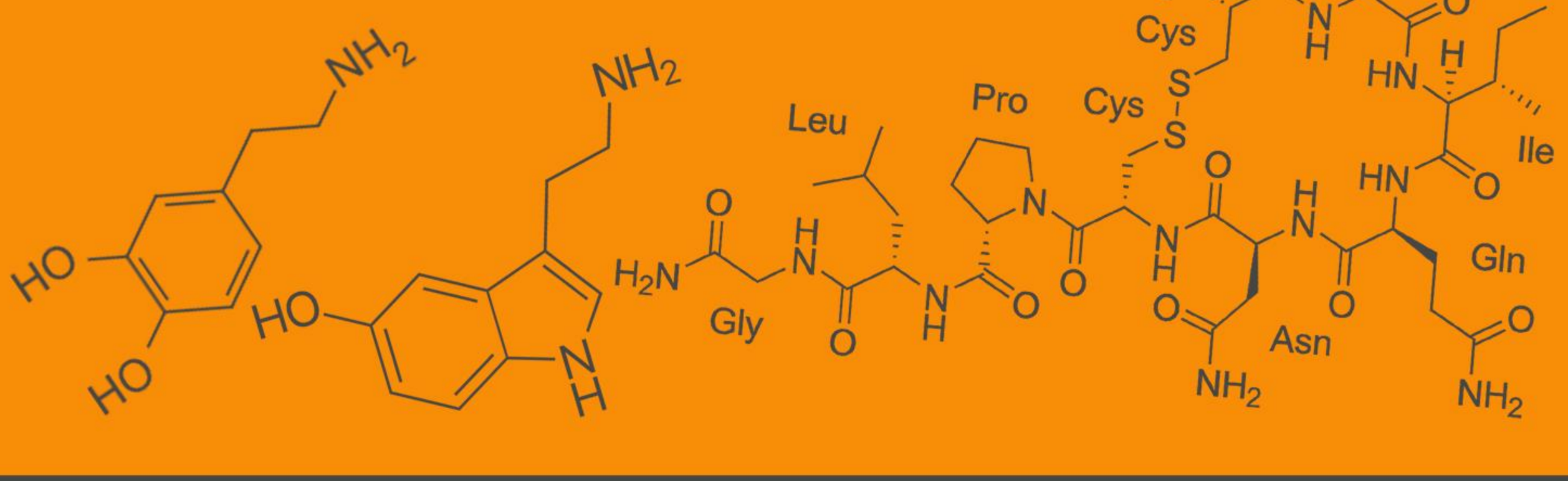
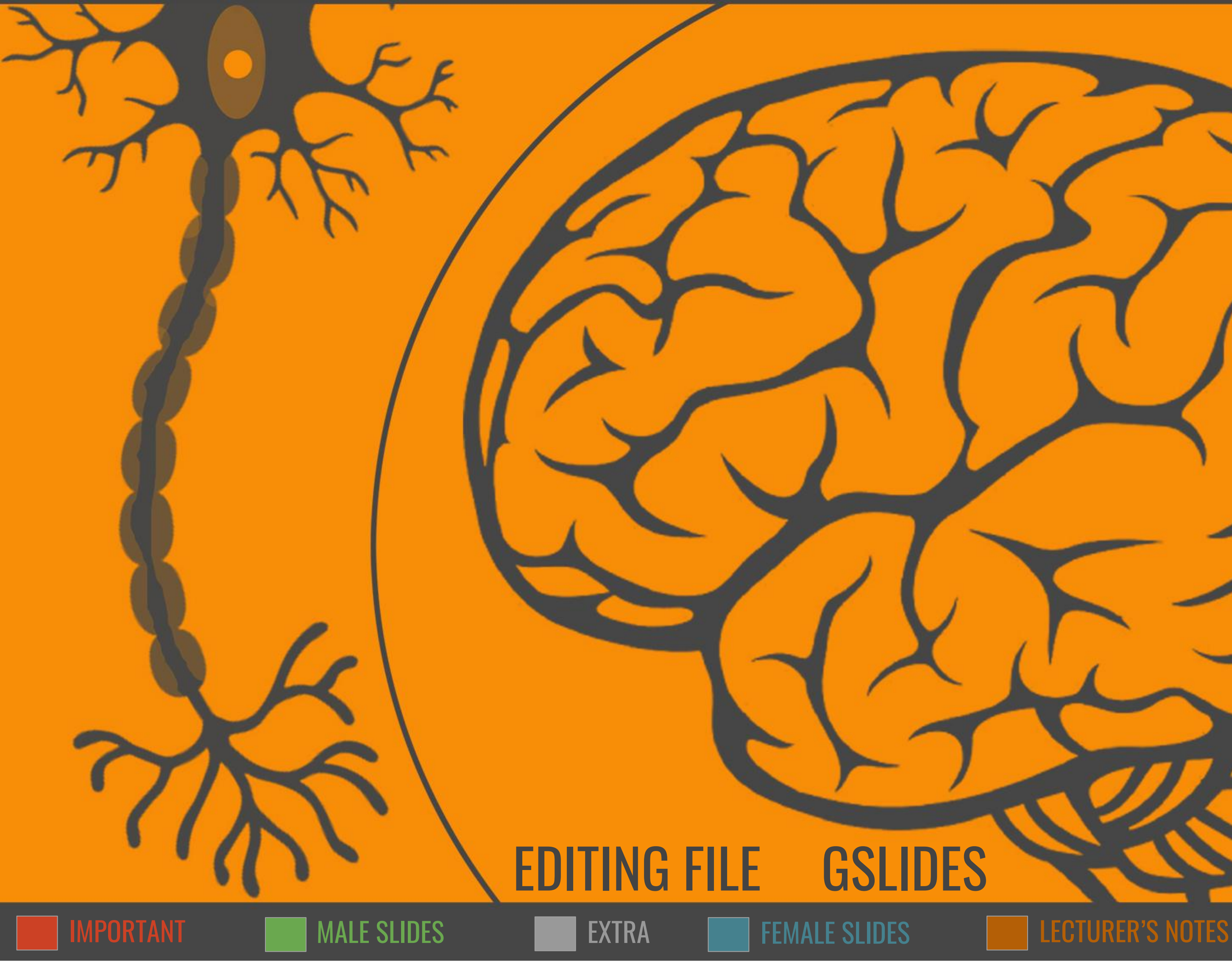


MEDICINE438's CNSPHYSIOLOGY

Lecture XXIV: Upper Motor Neuron and Lower Motor Neuron Lesions



EDITING FILE GSLIDES

IMPORTANT MALE SLIDES EXTRA FEMALE SLIDES LECTURER'S NOTES

1 UPPER MOTOR NEURON AND LOWER MOTOR NEURON LESIONS

Lecture 24

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.

Facts about Spinal Cord

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

Upper & Lower motor neuron

UMNs control LMNs through two different pathways:

- Pyramidal system.
- Extrapyramidal system.

