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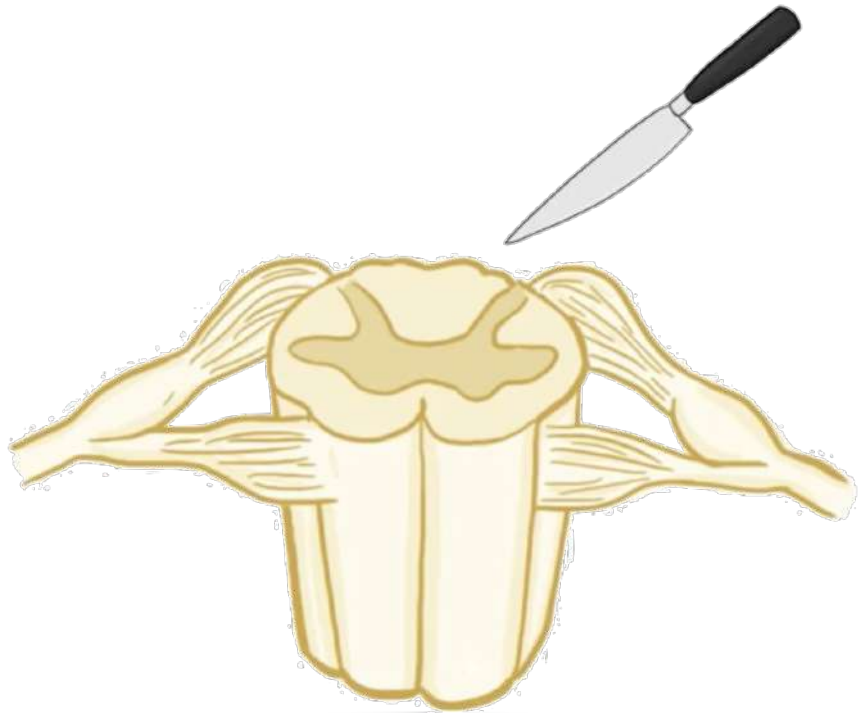


Motor neuron Lesions

PHYSIOLOGY
CNS BLOCK
TEAM 443

MED 443
King Saud University

تمت المراجعة
٤٤٣
Lama AlMutairi
Mohammed AlEssa
Abdulaziz AlFuhayd

Color Index:

- Main text
- Important
- Girls Slides
- Boys Slides
- Notes
- Extra

Editing File

Objectives..

1. Describe the functional anatomy of upper and lower motor neurons.
2. Describe and differentiate the causes & features of upper and lower motor neuron lesions.
3. Explain features of Brown Sequard Syndrome.
4. Correlate the site of lesion with pattern of loss of sensations.
5. Describe facial, bulbar and pseudobulbar palsy.

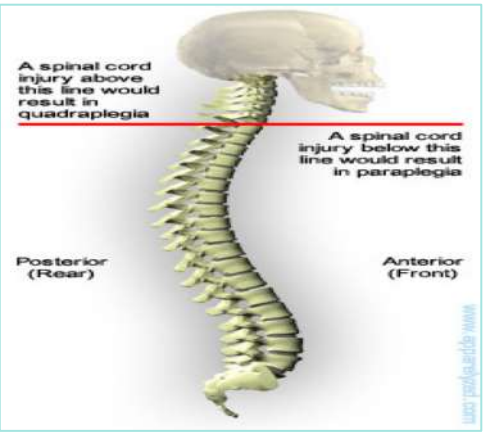
We advise you to revise the anatomy lecture (sensory tract) to make this lecture much easier.

- Najeeb
- JJ Medicine
- Upper & Lower Motor neuron lesions (Ghada Alsadhan)
- Osmosis (Brown sequard syndrome) really helpful

Introduction

1. Spinal cord injury above the cervical region would result in quadriplegia (paralysis of all 4 limbs).
2. Spinal cord injury below the cervical region (Thoracic and lumbar) would result in paraplegia,
   a. (paralysis of lower limb).
   b. (Thoracic and lumbar) would result in paraplegia, (paralysis of lower limb)
3. There are two cases of quadriplegia, either in:
   a. High cervical cord lesion → Diaphragm will be affected.
   b. Low cervical cord lesion **and (C3-5) is not involved** → Diaphragm will be intact.
4. Diaphragm is supplied by C3,4, 5, so if the lesion is below that level → diaphragm will be intact.
5. Upper motor neuron lesions prevent signals from traveling from your brain and spinal cord to your muscles.

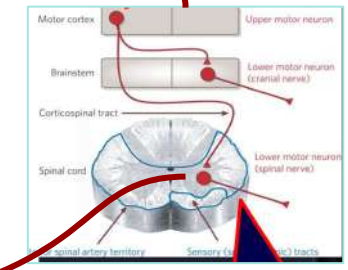
Your muscles can't move without these signals, therefore become stiff and weak.

