Upper & Lower Motor neuron Lesion

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Week 6 Lecture

Chapter 56

(Guyton & Hall)

Cortical and Brain Stem Control of Motor Function

Objectives

By the end of this session you are expected to be able to:

- Appreciate what is meant by upper and lower motor neurons
- Explain manifestations of lesions of the upper and lower motor neurons
- Describe effects of lesions in pyramidal tracts and in the internal capsule
- Explain the manifestations of complete spinal cord transection and hemisection.

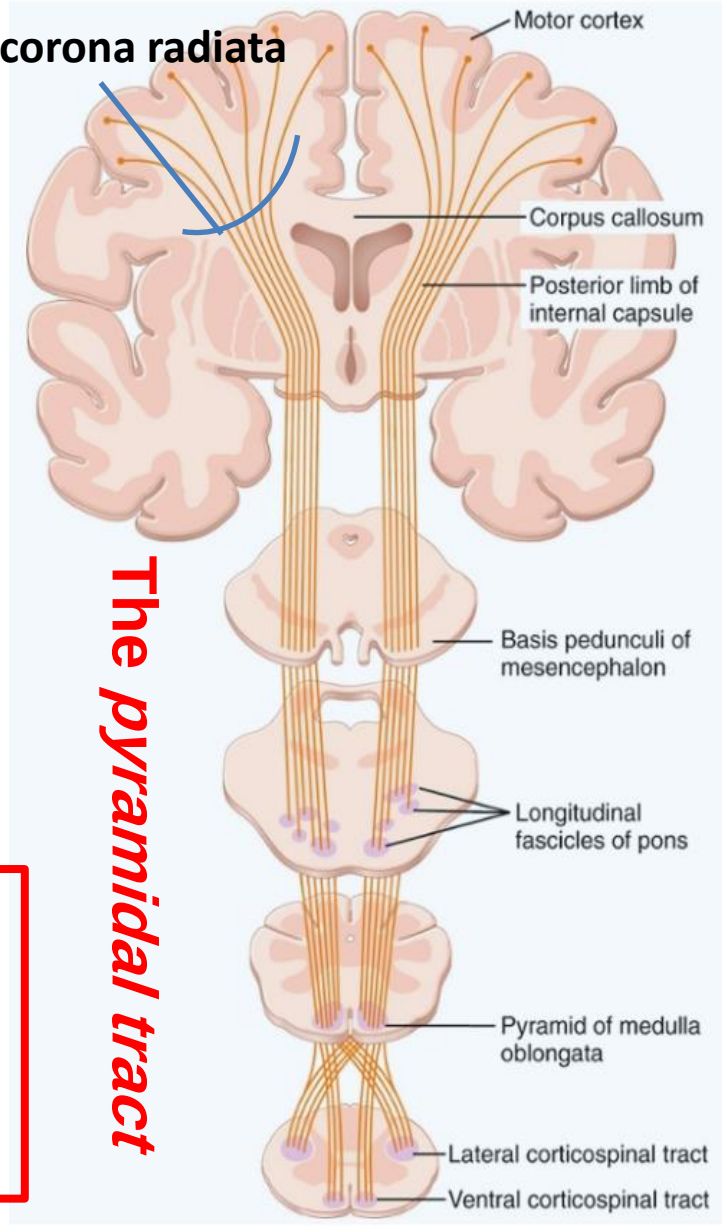
Transmission of Motor Signals from Motor Cortex

Motor signals are transmitted from the motor cortex to the spinal cord by **motor neurons** *directly & indirectly*

- **Direct:** through the ***corticospinal tract (most important)***
- **Indirect:** through multiple accessory pathways involving:
  - The basal ganglia,
  - Various nuclei of the brain stem.

Origin of pyramidal tracts:

1. ~ 30 % primary motor cortex
2. ~ 30% premotor & supplementary M areas
3. ~ 40% somatosensory areas



What Are Upper & Lower Motor Neurons?

