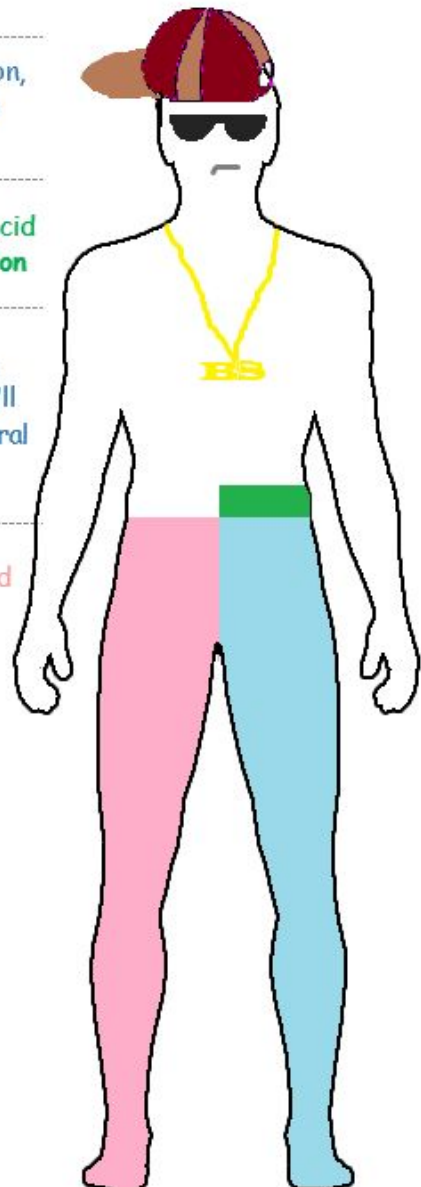
- fine touch , vibration , proprioception (dorsal column)
- leg ataxia (dorsal spinocerebellar)
- spastic paresis below lesion (lateral corticospinal)
- flaccid paralysis (vent horn destruction)
- dermatomal anesthesia (dorsal horn destruction)

Contralateral loss :

- loss of pain and temp (lateral spinothalamic)
- loss of crude touch and pressure (vent spinothalamic)
- minor contralat muscle weakness (vent corticospinal)
- leg ataxia (vent spinocerebellar)

The Rap of Brown-Séquard

| | |
|--|---|
| I'm a h IP Vi P I walk with a limp | Ipsilateral loss of Proprioception, Vibration + Paralysis (limp to remind of paralysis) |
| I'm At someone's Flat I sense I'm all that | At the level of the lesion = Flaccid paralysis and loss of all sensation |
| But they don't know I started from Below And Up I Go , so watch those toes | Below the level of the lesion, UMN/Spastic paralysis → you'll have a positive Babinski ipsilateral to lesion (up going toes) |
| Or ConTempl ate the Pain I will make | Contralateral loss of Temp and Pain sensation |



Brown sequard syndrome "Hemisecation of spinal cord" "Explained"

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 grey 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 damage, 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 result from the destruction of the posterior root and its entrance into the spinal cord at the level of the lesion

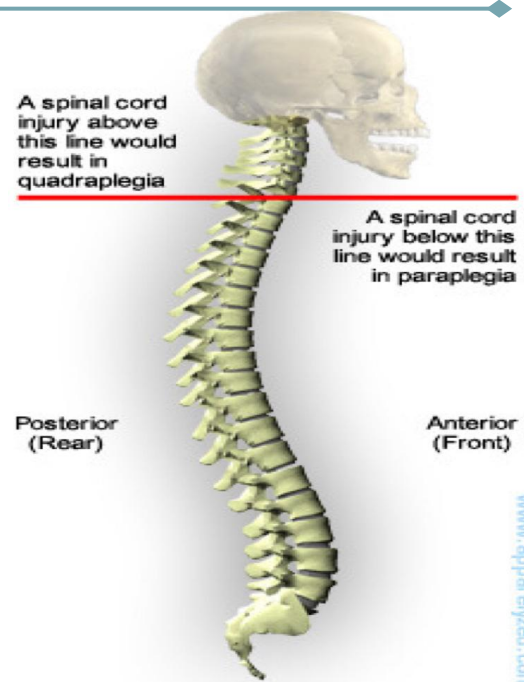
4- ipsilateral loss of tactile discrimination and of vibratory and proprioceptive sensation below the level of the lesion. These signs are caused by destruction of the ascending tracts in the posterior white column on the same side of the lesion.

5- contralateral loss of pain and temperature sensations below the level of the lesion. This 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 important because discriminative touch travelling in the ascending tracts in the contralateral posterior white column remains intact .