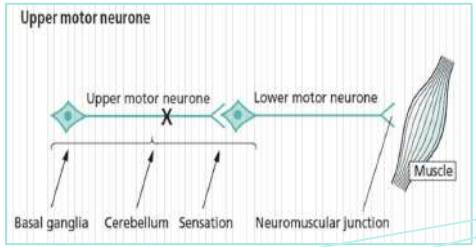


Extrapyramidal motor system

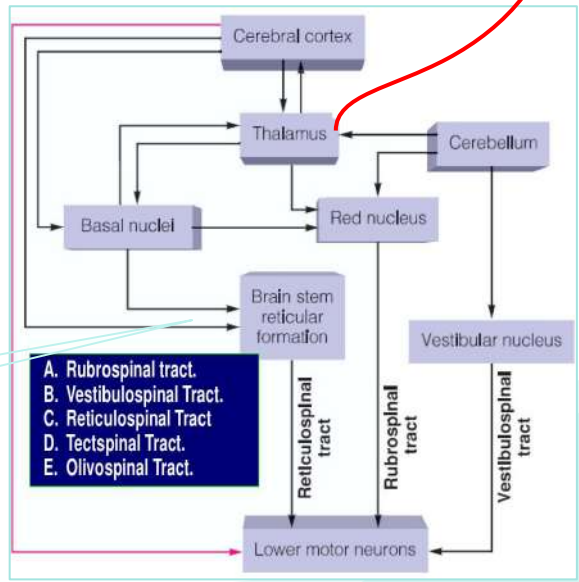
★ **Anything from the brain to anterior horn is UMN**

★ **Anything from anterior horn to the muscle is LMN**





Pyramidal system

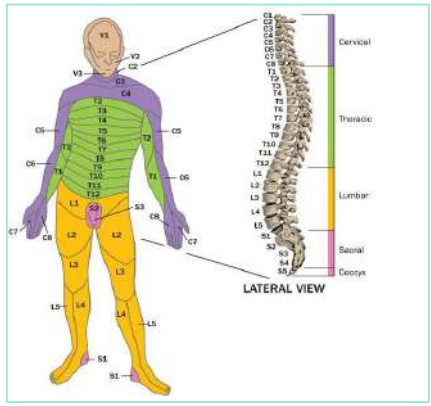


The term extrapyramidal motor system is widely used in clinical circles to donate all those portions of the brain and brain stem that contribute to motor control but are not part of the direct corticospinal-pyramidal system

Introduction

1 Spinal cord:

31 segments (31 spinal nerve pairs), embryological development growth of cord lags behind -> mature spinal cord ends at L1 (lower part of L1 or upper part of L2)

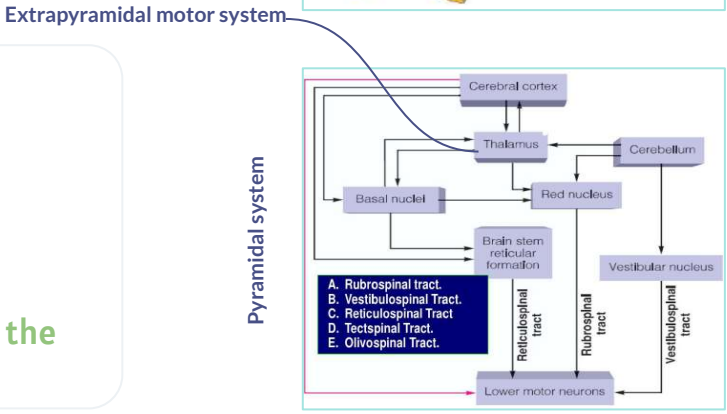


2 Upper & Lower motor neurons

UMNs control LMNs through two different pathways:

a. **Pyramidal system** "They **travel** from the cerebral cortex through the pyramidal system, and **terminate directly** in LMN of the spinal cord"

b. **Extrapyramidal system** "They **do not go directly** to LMN, rather they make a synapse either in the brainstem, basal nuclei, or cerebellum"