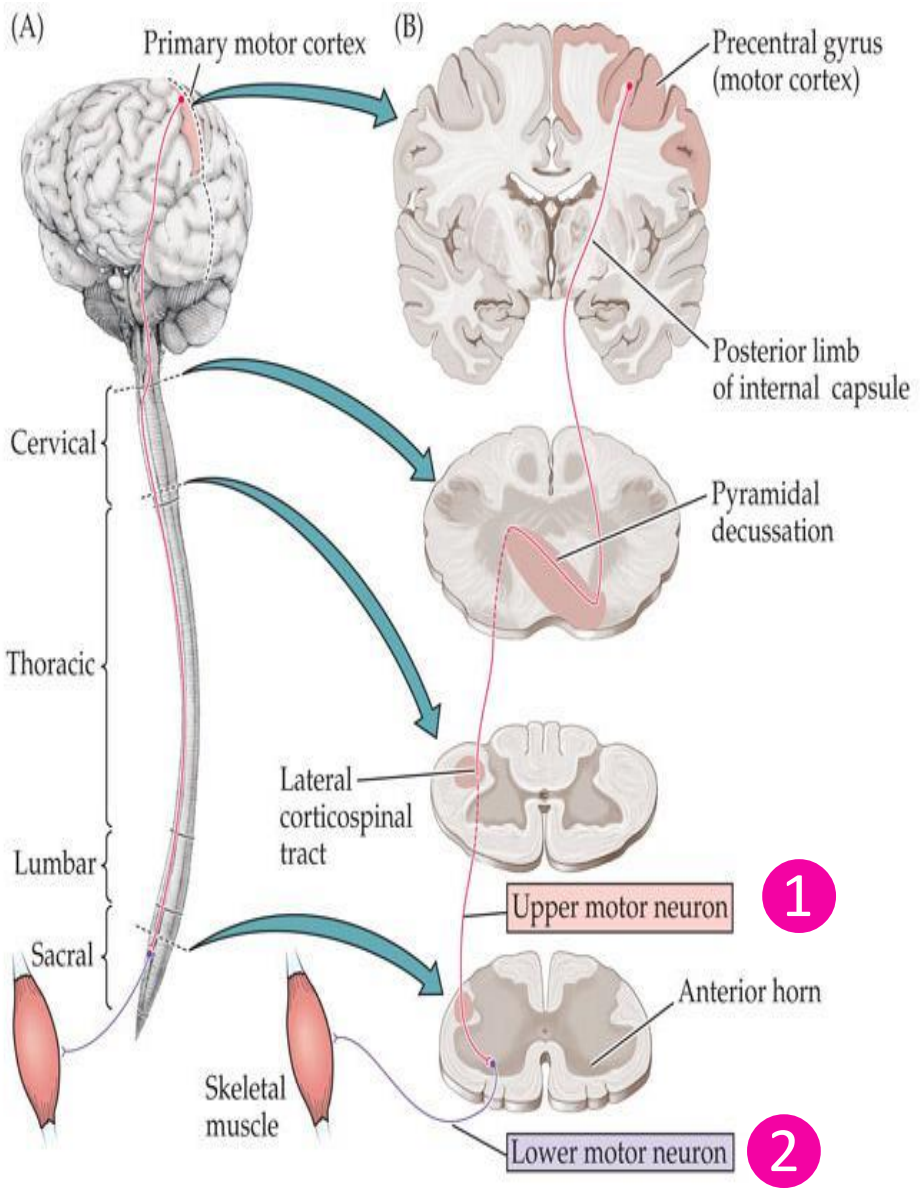
CNS controls the activity of **skeletal muscles** through two types of motor neurons:

1. **Upper motor neurons (UMN)**

- Are motor neurons that originate in the motor cerebral cortex or in the brain stem
- Convey motor information down to the lower motor neurons

2. **Lower motor neurons (LMN)**

- Located in either the ventral spinal cord or the cranial nerve nuclei of the brain stem
- Activate skeletal muscles to produce movements



What Are Upper Motor Neurons (UMN)?