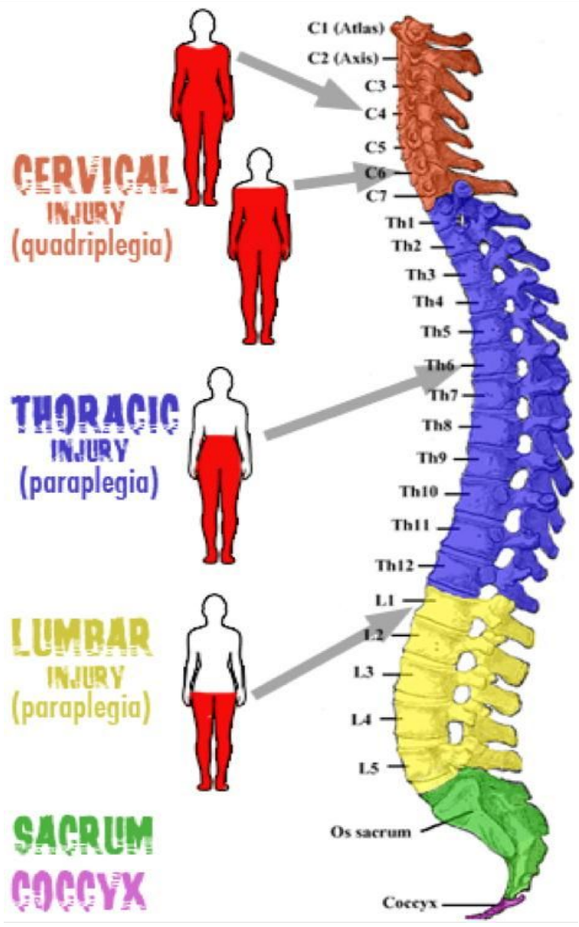


Figure 24-1

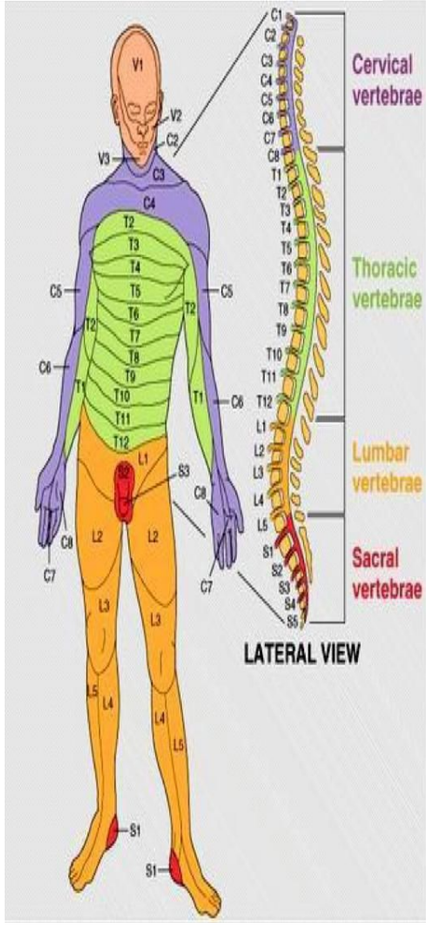


Figure 24-2

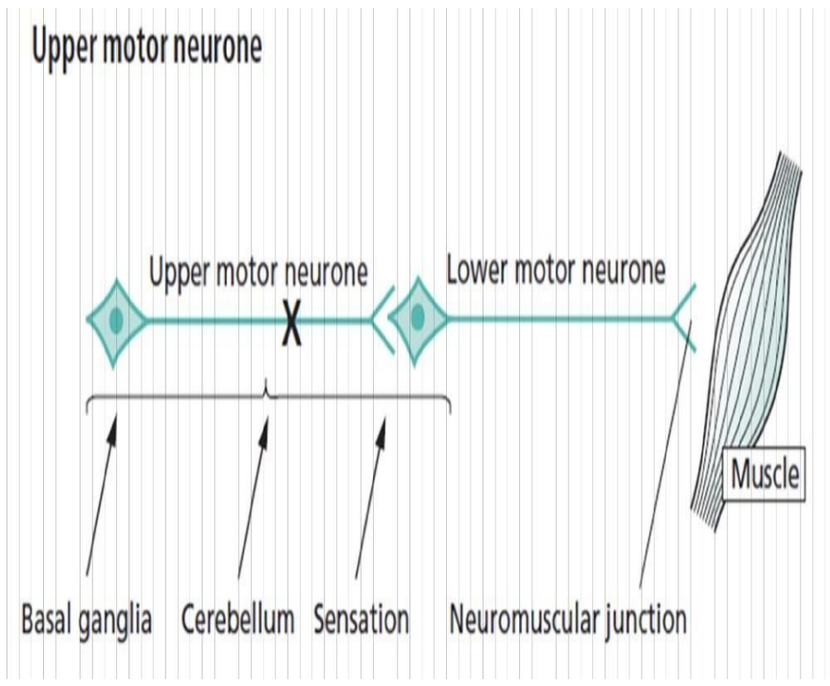


Figure 24-3

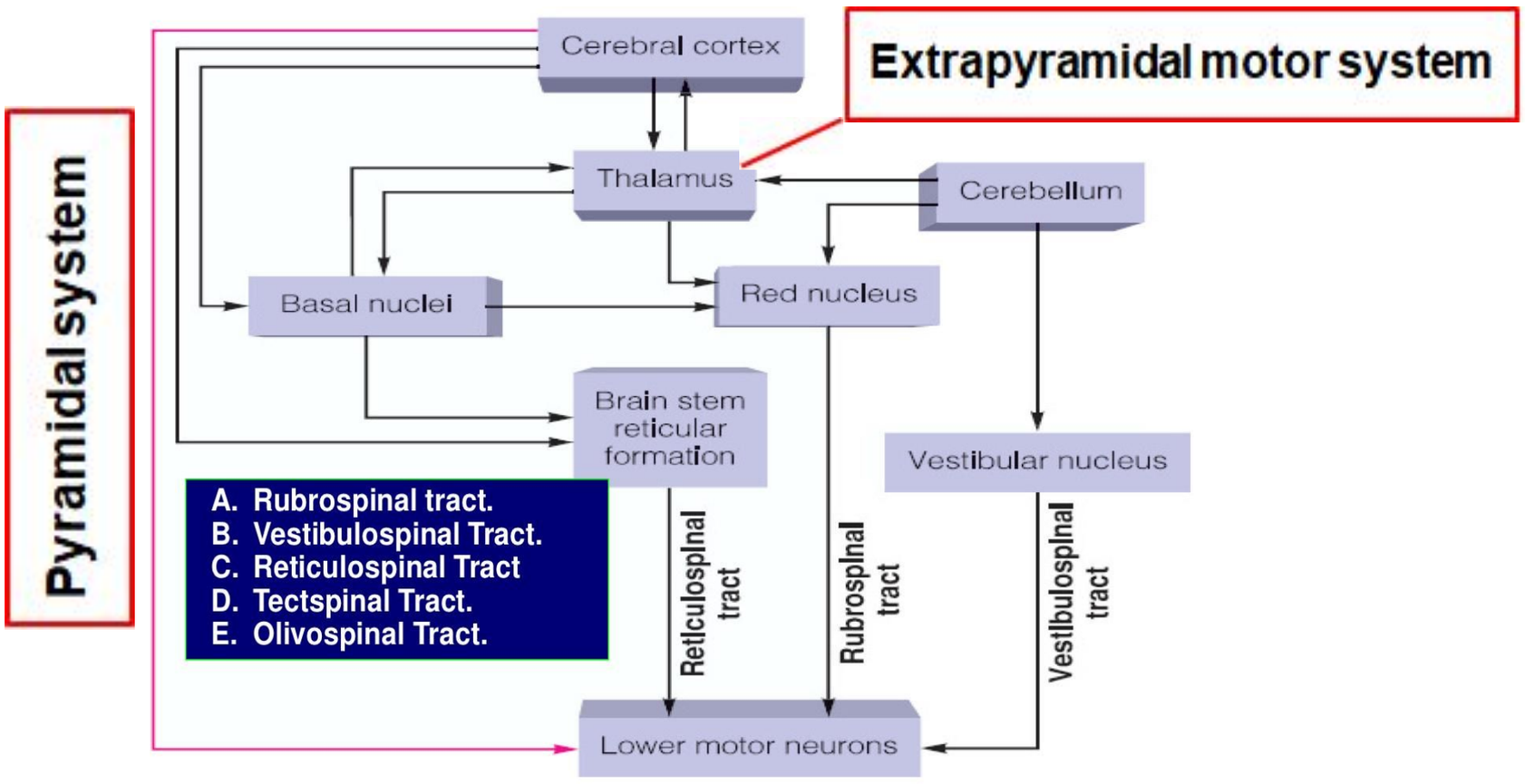


Figure 24-4

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 tract.

Internal Capsule

- Reciprocal connections between thalamus and cortex are found in four limbs of the internal capsule:
  1. Anterior limb with frontal lobe.
  2. Posterior limb with parietal lobe.
  3. Retrolenticular limb with occipital lobe.
  4. 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.

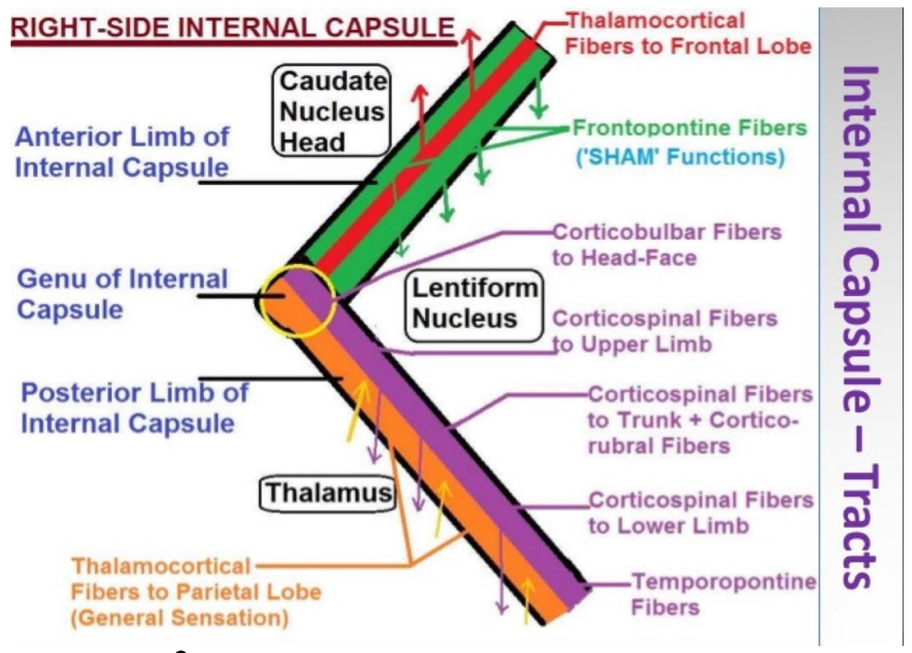


Figure 24-5

Patient came to the clinic complaining from paralysis of the upper and lower limbs. Which structure is affected? Posterior limb of internal capsule.

Lenticulostriate Arteries