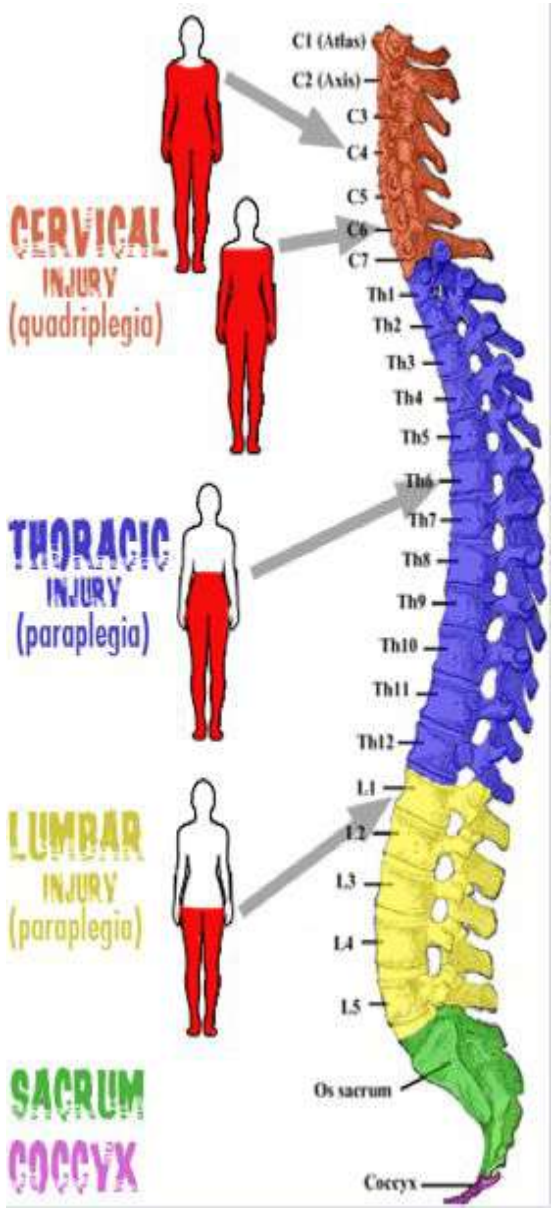


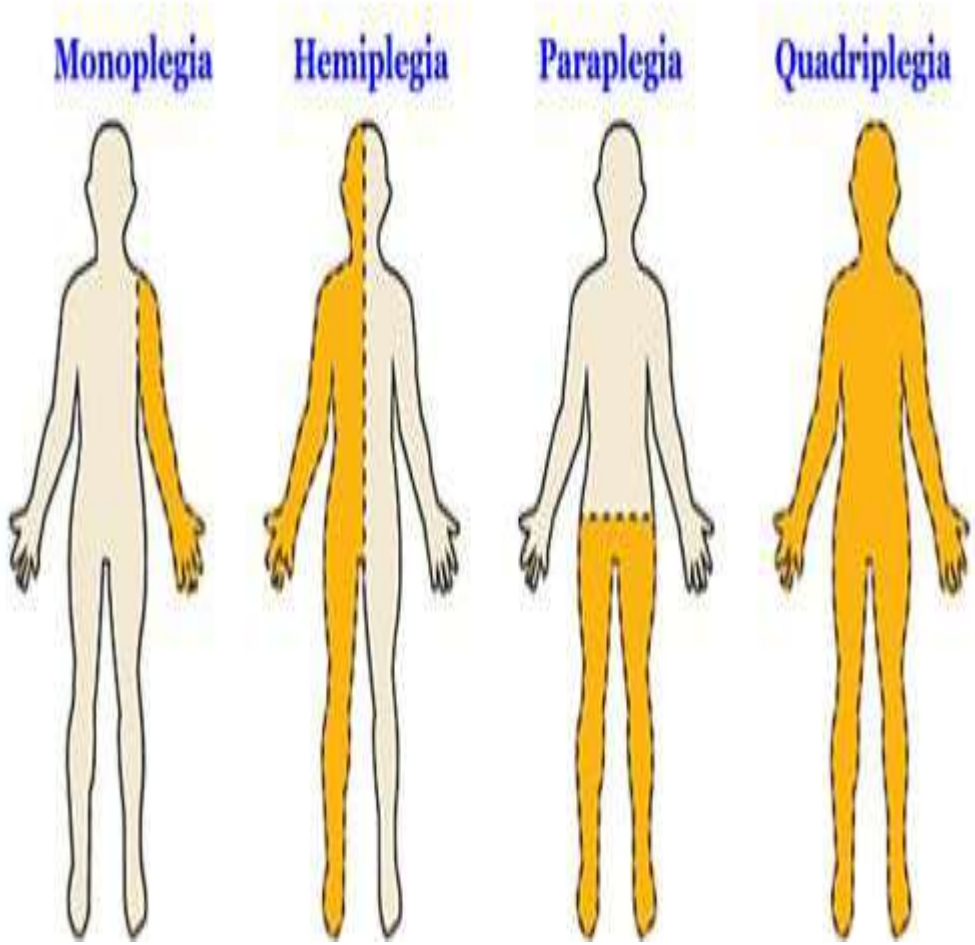
Males Slides →

Pure corticospinal tract lesion cause hypotonia(flaccidity) instead of spasticity.
The reason is that pure pyramidal tract lesion is very very rare(clinically isn't seen), and spasticity is due to loss of inhibitory control of extrapyramidal tract.

★ Causes of UMN and LMN lesions:

UMN		LMN
Cerebral stroke by **hemorrhage, thrombosis or embolism** (the most common)	**Result from**	**Anterior horn cell lesions** e.g: poliomyelitis, motor neuron disease
Spinal cord transection or hemisection (brown séquard syndrome)		**Spinal root lesions or peripheral nerve lesion** e.g: nerve injury by trauma or compressive lesion.


C1 (Atlas)
C2 (Axis)
C3
C4
C5
C6
C7
Th1
Th2
Th3
Th4
Th5
Th6
Th7
Th8
Th9
Th10
Th11
Th12
L1
L2
L3
L4
L5
Os sacrum
Coccyx
CERVICAL
INJURY
(quadriplegia)
THORACIC
INJURY
(paraplegia)
LUMBAR
INJURY
(paraplegia)
SACRUM
COCCYX


Monoplegia
Hemiplegia
Paraplegia
Quadriplegia

Introduction.

1

Right-Side Internal Capsule :

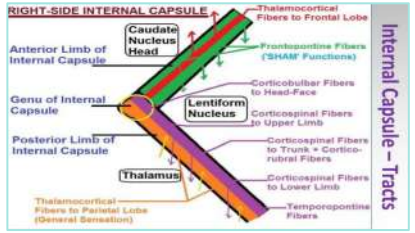
★ Reciprocal (reverse) connections between **thalamus** and **cortex** are found in **four limbs** of the internal capsule:

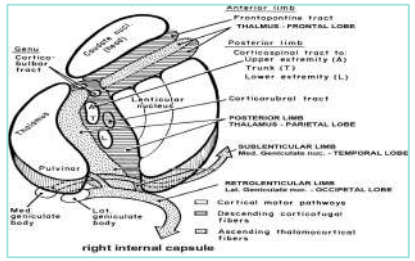
Anterior limb with frontal lobe

Retrolenticular limb with occipital lobe

Posterior limb with parietal lobe

Sublenticular limb with temporal lobe





★ **Genu** contains the **corticobulbar axons.**

★ **Posterior limb** contains **corticospinal axons and corticopontine axons**

★ **Anterior limbs** contains corticopontine axons

Note that corticopontine fibers are present in both Anterior and posterior limbs.