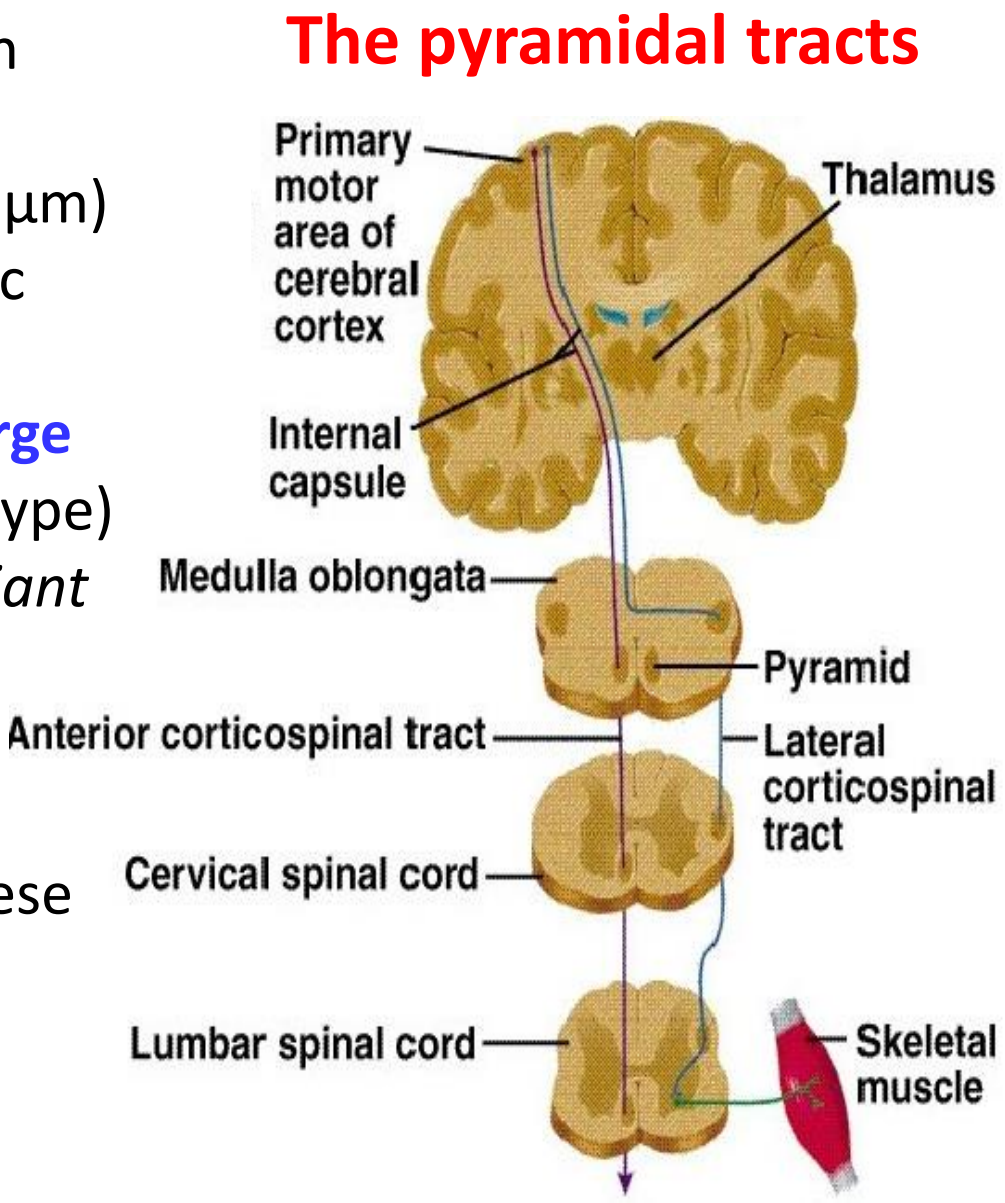
- **UMNs control lower LMNs through two different pathways:**
  - Pyramidal tracts
  - Extra pyramidal tracts

Extrapyramidal tracts:

- Tectospinal (terminate in upper cervical cord)
- **Rubrospinal tract** (facilitate flexor motor neurons).
- **Medial vestibulospinal** (terminate at C3)
- **Corticobulbar tract**
- **Lateral vestibulospinal** (facilitate **ipsilateral** extensor motor neurons and **gamma motor neurons)**
- **Pontine Reticulospinal** (facilitate extensor spinal reflexes)
- **Lateral (Medullary) Reticulospinal (powerfully suppress extensor spinal reflex activity)**

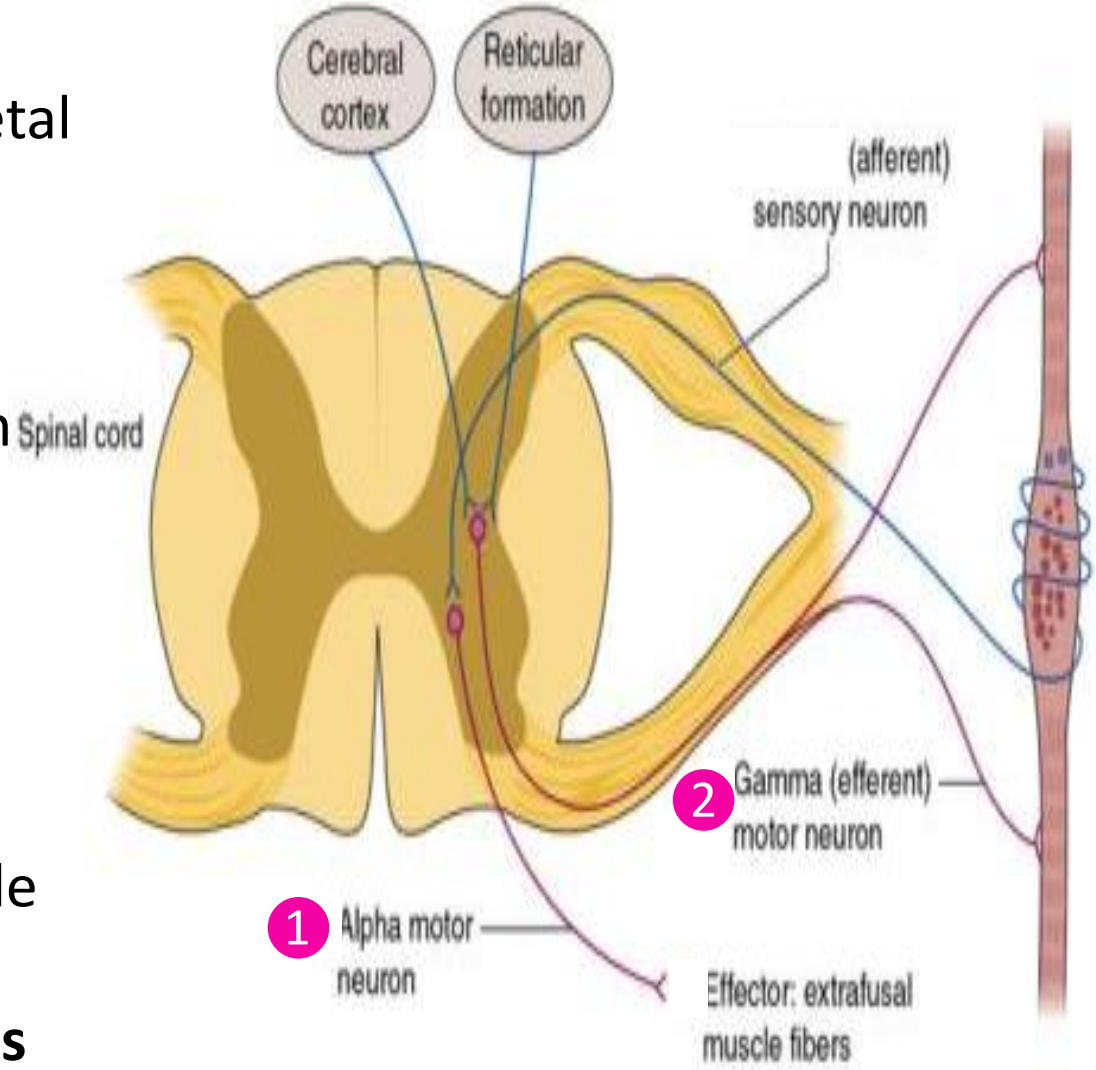
Upper Motor Neurons of the Pyramidal Tracts?