Lesions of the spinal cord

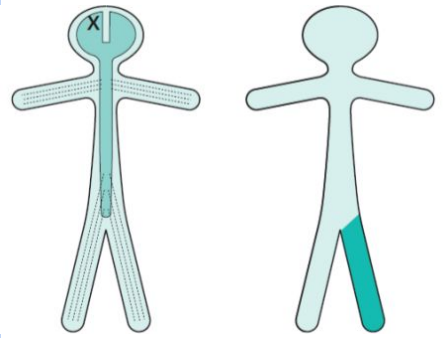
- **Upper cervical cord lesions** produced quadriplegia and weakness of the diaphragm
- **Lesions at C4-C5** produce quadriplegia
 - **Hemiparesis** means weakness
 - **Hemiplegia** means total paralysis



Comparison between lesions:

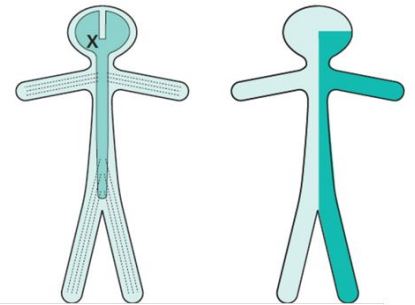
Contralateral monoparesis

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.



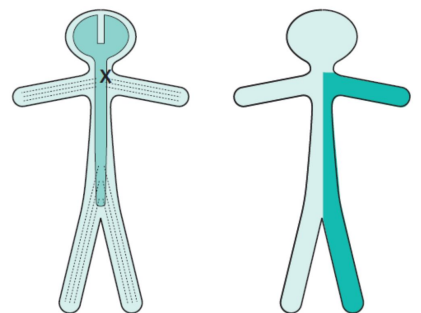
Contralateral hemiparesis

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.



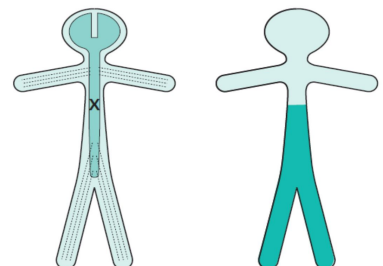
Ipsilateral hemiparesis

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.



Paraparesis

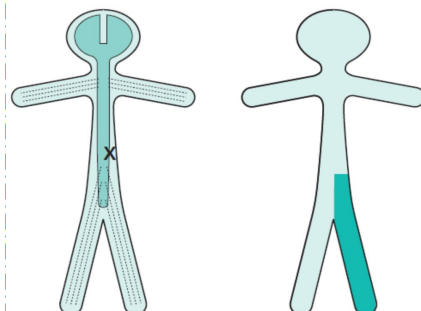
Paraparesis, if **the lesion is at or below the cervical portion** of the spinal cord.



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.

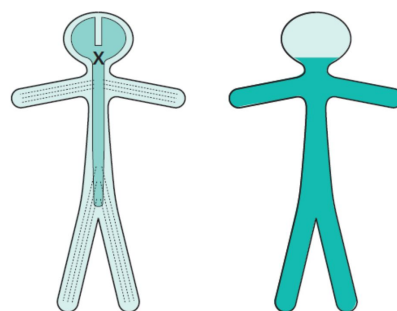
Ipsilateral monoparesis

A **unilateral lesion in the spinal cord below the level of the neck** produces **upper motor neuron 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**.

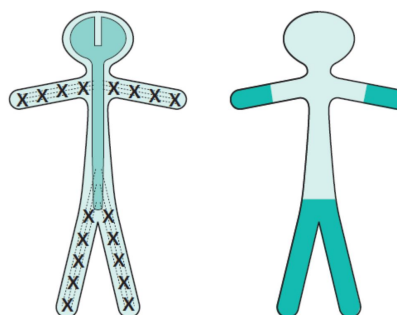


Tetraparesis or quadriparesis

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

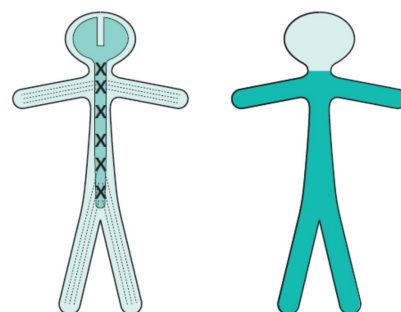


Generalized LMN weakness may also result from **widespread damage to the axons of the LMNs**. This is the nature of peripheral neuropathy (also called **polyneuropathy**). The axons of the dorsal root sensory neurons are usually simultaneously involved. The LMN weakness and sensory loss tend to be most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



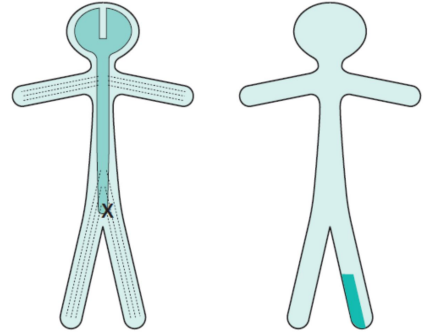
Generalized LMN weakness

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 of one spinal root

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.



Motor neuron disease

Dr. Shahid said that he **won't** ask you about it.

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

Ipsilateral spastic paralysis & loss of joint/position sense. contralateral loss of pain/temperature sensation.