★ If the lesion is in the **corona radiata** due to artery blockage for example, the affected area will be depended on the fibers located in that area (partial paralysis)

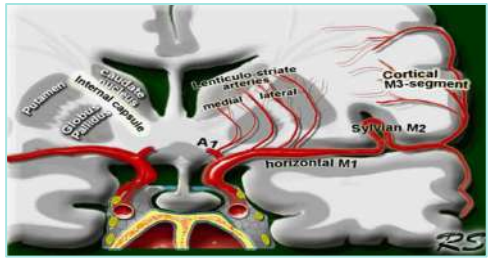
★ If the lesion is in the **internal capsule** there will be contralateral total paralysis because all tracts are passing through the internal capsule

Females Slides

2

Lenticulostriate Arteries:

The superior parts of both the **anterior** and **posterior** and the **genu of the internal capsule** are supplied by the **lenticulostriate arteries**, which are branches of the **MI segment** of middle cerebral artery.



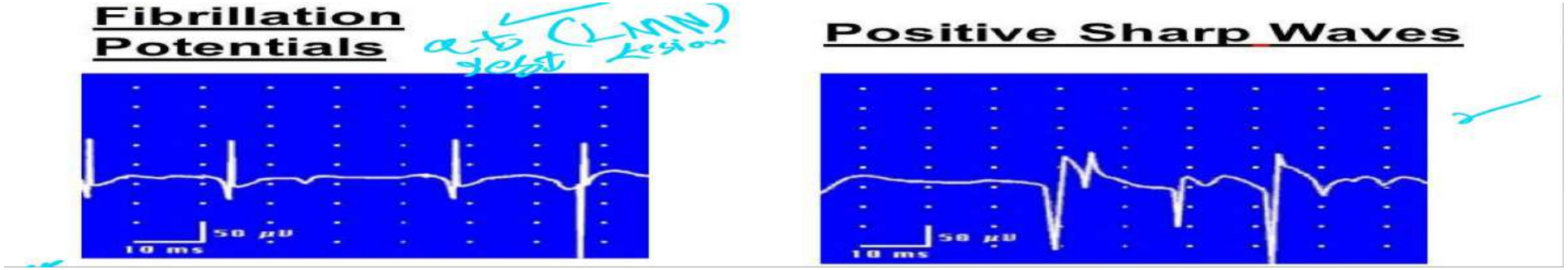
Important

Comparison between UMN and LMN lesions

	Upper Motor Neuron Lesion	Lower Motor Neuron Lesion
Pattern	Paralysis affect movements rather than muscles (Whole lower limb, or both so it affects movement)	Individual muscle or group of muscles are affected (Doesn't involve the whole limb, so limited effect on the movement)
Wasting " if you don't use it, you lose it" (438: muscle waste due to disuse but less than LMN)	Not pronounced (About 20-30% wasting)(20-30%) Muscle is intact and respond to reflexes because reflexes are mediated by **SC (LMN)**, but cannot be controlled that's why wasting is less.	Pronounced (About 70-80% wasting) More wasted than UMN because the muscle supply is damaged. 
Tone	Spasticity muscles hypertonic (clasp knife) (spastic muscle)	(Hypotonic muscle)Tendon reflexes diminished or absent, Hypotonic paralysis(flaccidity muscle)
Tendon reflexes (Deep)	brisk/increased (due to loss of inhibition signals coming from UMN, **the reflex is exaggerated**)	Diminished or absent (due to damaged fibers that initiates reflexes, e.g. gamma motor neurons) 
Superficial reflexes "Polysynaptic"	Absent/ Decreased/ Changed (UMNs have a main role in polysynaptic reflexes, so if it gets damaged, this reflex will be inhibited)	Absent(Notice that all reflexes will be absent in case of LMN lesion)
NCV "Nerve conduction velocity"	Normal (Nerves roots are not involved)	Decreased (Nerves roots are affected)
Denervation potentials (Fibrillations) on EMG only, can't be seen with eyes **and Fasciculations (Visible)** can be seen with eyes	Absent Group of muscle fibrils make one muscle fasciculus	Present
Trophic changes	Less (Because nerves supply are not involved)	Pronounced in skin & Nails Trophic changes are **simply changes in soft tissue (skin, fascia, muscle)**, resulting from interruption of nerve supply.
Clonus (involuntary movements alternate between contraction and relaxation)	Present (Clonus is involuntary and rhythmic muscle contractions caused by a permanent lesion in upper motor neurons.)	Absent
Babinski's sign A normal response is flexion of the large toe and adduction of the other toes – this a negative Babinski sign. However, in patients who have an UMN syndrome **an abnormal plantar reflex is elicited whereby the large toe extends and there is abduction of the other toes**	Extensor plantar response (positive)	Flexor or absent plantar responses

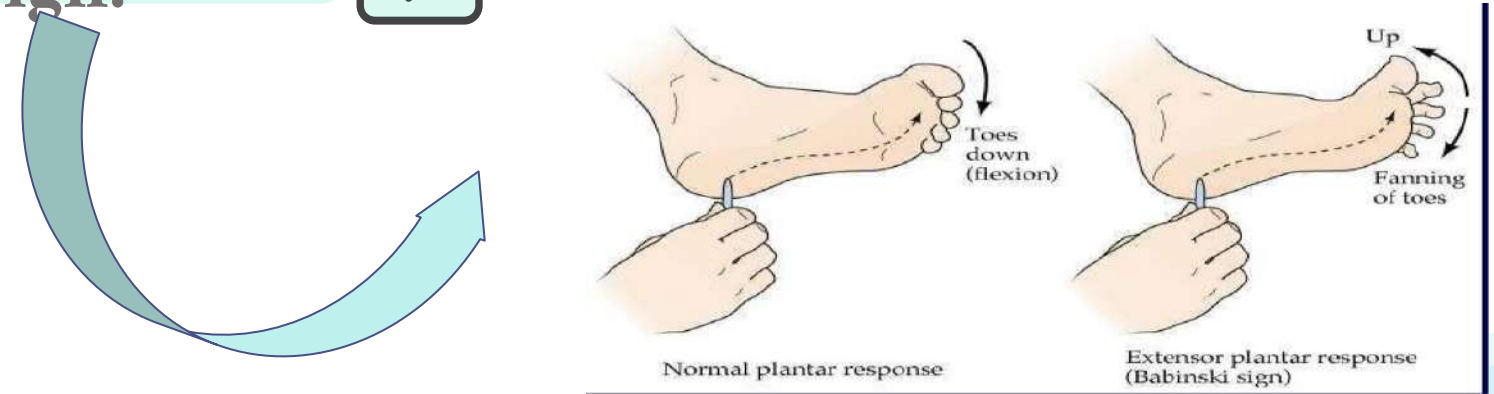
Comparison between UMN and LMN lesions

Fibrillation & fasciculations in LMNL:



The hallmarks of denervation on needle EMG include the presence of fibrillation potential, positive sharp waves , and increased insertional activity.

Babinski's Sign:



BROWN-SÉQUARD SYNDROME

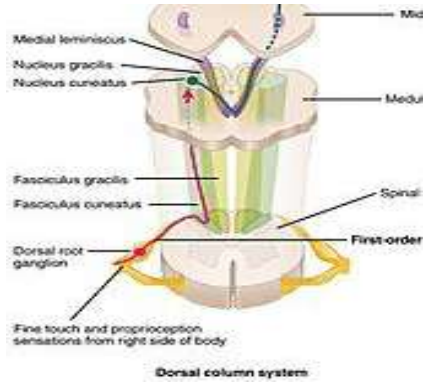
★ **What is BROWN-SÉQUARD SYNDROME ?**

It is is a condition that results from damage to one half of the spinal cord, either right or left side.

★ **What can BROWN-SÉQUARD SYNDROME cause?**

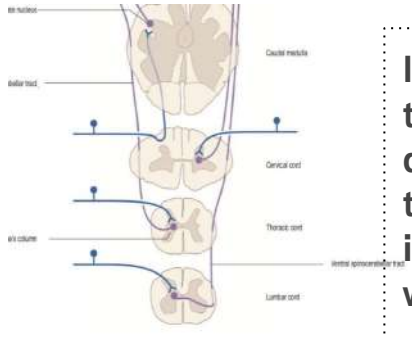
1. The hemisection causes damage of the spinal cord tracts which are pathways for transfer of information to and from the brain.
2. Results in weakness or paralysis (hemiparaplegia) on one side of the body.
3. Results in loss of thermal and pain sensation (hemianesthesia) on the opposite side of the body.(because the lateral spinothalamic tract is damaged).
4. Results in loss of vibration and proprioception on the same side (because the dorsal column tracts are damaged).

Dorsal column pathway diagram



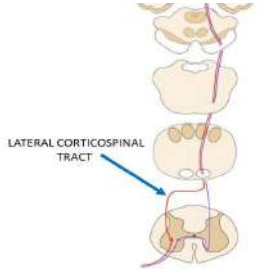
If the lesion occurs in dorsal column, the effect will be ipsilateral because it occurs **at the level below the decussation**

Spinocerebellar tracts pathway diagram



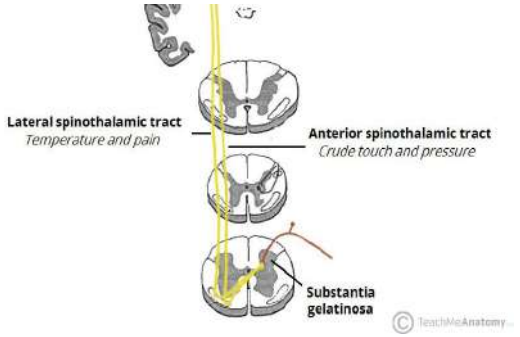
If the lesion occurs in the **dorsal spinocerebellar tract,** the effect will be ipsilateral because this tract doesn't decussate. Whereas in the **ventral spinocerebellar tract,** the effect will be contralateral because it decussate immediately at the level of spinal cord where it synapse with Ist order neuron

Lateral corticospinal tract pathway diagram



If the lesion occurs in LCST, the effect **will be ipsilateral because it initially goes ipsilateral, and the decussation occurs at the level above the spinal cord (MO).**

Spinothalamic tracts pathway diagram



If the lesion occurs in spinothalamic, the **effect will be contralateral because decussate immediately at the level of spinal cord** where it synapse with Ist order neuron.

BROWN-SÉQUARD SYNDROME

HEMISECTION OF SPINAL CORD

Ipsilateral loss

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

Contralateral loss

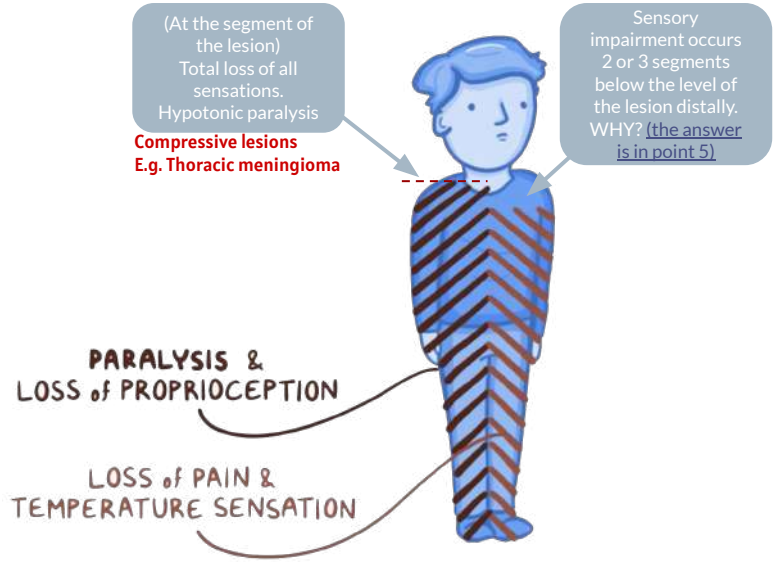
- Loss of pain and temperature (lateral spinothalamic)
- Loss of crude touch and pressure (ventral spinothalamic)
- Minor contralateral muscle weakness (ventral corticospinal)
- Leg ataxia (ventral spinocerebellar)