- There is > 1 million of fibers in each corticospinal tract
- ~ **97 %** of these are **small** (<4 µm) and conduct background tonic signals to the the spinal cord.
- Only ~ **3%** of the fibers are **large** (16 µm) fast conducting (Aβ-type)
- These fibers originate from *giant* ***Betz cells (~*** 60 *µm)*
- These cells are found only in the **primary motor cortex**.
- There are about 34,000 of these large Betz cell fibers in each corticospinal tract.



What Are Lower Motor Neurons (LMNs)?

- They are the final link between the CNS and skeletal muscles
- Found in the ventral spinal cord and motor nuclei of cranial nerves in brain stem
- Those of **cranial nerves** control eye movements, chewing, swallowing etc.
- **There are 2 types:**
  1. **Alpha motor neurons** innervate extrafusal muscle fibers
  2. **Gamma motor neurons** innervate intrafusal muscle fibers (control **muscle tone**).



γ-motor neurons fire spontaneously in the absence of input from M reticular formation

Muscle Tone

- The resistance of a muscle to stretch is often referred to as its **tone** or **tonus**.
- If the motor nerve to a muscle is severed, the muscle offers very little resistance and is said to be **flaccid**.
- **A hypertonic (spastic) muscle** is caused by **hyperactive stretch reflexes** due to excessive firing of gamma motor neurons resulting from loss of inhibition normally caused by the medullary reticulospinal tract (which is cut in UMN lesion).

Causes of muscle spasticity:

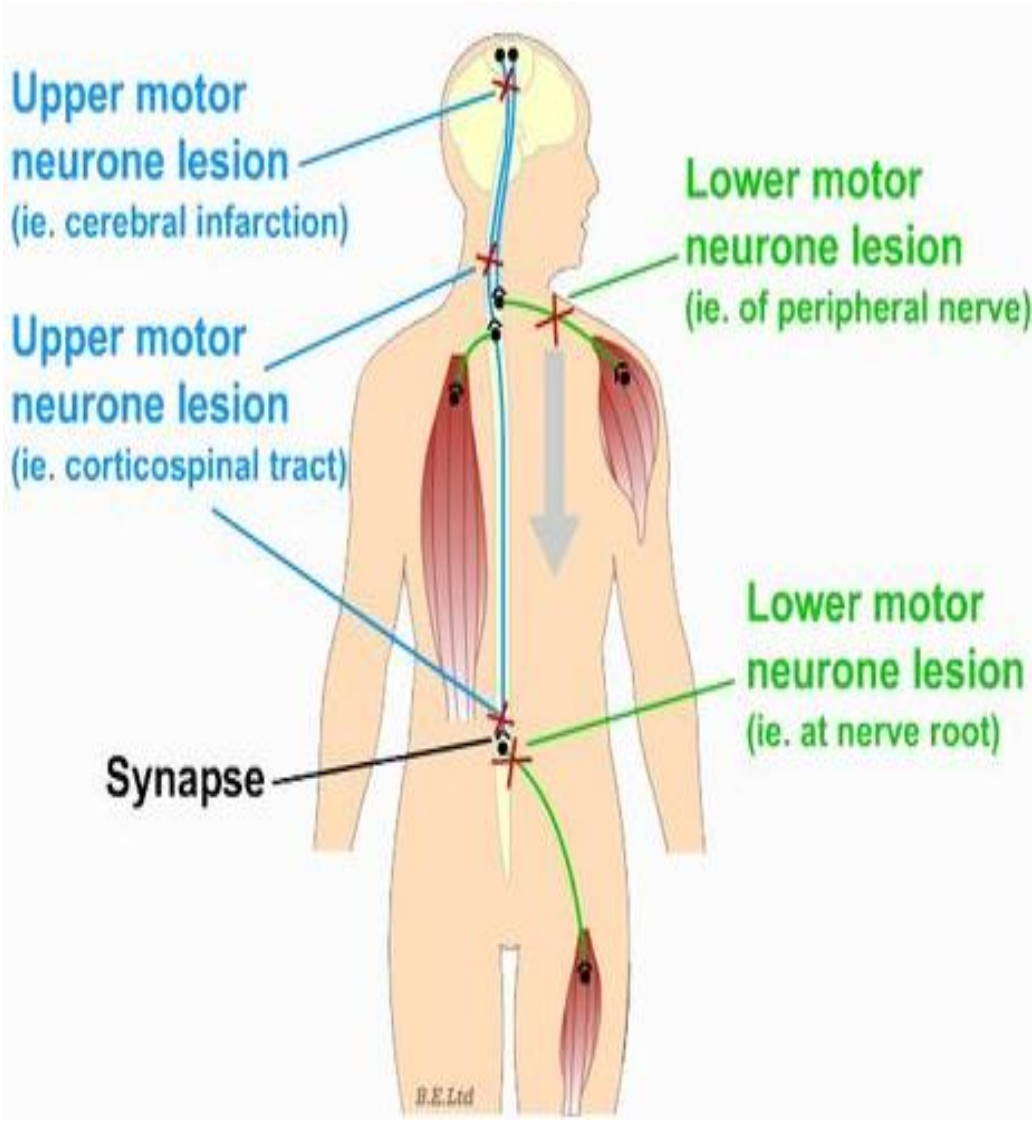
1 Stroke 2 Spinal cord injury 3 Multiple Sclerosis 4 Brain injury (trauma , etc), 5 Parkinsonism

Patients complain of pain, stiffness & inability to relax. Prolonged stiffness leads to bone & joint deformities with disability

What Are Upper & Lower Motor Neuron Lesions?

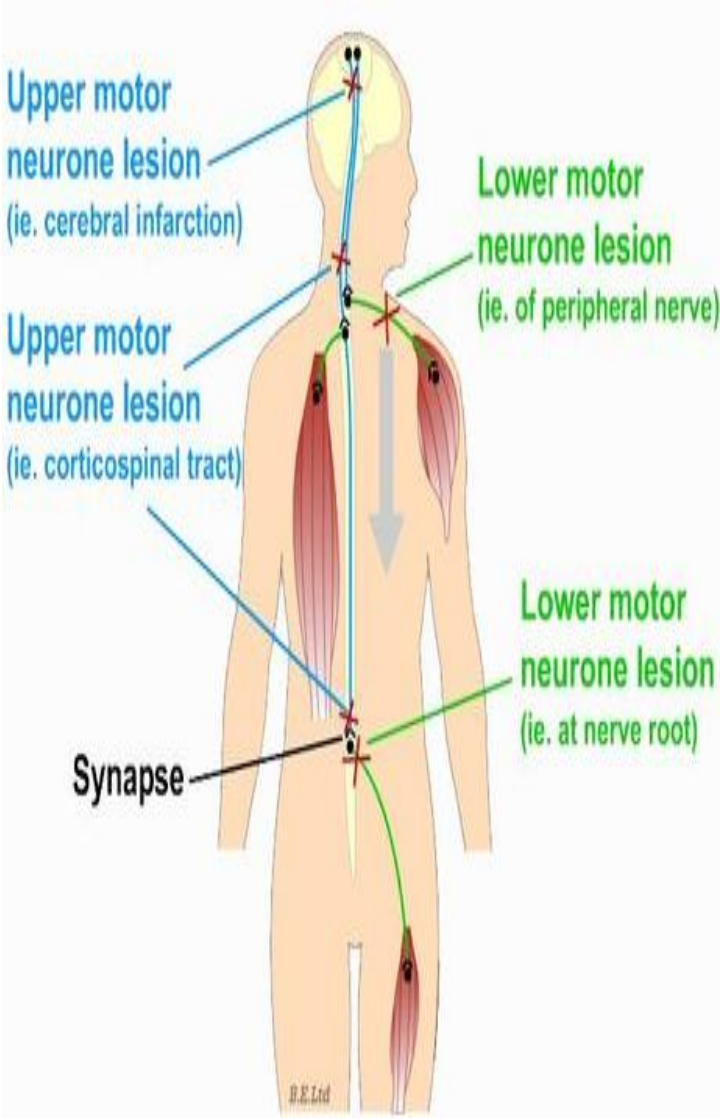
- **Upper motor neuron lesion (UMNL):** is a lesion of the descending neural pathway above the level of lower motor neurons

- **Lower motor neuron lesion (LMNL)** is a lesion that affects LMNs and their **efferent nerve fibers** traveling to their effectors (skeletal muscles)



What Are Upper & Lower Motor Neuron Lesions?