Bulbar and pseudobulbar palsy

| Bulbar palsy | Pseudobulbar palsy |
|--|---|
| B/L <u>LMN</u> defect of IX,X,XI,XII cranial nerves | B/L <u>UMN</u> defect of IX,X,XI,XII cranial nerves |
| Dysphagia (liquid>solid), nasal regurgitation, slurred speech | Dysphagia, dysarthria, emotional lability |
| <u>Nasal speech</u> , <u>wasted tongue</u> with fasciculation, absent gag reflex | Slow indistinct speech, <u>spastic tongue</u> , brisk jaw jerk Frontal release signs |

Unilateral facial weakness

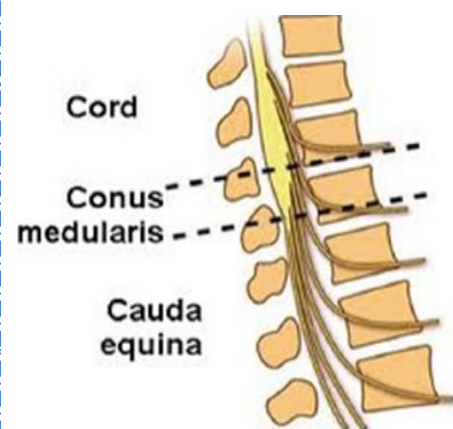
→ **Upper motor neuron lesion** cause weakness of the lower part of the face on the opposite side. Frontalis is spared: normal furrowing of the brow is preserved; eye closure and blinking are largely unaffected.

Because it receives **dual innervation** (R & L)

→ **Lower Motor Neuron 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.

Cauda equina and conus medullaris lesions

| Conus medullaris | cauda equina |
|---|--|
| Bilateral 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 <u>areflexia</u> in lower extremities |
| Muscle strength largely preserved | Low back and radicular pain |



Intramedullary and Extramedullary Syndromes ★

Extramedullary lesions

- **radicular pain** is often prominent
- there is **early sacral sensory loss** (lateral spinothalamic tract)
- **spastic weakness in the legs** (corticospinal tract) due to the superficial location of leg fibers in the corticospinal tract
- **Early UMN signs**

Intramedullary lesion

- Tend to produce **poorly localized burning pain**, rather than radicular pain
- **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**

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. (e.g: urinate in front of people)

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.

| Clinical feature | Site of lesion |
|--|-------------------------|
| Ipsilateral LMN paralysis in the segment | Anterior horn cell |
| Ipsilateral spastic paralysis below the level | UMNL |
| Ipsilateral band of cutaneous anesthesia | Posterior root damage |
| Ipsilateral loss of tactile, vibratory and proprioceptive sensations below the level of the lesion | Dorsal column |
| Contralateral loss of pain and temperature sensations below the level | Lateral spinothalamic |
| Contralateral but not complete loss of tactile sensation | Anterior spinothalamic |
| Ipsilateral dystaxia | Dorsal spinocerebellar |
| Contralateral dystaxia | Ventral spinocerebellar |
| Bilateral pain and temperature loss upper limbs | Anterior commissure |
| All sensory lost | Dorsal horn |
| All motor lost | Anterior horn |

1. Lower motor neuron lesion can result from :
 - A. Cerebral stroke by hemorrhage, thrombosis or embolism
 - B. Anterior horn cell lesions
 - C. Spinal cord transection or hemisection
 - D. Posterior horn cell lesion
2. NCV (nerve conduction velocity) in LMN lesion is :
 - A. Increased
 - B. Decrease
 - C. Normal
 - D. Absent
3. Hemiparesis means :
 - A. Partial paralysis
 - B. Complete paralysis
 - C. Weakness
 - D. Sensory impairment
4. contralateral loss of pain and temperature sensations below the level of the lesion is due to :
 - A. destruction of the crossed anterior spinothalamic tracts on the side of the lesion
 - B. destruction of the crossed lateral spinothalamic tracts on the same side of the lesion
 - C. destruction of the descending tracts
 - D. damage to the neurons on the anterior grey column
5. Tendon reflex in UMN lesion is :
 - A. Brisk /increased
 - B. Absent
 - C. Decreased
 - D. Lost
6. Bilateral lesion in the spinal cord lead to :
 - A. quadriplegia
 - B. monoplegia
 - C. paraplegia
 - D. hemiplegia
7. Which one of the following is a manifestation of brown sequard syndrome ?
 - A. Loss of dorsal column sensation above the level of lesion.
 - B. loss of all reflexes below the level of lesion
 - C. contralateral loss of pain and temperature below the level of lesion.
8. Contralateral hemianesthesia is:
 - A. Loss of sensation on some parts of the opposite side of the body
 - B. loss of sensation of both legs
 - C. loss of sensation on the upper part of the opposite of the body
 - D. loss of all sensation on the opposite side of the body.
9. A patient is having poliomyelitis, what type of lesion are you expecting him to have?
 - A. .Upper motor neuron lesion
 - B. .Lower motor neuron lesion

Answers:
 1. B
 2. B
 3. C
 4. B
 5. A
 6. C
 7. C
 8. B
 9. B