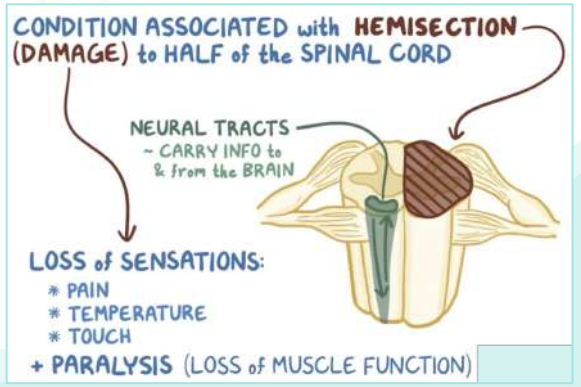
Make sure to read the previous slide

Upper cervical cord lesions produce quadriplegia and weakness of the diaphragm and lower lesions spare diaphragm with good prognosis (if C3,4,and 5 not involved)

Lesions at C4-C5 produce quadriplegia

Hemiparesis means **weakness**
Hemiplegia means **total paralysis**
limerick C3,C4,and C5 keep the diaphragm alive.





BROWN-SÉQUARD SYNDROME (1)

1. **Damage to the neurons on the anterior gray column** and possibly by **damage to the nerve roots of the same segment** cause Ipsilateral lower motor neuron paralysis in the segment of the lesion and muscular atrophy.
   Anterior grey column damage (motor) → ipsilateral LMN paralysis → if you don't use it, you loss it (muscular atrophy)

2. **Loss of the corticospinal tracts on the side of the lesion** causes 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.

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. **Destruction of the ascending tracts in the posterior white column on the same side of the lesion** cause Ipsilateral loss of tactile discrimination and of vibratory and proprioceptive sensations below the level of the lesion.

BROWN-SÉQUARD SYNDROME (2)

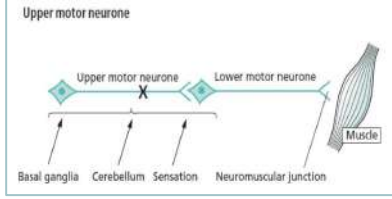
5

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

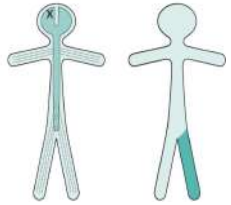
Contralateral but not complete loss of tactile sensation below the level of the lesion. This condition is brought about by **destruction of the crossed anterior spinothalamic tracts** on the side of the lesion. Sensory impairment occurs two or three segments below the level of the lesion distally.

Patterns of UMN disorders (1)



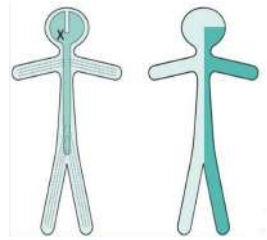
CONTRALATERAL MONOPARESIS
Lesion situated peripherally in the cerebral hemisphere
(Boys slides only)

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
In the Internal Capsule

Lesions situated deep 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.**

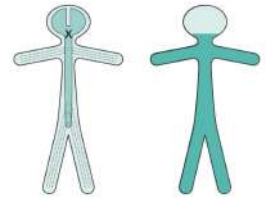


IPSILATERAL HEMIPARESIS

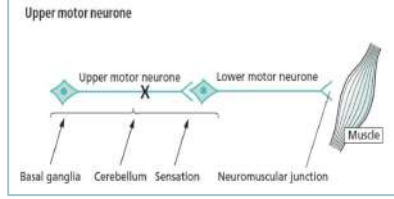
A unilateral high cervical cord lesion will produce a hemiparesis similar to that which is caused by a contralateral cerebral hemisphere lesion, except that the face cannot be involved in the hemiparesis, vision will be normal, and the same dissociation of sensory loss (referred to above) may be found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

TETRAPARESIS

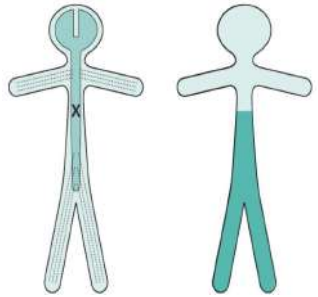
Tetraparesis or quadriparesis, if the lesion is in the upper cervical cord or brainstem.
A spinal cord lesion more usually causes upper motor neuron signs in both legs, often asymmetrically since the pathology rarely affects both sides of the spinal cord equally.



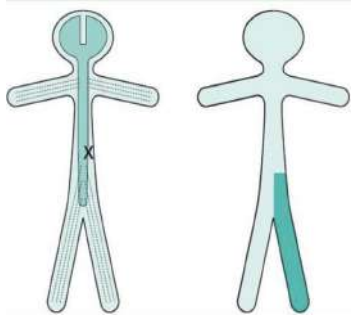
Patterns of UMN disorders (2)



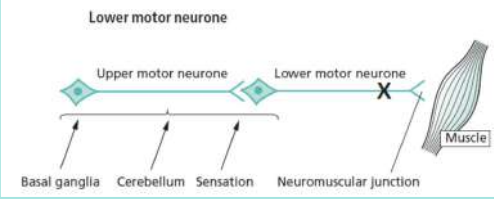
PARAPARESIS (Boys slides only)	**Paraparesis, if the lesion is at or below the cervical portion of the spinal cord.** A spinal cord lesion more usually causes upper motor neuron signs in both legs, often asymmetrically since the pathology rarely affects both sides of the spinal cord equally.