- **An UMNL** indicates pathology in the cerebral cortex, brain stem or spinal cord
- Interruption of the inhibitory influences leads to increased reflexes and increased muscle tone (stiffness or **spasticity)**
- Pathologic reflexes such as Babinski may also appear
- **A LMNL** indicates pathology in lower motor neurons
- Interruption of the reflex arc leads to **absent** or **decreased reflexes**
- Disconnection of the muscles from motor neurons result in atrophy (wasting)
- Muscle tone may be normal or decreased (**flaccid paralysis**).



What Causes Lesions of Upper & Lower Motor Neurons

Upper Motor Neuron Lesion (UMNL)

Can be due to:

1. **Cerebral stroke** by haemorrhage, thrombosis or embolism
2. **Spinal cord transection** (e.g. **tumor or trauma)** or hemisection (Brown-Sequard syndrome)

Lower Motor Neuron Lesion (LMNL)

Can result from:

1. **Lesions of ventral horn neurons** (e.g. , poliomyelitis, motor neuron disease)
2. **Lesions of spinal root or peripheral nerve lesion** (e.g. trauma or compression)

Upper Motor Neuron Lesion (UMNL)

- The supraspinal centers exert both **inhibitory** & **facilitatory** effects on the spinal cord, but the net inhibition exceeds the net excitation

➢ **In upper motor neuron (pyramidal) lesion:**

- The spinal cord is disconnected from the modulating influences of the supraspinal controlling centers.
- After a period of "**spinal shock** ", the stretch reflex recovers, but resumes function in a primitive and uninhibited manner:
  - There are exaggerated tendon reflexes and " spastic " increase in muscle tone
  - Hyperflexia is greater in the extensors of the lower limbs and the flexors of the upper limbs which are normally inhibited by the **medullary reticulospinal tract.**

Hallmarks of Upper Motor Neuron Lesion

- **Spasticity** (an increase in tone in the **extensor** muscles (lower limbs) or **flexor** muscles (upper limbs)
- **Weakness** in the **flexors** (lower limbs) or **extensors** (upper limbs), but no muscle atrophy/wasting
- **Clasp-knife response** (initial resistance to movement is followed by relaxation)
- **Hyperreflexia** (deep tendon reflex) and **Babinski sign (**present)
- **Loss of voluntary skillful movements**
- **Pseudobulbar palsy:** results from bilateral lesions of UMNs that transmit signals to the brain stem (**bulb-shaped structure**) LMNs (that control the muscles of the tongue (**XII)**, face (**VII**), speech and swallowing (**IX and X**):
  - Progressive loss of the ability to speak, chew, and swallow
  - Individuals may have outbursts of laughing or crying.