- The superior parts of both the anterior and posterior limbs and the genu of the internal capsule are supplied by the lenticulostriate arteries (it also supply part of the basal ganglia) which are branches of the M1 segment of the middle cerebral artery.

Lenticulostriate artery is the most common artery to be blocked during a stroke.

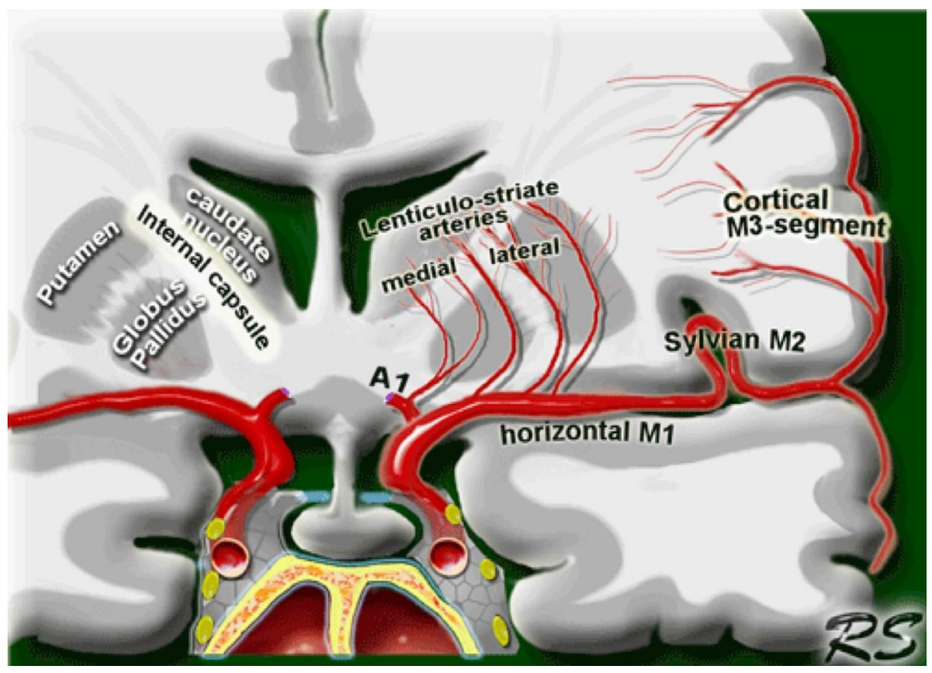


Figure 24-6

Causes Of UMN And LMN Lesions

Upper motor neuron lesion, Can result from:

1. Cerebral stroke by hemorrhage, thrombosis or embolism.
2. Spinal cord transection or hemisection (Brown-Séquard syndrome). often in the cervical cord region.

Lower motor neuron lesion, Can result from:

1. Anterior horn cell lesions (e.g. poliomyelitis,motor neuron disease).
2. Spinal root lesion or peripheral nerve lesion (e.g. nerve injury by trauma or compressive lesion such as disc prolapse).