IPSILATERAL MONOPARESIS Brown-Séquard syndrome	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-Séquared syndrome .**

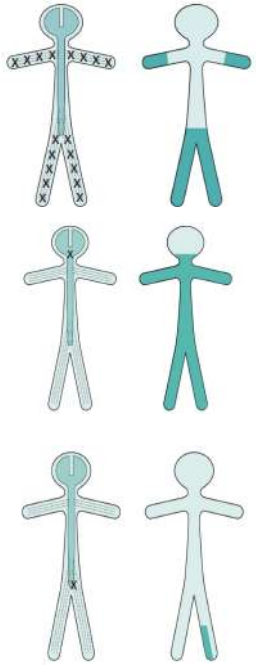


Patterns of LMN lesions



Male slides

GENERALIZED LMN WEAKNESS	May result from **widespread damage to the axons of LMNs**. This is the nature of **peripheral neuropathy (polyneuropathy)** in DM. 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. (The body will be affected)
LMN WEAKNESS OF ONE SPINAL ROOT (Most common)	**ONE SPINAL ROOT OR ONE 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 **SI nerve root syndrome caused by a prolapsed intervertebral disc, or a common peroneal nerve palsy (neck of the fibula), Median (CTS), Femoral (Lnguinal Ligament), Post tibial (Tarsal Tunnel S) and Ulnar (wrist;Guyon's canal).** CTS = carpal tunnel syndrome.



Female slides

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.

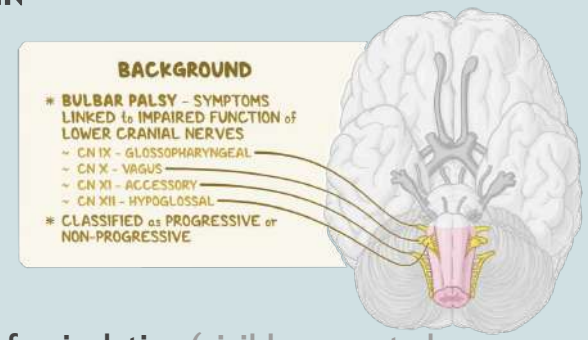
It was mentioned in Dr.Shahid's older slides, but he removed it because it's not that important and they don't ask about it.

Bulbar vs Pseudobulbar palsy

Bulbar = Medulla
Medulla houses 9 - 12 CNs.
Dysfunction of lower CNs

Bulbar Palsy (Nuclear)

- **Means:** Defect of IX, X, XI, and XII cranial nerves or **their nuclei** in Medulla Oblongata, Bilateral affection of **LMN**
- **LMN** lesion **Peripheral** palsy.



- **Result in:**
  1. Dysphagia (liquid>solid)
  2. Nasal regurgitation
  3. Slurred speech.
  4. Nasal speech
  5. **Wasted tongue** (atrophied) with fasciculation (visible unwanted muscle twitches)
  6. Absent gag reflex
  7. Atrophy to all bulbar muscles
  8. No emotional effect

- **Caused by:** polyradiculoneuritis (GBS): brainstem lesions, tumors, meningoencephalitis, MND

Pseudobulbar Palsy (supranuclear)

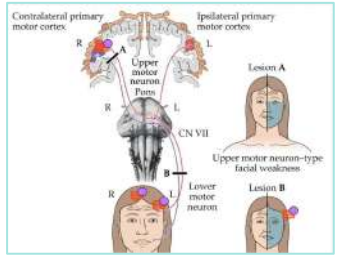
- **Bilateral lesion of corticobulbar tract**
- **Means: UMN** defect of central palsy IX, X, XI, and XII cranial nerves.
- **Result in:**
  1. Dysphagia (especially in liquid)
  2. No Nasal regurgitation
  3. Spastic **Dysarthria** (due to cortical region involvement)
  4. **Emotional lability** (unprovoked uncontrollable crying or laughing)
  5. **spastic tongue** (UMN)
  6. Hyperreflexive gag (UMN)
  7. Slow indistinct speech
  8. Exaggerated **Brisk jaw jerk** (masseter reflex).
  9. Frontal release signs.

Anything spastic, we have to think of hyperreflexia

- **Caused by:** CVA, arteriosclerosis

VII Cranial Nerve lesion.

Bell's palsy vs UMNL



UMN VII Cranial Nerve Lesion

Lower motor neuron (LMN) lesions.
Cause weakness of the **lower part of the face** on the **opposite side**.
Frontalis & orbicularis is spared: **normal furrowing of the brow** is preserved; **eye closure and blinking are largely unaffected**.
(because the upper nuclei are innervated by two fibers , so the lower part of the face will be spared)

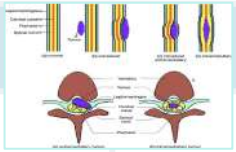
LMN VII Cranial Nerve Lesion

LMN VIIth Cranial Nerve lesion.
Causes weakness **(ipsilateral)** of **all facial expression muscles**.
Results:
I. The angle of the mouth falls; unilateral dribbling develops.
2. **Frowning (frontalis) and eye closure are weak.**
3. **Corneal exposure and ulceration** occur if the eye does not close during sleep. called **Bell's palsy**.

It was mentioned in Dr.Shahid's older slides, but he removed it because it's not that important and they don't ask about it.