Effect of a Lesion in Different Parts of the Motor System-1

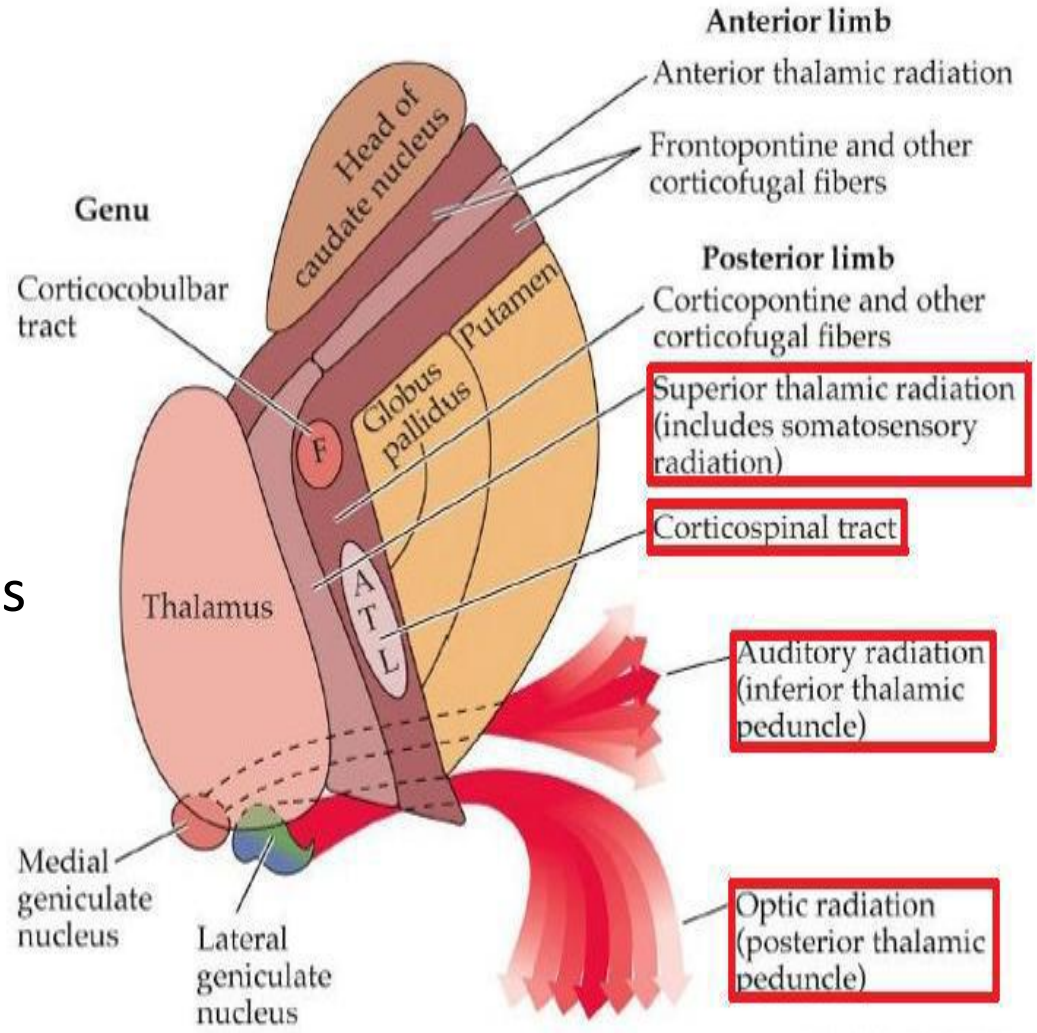
- Lesions of pyramidal tract cause paralysis of the UMNL type below the level of the lesion
- The side affected and the extent of paralysis vary according to the site of the lesion:

1. **In area 4:** restricted paralysis (e.g. contralateral monoplegia, because area 4 is widespread and is rarely damaged completely)
2. **In the corona radiata:** contralateral monoplegia or hemiplegia, depending on the extent of the lesion.
3. **In the internal capsule:** contralateral hemiplegia because almost all fibers are injured
4. **In the brain stem:** contralateral hemiplegia & ipsilateral paralysis of the cranial nerves as follows:
   - Midbrain lesion: the III and IV
   - Pons lesion: V, VII and VIII.
   - Medulla lesion: IX, X, XI and XII

Bilateral lesion in brain stem is rare and leads to quadriplegia and bilateral paralysis of the cranial nerves.

The internal capsule-1

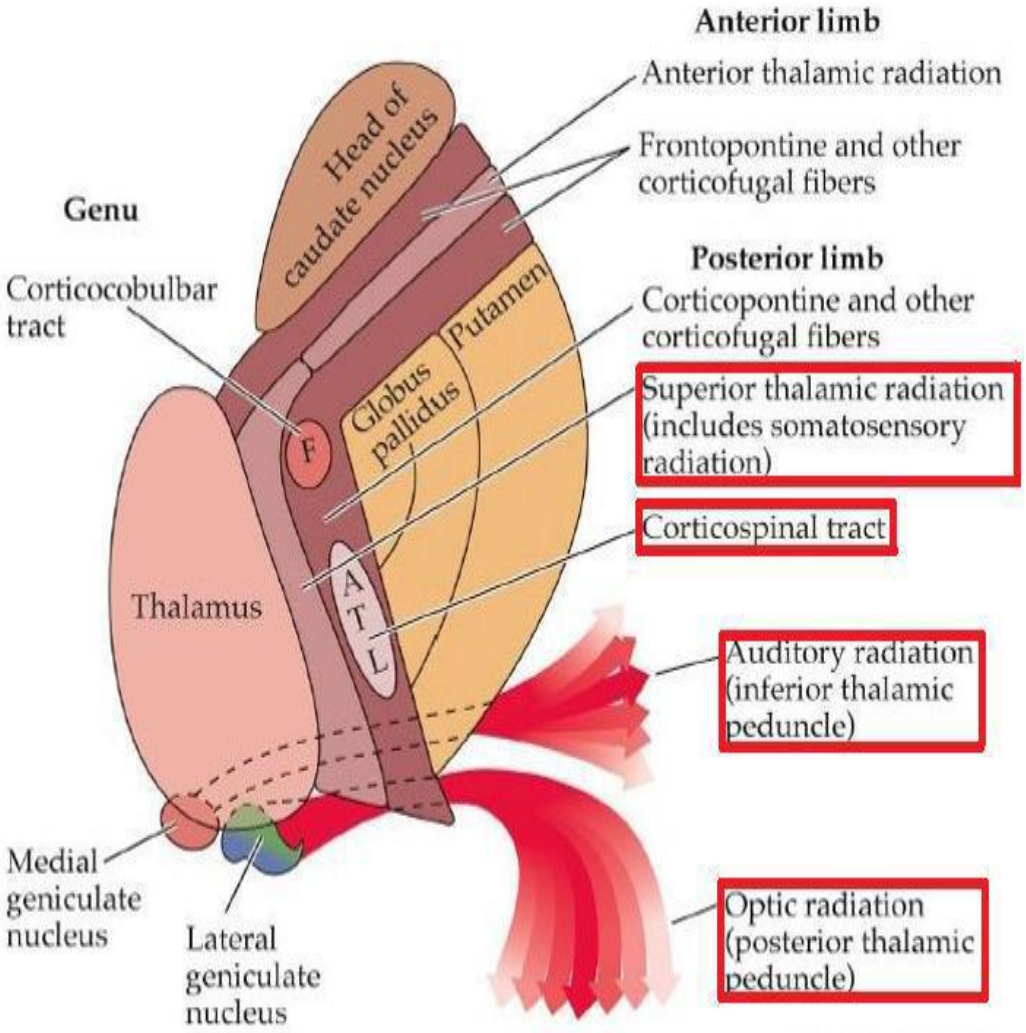
- The internal capsule is the **only pathway** containing cortical ascending and descending nerve fibers
- It is V-shaped, consisting of anterior & posterior limb and a genu (knee).
- It is surrounded by the putamen, globus pallidus, caudate nucleus & thalamus
- **The anterior limb**: contains fibers to red nucleus, pons cerebellum & thalamus,
- **The genu** Contains corticobulbar tract