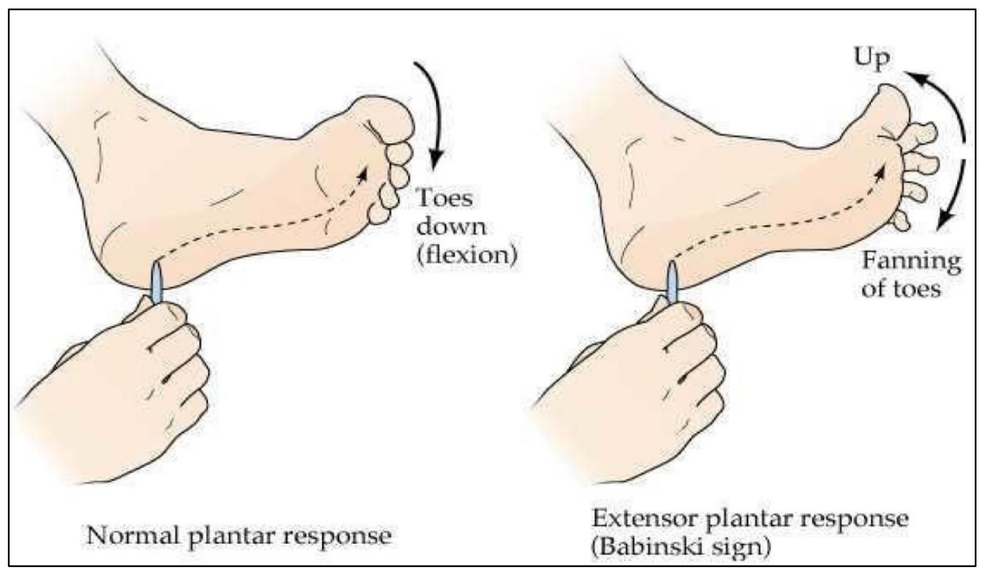


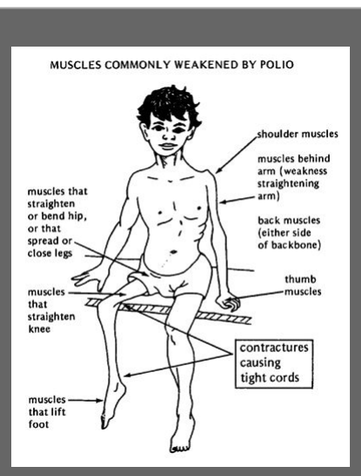
Figure 24-7

BOX 24-1: ROBBINS AND COTRAN PATHOLOGIC BASIS OF DISEASE

POLIOMYELITIS

Poliomyelitis is a disease that mostly has been eradicated by vaccinations, there are few developing countries however where polio still poses a problem.

- It is caused by poliovirus, it affects the GIT and in a small vulnerable fraction of affected people it affects the nervous system.
- People with poliomyelitis show inflammation in AHCs but the inflammation can also extend to the posterior horn, sometimes producing cavitation within the cord!
- As we know, viruses integrate their genome within affected cells, poliovirus genome has been detected in AHCs. This causes the cell to produce proteins from poliovirus's genome, causing damage to the cell. This causes destruction of AHCs, as well as muscle atrophy and flaccid paralysis of affected muscles.



COMPARISON UPPER AND LOWER MOTOR NEURON LESIONS

Features	UMNs Lesions	LMNs Lesions
Pattern	Paralysis affect movements (The whole muscles are affected)	Individual muscle or group of muscles are affected
Wasting	NOT pronounced (20-30% wasting) (There is muscle waste due to disuse[1] but less than LMN > Tropic)	Pronounced (70-80% wasting) (more wasted than UMN because the muscle supply is damaged > Atropic)
Tone	Spasticity muscles; hypertonic (Clasp Knife) (increase disinhibition)[2]	Tendon reflexes diminished or absent
Tendon Reflex	Brisk / increased (due to increases gamma discharge)	Diminished or absent
Superficial reflexes	Absent (because it's polysynaptic)[3]	Absent
NCV (nerve conduction velocity)	Normal	Decreased because the nerve root is affected
Denervation potential(fibrillation) [on EMG ONLY] (LMN)	Absent	Present
Fasciculations[4] (visible) (LMN)	Absent	Present
Trophic changes	Less	Pronounced in skin & nails
Clonus (rapid repetitive contraction) (UMN)	Present	Absent
Babinski's sign[5]	Extensor plantar response (positive)	Flexor or absent plantar response

Table 24-1

FOOTNOTES

1. 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 UMNs.
2. 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 UMNs.
3. UMN has a main role in polysynaptic reflexes so, damage in UMN will inhibit the superficial reflex.
4. Damaged alpha motor neurons can produce spontaneous action potentials. These spikes cause the muscle fibers to fire, resulting in a visible twitch of the affected muscle. While in UMNLs, fasciculations do not occur because alpha motor neurons themselves are spared.
5. Babinski sign occurs in UMNLs, it is a sign of an underdeveloped corticospinal tract, hence why it is seen in infants and people with spinal cord transection affecting UMNs, in grownups, this reflex disappears due to complete myelination.

BROWN-SÉQUARD SYNDROME "HEMISECTION OF SPINAL CORD"

Ipsilateral loss

- Fine touch, vibration, proprioception (dorsal column).
- Leg ataxia (dorsal spinocerebellar).
- Spastic paresis[1] below lesion (lateral corticospinal).
- Flaccid paralysis (ventral horn destruction).[1]
- Dermatomal anesthesia (dorsal horn destruction).

Contralateral loss

- Loss of pain and temp (lateral spinothalamic)
- Loss of crude touch and pressure (ventral spinothalamic).
- Minor contralateral muscle weakness (ventral corticospinal).
- Leg ataxia (ventral spinocerebellar).

Table 24-2

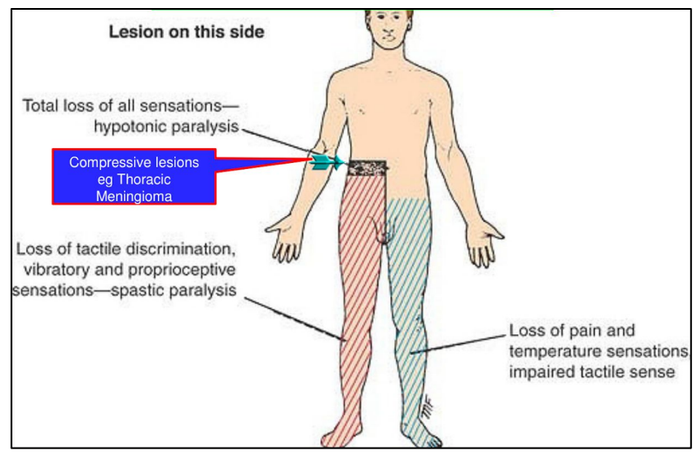


Figure 24-8

Explanation Of BROWN-SÉQUARD SYNDROME

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.

FOOTNOTES

1. Spastic paresis is characteristic of UMN lesions, it means (1) weakness, (2) loss of efficiency (3) trouble in controlling muscles.
2. Flaccid paralysis means paralysis with loss of muscle tone, this type of paralysis is characteristic of a LMN lesion.