Intramedullary & extramedullary lesions:

Females Slides



Extramedullary lesion	Intramedullary lesion
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**

Spinal cord and bladder control

It was mentioned in Dr.Shahid's older slides, but he removed it because it's not that important and they don't ask about it.

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

It was mentioned in Dr.Shahid's older slides, but he removed it because it's not that important and they don't ask about it.

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.

Summary.

Pattern of UMN lesions

Lesion name	Part involved	Result in	Other
Contralateral monoparesis	I. Motor homunculus 2. +/- Sensory homunculus (postcentral gyrus)	★ Hemiparesis of a part of the contralateral body side. ★ Sensory loss in the same part (that already affected, so contralaterally as well) if Sensory homunculus involved	Lesion situated peripherally in the cerebral hemisphere
Contralateral hemiparesis	Internal capsule It is funnelling of fibre pathways in the region of the internal capsule	★ Hemiparesis of the whole of the contralateral body side, including face, arm, leg. ★ **Vision loss** (homonymous hemianopia) ★ Significant contralateral sensory loss (hemianesthesia)	Lesion situated deep (IC), so more likely to produce weakness of the whole contralateral body side
Ipsilateral hemiparesis	Unilateral high cervical	★ Hemiparesis of the whole ipsilateral side, FACE, is not involved. ★ **Normal vision** ★ Same dissociation of sensory loss, below the lesion level	A hemiparesis similar to that caused by contralateral cerebral hemisphere lesion, but face is intact and vision is normal.
	Unilateral lesion in the spinal cord below the level of the neck	★ UMN hemiparesis in one leg ★ **Dorsal column** ipsilateral sensory loss (position sense) in the same leg. ★ **Spinothalamic** contralateral sensory loss (pain & temperature) in the opposite leg. Dissociated sensory loss is a pattern of **sensory loss** with selective loss of touch sensation and proprioception without loss of pain and temperature, or **vice-versa.**	Brown-séquard syndrome. Sensory loss here is known as dissociated sensory loss.
Tetraparesis (quadriparesis)	Upper cervical cord or brain stem	★ UMN lesion signs, in both legs (bilaterally) asymmetrically	Pathology rarely affect both side of spinal cord equally
Paraparesis	At or below the cervical portion of SC		

Summary

Patterns of LMN lesions			
Lesion name	**Parts involved**	**Result in**	**Other**
Generalized LMN	Wide spread axons of LMNs	★ Hemiparesis in the distal limb ★ sensory loss is the distal limb	This is the nature of peripheral neuropathy (polyneuropathy
	LMNs throughout the SC and brainstem	★ Generalized limb weakness (distal and proximal) ★ **Trunk weakness** ★ **Bulbar weakness**	As in motor neuron disease or poliomyelitis
LMN weakness one spinal root	One spinal root or one individual peripheral nerve.	★ LMN signs are only found in the muscles supplied by the particular nerve root or peripheral. ★ Sensory impairment in the area supplied by the nerve or nerve root.	E.g. Lesions in SI nerve root syndrome caused by prolapsed intervertebral discs (other examples are mentioned in the previous slide)

Correlation between site of the lesion and clinical feature

Clinical Feature	Site of Lesion
Ipsilateral LMN flaccid paralysis in the segment	Ant Horn cell
Ipsilateral spastic paralysis below the level	UMN Lesion
Ipsilateral band of cutaneous anesthesia (only one root)	Posterior root damage
Ipsilateral loss of tactile vibratory and proprioceptive sensations below the level	Dorsal Column
Contralateral loss of pain and temperature sensations below the level of the lesion.	Lat Spinothalamic
Contralateral but not complete loss of tactile sensation	Ant Spinothalamic
Ipsilateral Dystaxia	Dorsal Spinocerebellar
Contralateral Dystaxia	Ventral Spinocerebellar
Bilateral Pain & Temp Loss Upper limbs (Bilateral because all fibers are damaged)	Ant Commissure
All sensory lost	Dorsal Horn
All motor lost	Ant Horn

How to localize LMN lesion :

Presenting features	Anterior horn cell disease	Peripheral nerve	Neuromuscular junction disease	Muscle disease
Distribution	Asymmetric limb/bulbar	Symmetric & distal	Extra ocular, bulbar and proximal limb muscles	Symmetric proximal or distal limb muscles
Muscle atrophy	Marked and early	Moderate	None	-Early stage - slight -Late -marked
Sensory involvement	None	Parasthesia	None	None
Characteristics Features	Fasciculations and cramps	Combined sensory and motor	Diurnal fluctuation	Usually proximal involvement
Reflexes	Variable	Decreased	Normal	Decreased

TEST YOURSELF !

1- which one of the following is the result of unilateral cervical spinal cord lesion:

A) Contralateral monoplegia

B)Ipsilateral hemiparesis.

C)Unilateral hemiplegia.

D)paraplegia

*answers:
1-B
2-B
3-C
4-A

2-which of the following is a feature of UMNL?

A) Marked muscle wasting.

B) Spastic paralysis.

C) Hypotonia.

D) Absent knee jerk.

3-Damage to left internal capsule:

A) Left side paralysis

B) Left side hemianstheisa

C) Babinski's sign on the right side.

D)Deafness

4-In lower motor neuron lesion:

A) Absence of deep tendon reflex.

B) Wide spread of contralateral paralysis.

C)Exaggerated deep reflex.

D)Disuse atrophy.

SAQ

1-compare between UMNL & LMNL:

Turn into slide number 7.

2-Explain briefly about Brown-sequard syndrome:

Turn into slide number 10.

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Special Thanks to Physiology **Team441**
Team logo and design was done by **Rafan Alhazzani**
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