LESIONS OF SPINAL CORD

- Upper cervical cord lesions produced quadriplegia and weakness of the diaphragm (because it's supplied by Phrenic nerve which originates from C3-C5)

- Lesions at C4-C5 produce quadriplegia (Diaphragm is intact).

- Hemiparesis means weakness.
- Hemiplegia means total paralysis.

- Limerick "C3, 4 and 5 keep the diaphragm alive".

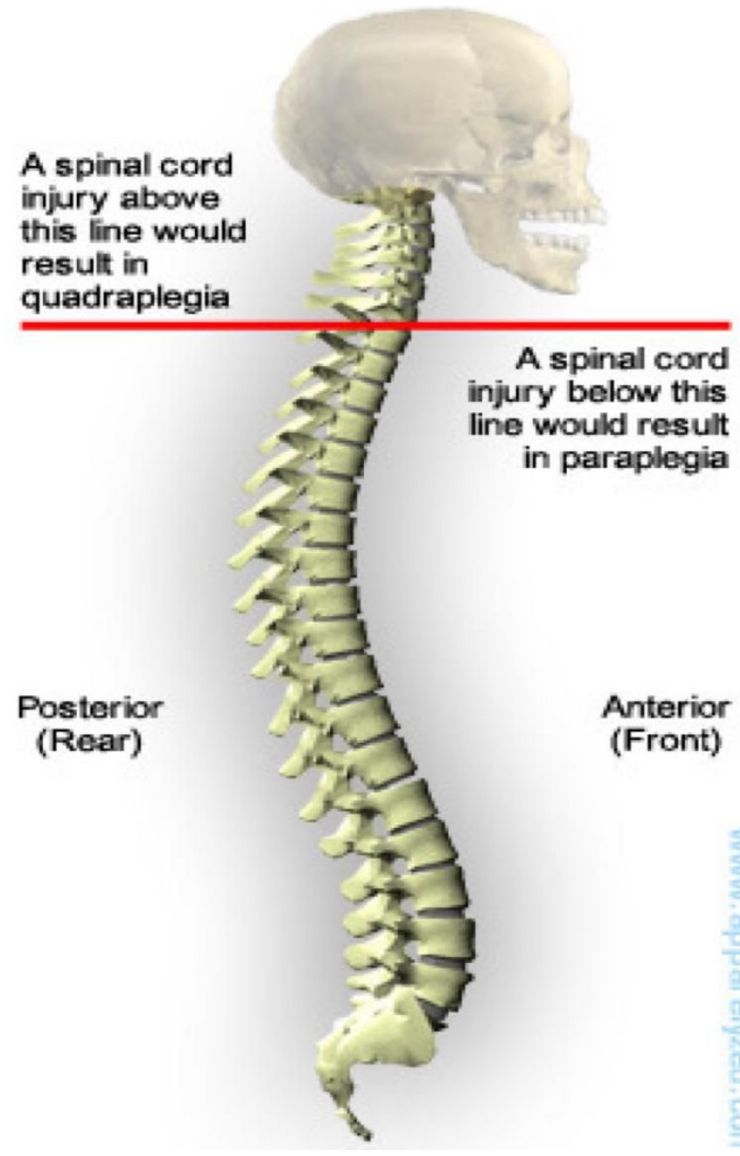
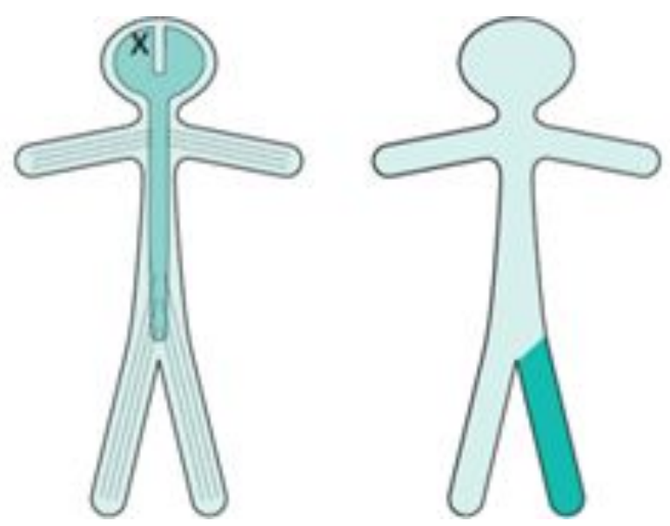
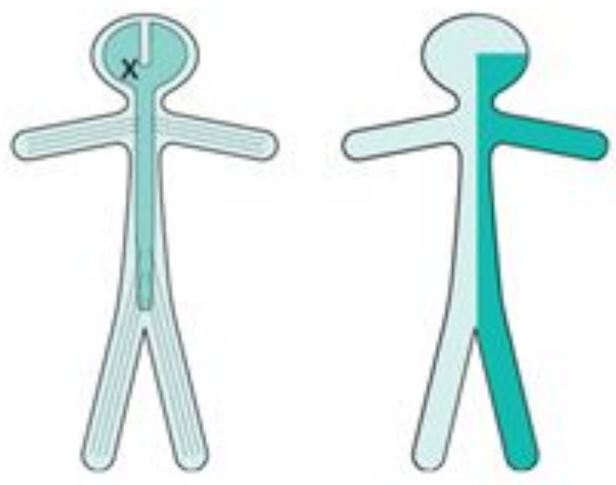
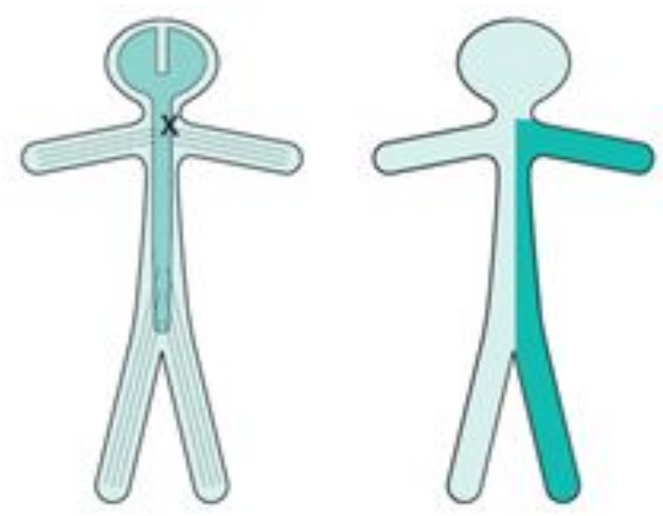
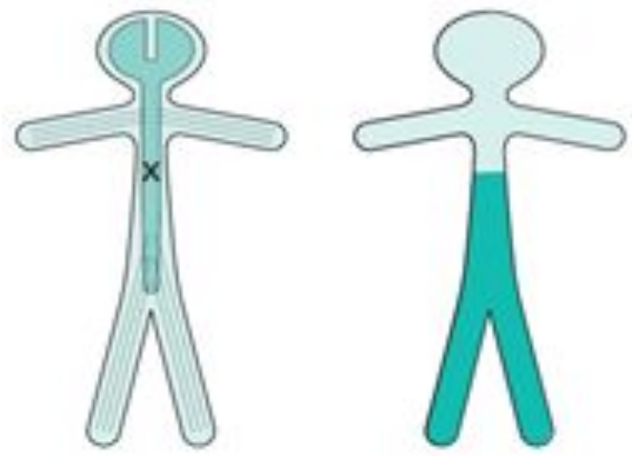
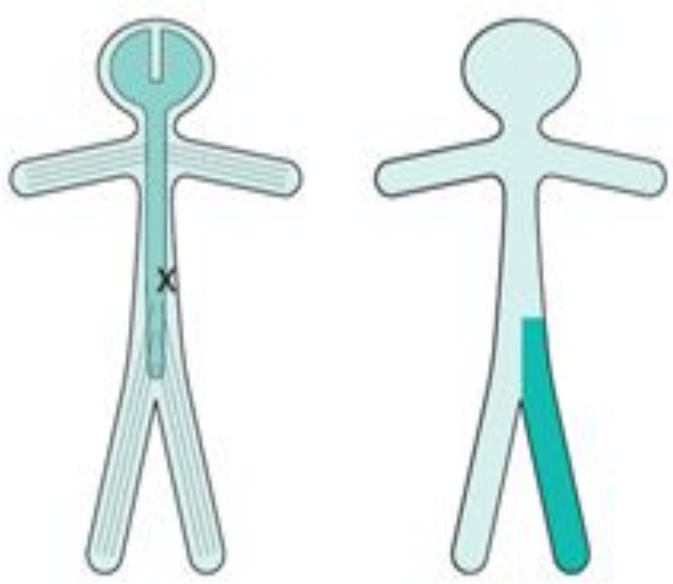


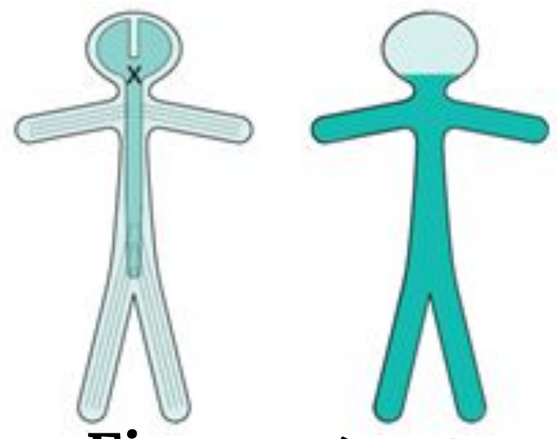
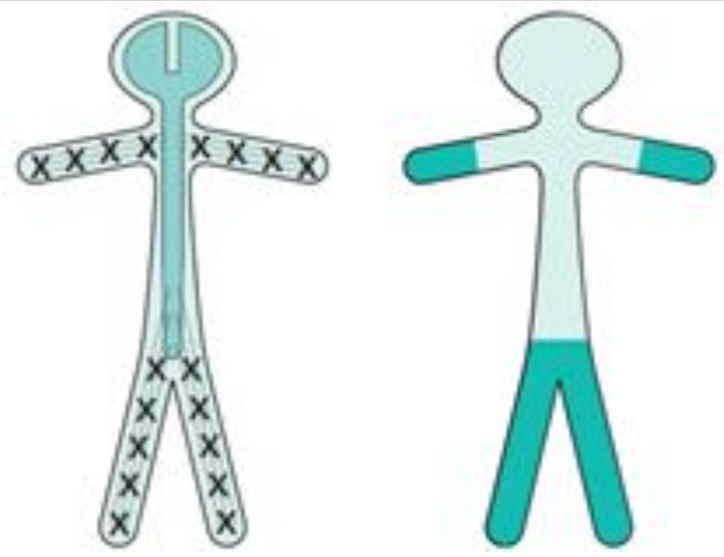
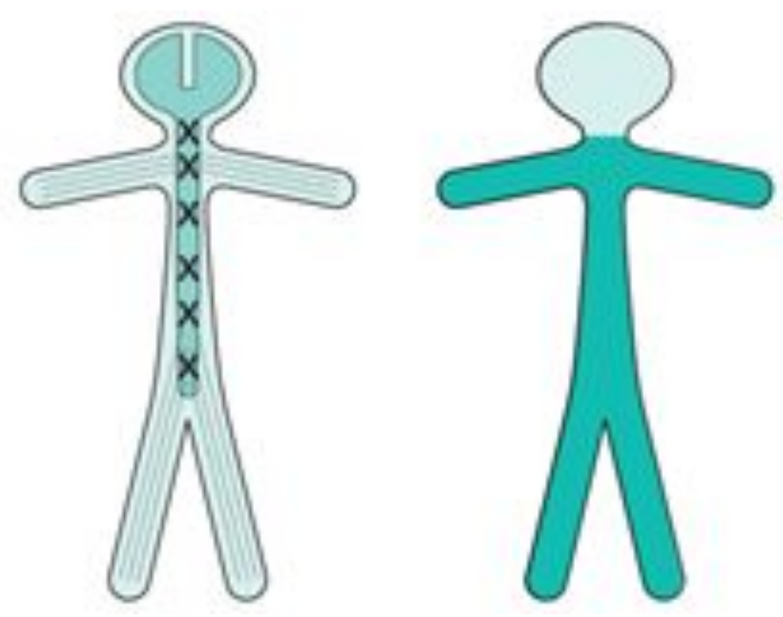
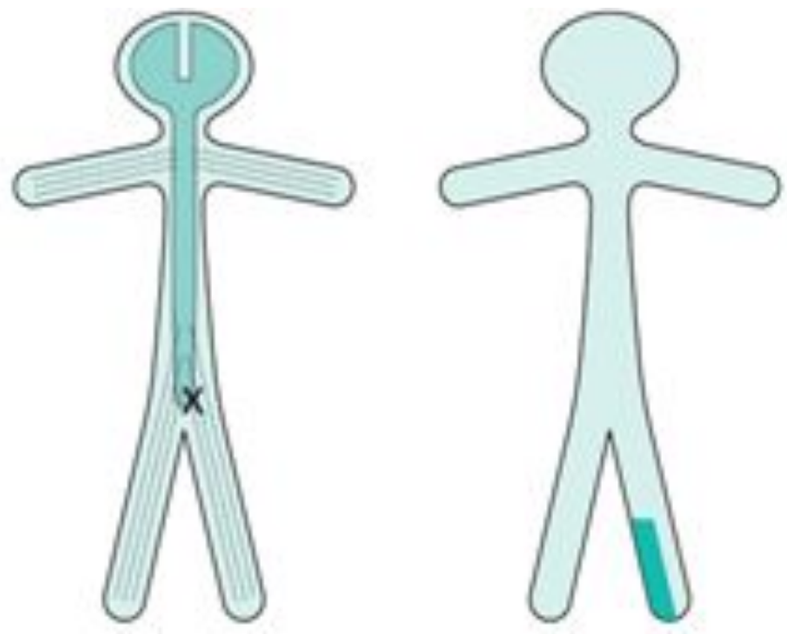
Figure 24-9

COMPARISON BETWEEN LESIONS

Contralateral monoparesis	- A lesion situated <u>peripherally</u> 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.	 **Figure 24-10**
Contralateral hemiparesis	Lesions situated <u>deep</u> 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 (hemianesthesia) and visual loss (homonymous hemianopia), in addition to the hemiparesis.	 **Figure 24-11**

Ipsilateral hemiparesis	A unilateral high cervical cord lesion will produce a hemiparesis similar to that which is caused by a contralateral cerebral hemisphere lesion (deep in in the internal capsule), 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.	 **Figure 24-12**
Paraparesis	Paraparesis, if the lesion is at or below the cervical portion of the spinal cord.	 **Figure 24-13**
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.	 **Figure 24-14**

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.	 Figure 24-15
Generalized LMN weakness	May 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.	 Figure 24-16
	May also 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.	 Figure 24-17
LMN weakness of one spinal root	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. 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. Also, Carpal tunnel syndrome which involved median nerve compression.	 Figure 24-18

8 UPPER MOTOR NEURON AND LOWER MOTOR NEURON LESIONS

Lecture 24

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

Anterior spinal artery syndrome

Upper sensory level for pain/temperature, sparing of posterior columns, UMN signs distally

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

Brown-Sequard syndrome

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

Table 24-3

Bulbar palsy (Nuclear)	Pseudobulbar palsy (Supranuclear)
Bilateral lesion of LMN: • LMN defect of peripheral palsy. • Defect in IX-XII cranial nerves or their nuclei in medulla Oblongata.	**Bilateral lesion of corticobulbar tract (UMN):** • UMN defect of central palsy. • Defect in IX-XII cranial nerves.
• Dysphagia (liquid>solid), nasal regurgitation, slurred speech. • Nasal speech, wasted tongue with fasciculation, absent gag reflex. • Cause: polyradiculoneuritis (guillain-barré syndrome): brainstem lesions, tumors meningoencephalitis, Motor Neuron Disease.	• Dysphagia (solid> liquid), dysarthria, emotional lability (unprovoked crying or laughing) (because it's affecting the limbic system) • Slow indistinct speech, spastic tongue, brisk jaw jerk (masseter reflex). • Frontal release signs. • Cause: CVA, Arteriosclerosis.

Table 24-4

BOX 24-2: ROBBINS AND COTRAN PATHOLOGIC BASIS OF DISEASE

GUILLAIN-BARRÉ SYNDROME It's an acute demyelinating disease affecting motor axons, resulting in weakness that can lead to death due to paralysis of respiratory muscles.

- Physiology Team: The disease affects peripheral nerve, it is triggered by an infection that leads to loss of self-tolerance, this is most likely due to molecular mimicry, an autoimmune mechanism in which an infectious agent which carries antigen similar to antigens present in the body triggers an immune response, then the body attacks both the pathogen and similar proteins within the body. Remember that the antigens taken from pathogens are digested into smaller peptides that are presented to T-cells, if those peptides resembled those present from self-proteins, an autoimmune attack will occur.

MENINGOENCEPHALITIS It is an infectious disease that can be caused by bacteria, fungi, viruses. It causes meningitis, vasculitis, and invasion of brain's parenchyma. Vasculitis can cause brain infarction. The name comes due its resemblance to meningitis and encephalitis.

VIIth Cranial Nerve lesions

UMN VIIth CN lesion

Causes **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

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

Table 24-5

INTRAMEDULLARY AND EXTRAMEDULLARY SYNDROMES

Intramedullary lesion	Extramedullary lesions
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)**	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)**

Table 24-6

BLADDER CONTROL

Cortical	Spinal cord	LMN
Post-central lesions cause loss of sense of bladder fullness	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	Sacral lesions (conus medullaris, sacral root and pelvic nerve – bilateral) cause a **flaccid, atonic bladder** that overflows (cauda equina), often unexpectedly
Pre-central lesions cause difficulty initiating micturition	Frontal lesions can also cause a **hypertonic bladder**	
Frontal lesions cause socially inappropriate micturition (e.g: urinate in front of people)		

Table 24-7

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

Table 24-8

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

Table 24-9

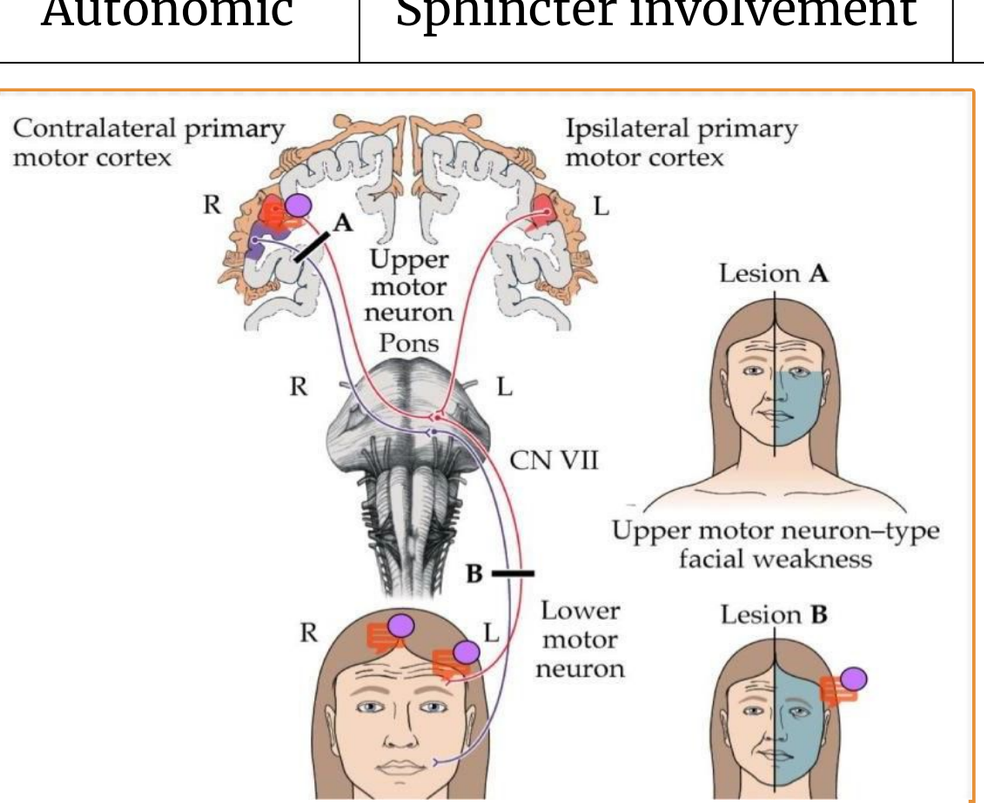


Figure 24-19

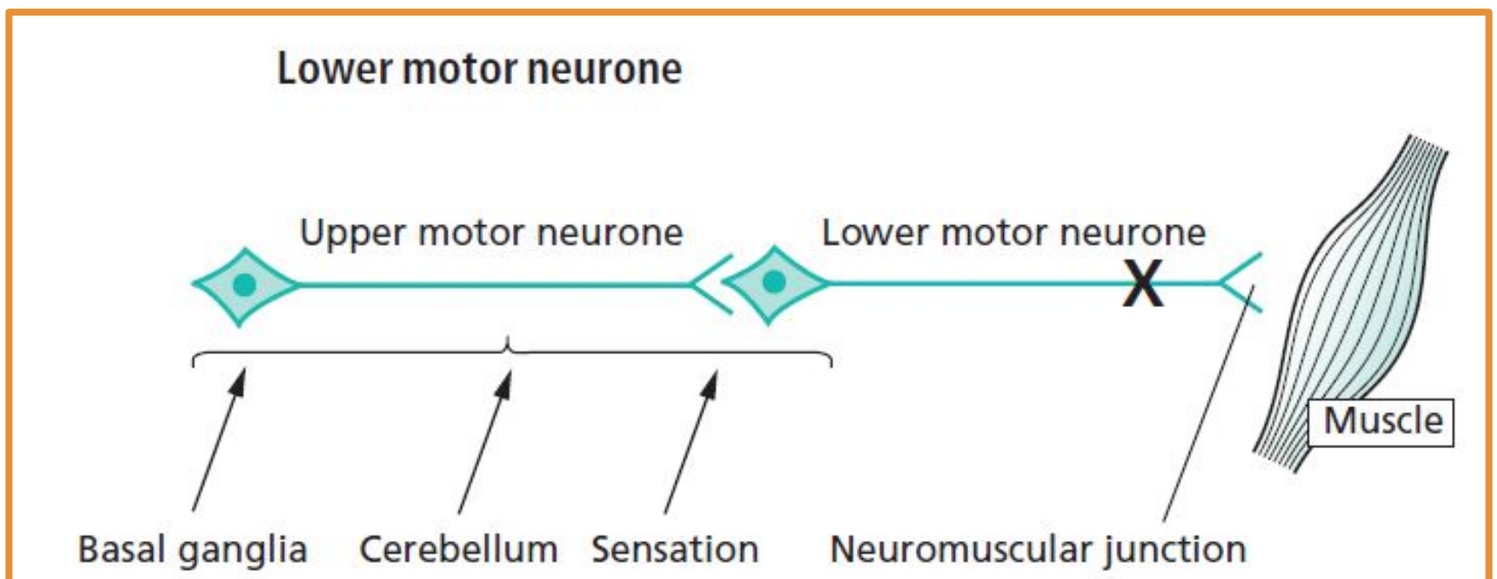


Figure 24-20

SUMMARY

Causes of LMN and UMN lesions

LMN lesion		UMN lesion	
Anterior Horn cell lesion Eg; poliomyelitis	Spinal root or peripheral nerve lesions	Cerebral stroke	Spinal cord transection and hemisection

Comparison between UMN and LMN lesions

	UMN	LMN
Tone	Spastic	Tendon reflex diminished or absent
NCV	Normal	decreased
Denervation Potential	absent	present
fasciculation	absent	present
clonus	present	absent
Babinski sign	positive	Flexor or negative

SUMMARY

LESIONS OF SPINAL CORD

Lesion	Site	Result in
Contralateral monoparesis	Peripherally in cerebral hemispheres	Contralateral weakness of a specific area
Contralateral hemiparesis	Internal capsule	Weakness of the whole contralateral side of the body + hemianesthesia (sensory loss) + visual loss
Ipsilateral hemiparesis	High cervical spinal cord	Same as contralateral hemiparesis but without involvement of the face thus vision will be fine
Paraparesis	Cervical position	-
Ipsilateral monoparesis	Below the level of the neck	Upper motor neuron weakness in one leg, may also have posterior column loss (sensory) in the same leg and spinothalamic loss (pain) on the contralateral leg
Tetraparesis or quadriparesis	Upper cervical cord or brain stem	-
Generalized LMN weakness	1-widespread damage to the axons of the LMN (marked distally at limbs) 2-pathology in LMN either spinal cord or brain stem (poliomyelitis)	1-causes alongside the weakness: sensory loss
LMN weakness of one spinal roots	Spinal root or nerve	Weakness only in the supplied area by that nerve or root (muscle or sensory loss)

QUIZ

MEDICINE438's
CNSPHYSIOLOGY

MCQs

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.

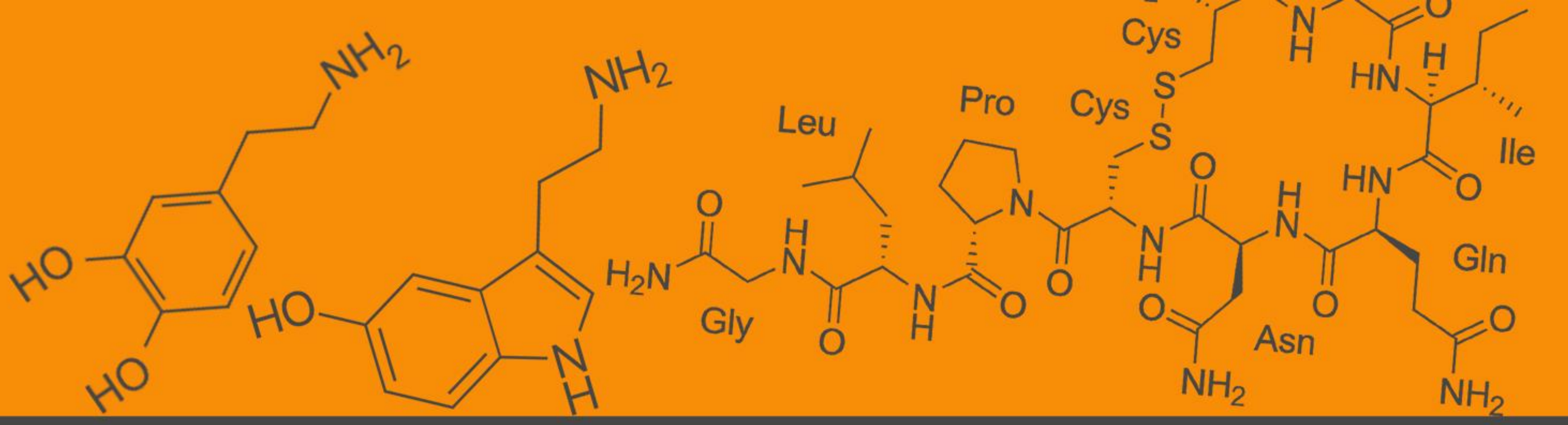
8. Contralateral hemianesthesia is:
A. Loss of sensation on some parts of the opposite side of the body
B. loss of sensation of both legs
C. loss of sensation on the upper part of the opposite of the body
D. loss of all sensation on the opposite side of the body.

SAQ

- A patient is having poliomyelitis, what type of lesion are you expecting him to have?

SAQ: Lower motor neuron lesion

ANSWER KEY: B, B, C, B, A, C, C, D



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