The internal capsule-2

The posterior limb contains:

- Descending pyramidal & extrapyramidal fibers
- The somatosensory fibers from thalamic nuclei to cortical sensory areas
- The ascending fibers from the lateral geniculate nucleus (thalamus) to visual cortex.
- The ascending fibers from the medial geniculate nucleus (thalamus) to the auditory cortex



Effects of a Unilateral Lesion in Posterior limb of internal capsule-1

- Such lesion is called **cerebral stroke**
- It is usually caused by **thrombosis** or **hemorrhage** of **lenticulo-striate artery** (a branch of the middle cerebral artery).
- Patients pass into an **acute** then **chronic stage**.

1 **Acute stage**: lasts a few days up to 2-3 weeks. It is characterized by acute UMNL manifestations in the opposite side:

- Paralysis including the upper and lower limbs, the lower parts of the face and half of the tongue.
- Hemianaethesia (loss of all sensations, due to damage of the thalamocortical fibers).
- Hypotonia and areflexia & loss of the superficial reflexes.
- Babinski's sign may be present

Manifestations of this stage are similar to those of LMNL, but the extent of paralysis is much more than that of LMNL.

Effects of a Unilateral Lesion in Posterior limb of internal capsule-2

❷ Chronic (permanent or spastic) stage

The main manifestations of this stage include:

- **Contralateral hemiplegia (**paralysis of the opposite half of the body) of the UMNL type (partial recovery occurs after a variable period (possible walking), but the fine skilled movements are permanently lost).
- **Permanent loss of fine sensations** in the opposite side, but the crude sensations recover gradually.
- **Contralateral homonymous hemianopia** (loss of vision in the two corresponding haves of the visual fields opposite to side of lesion due to injury to optic radiation
- Injury of left optic radiation causes blindness of the right halves of visual field
- **Diminished hearing power** in both ears (by about 50 %), because of damage of auditory radiation.

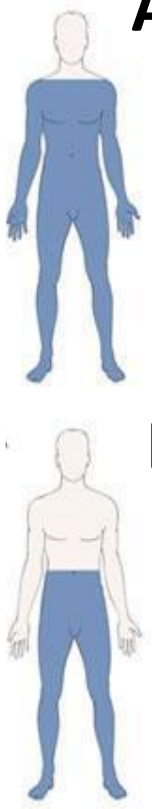
Transection of Spinal Cord-1

The effects of spinal cord transection are dependent on whether it is:

1. Complete transection 2. Hemisection

A. The effects of complete transection of the spinal cord (e.g. tumor or trauma) vary according to the level of transection:

- In the upper cervical region **immediate death** follows, due to paralysis of all respiratory muscles
- In lower cervical region below C5 → quadriplegia (paralysis in **4 limbs, A**)
- In the thoracic region → **paraplegia** (paralysis in **both lower limbs, B**)



Paraplegia has 3 stages:

A. **Spinal shock (2-6 weeks)**
B. **Recovery of reflex activity**
C. **Paraplegia in extensors**

Voluntary movements and sensations are permanently lost

Paraplegia Stages: A. Spinal Shock

Spinal shock (2-6 weeks): loss of **sensations** accompanied by **motor paralysis** with initial loss but gradual **recovery of reflexes**

Immediately following transection there is:

- Paralysis of all muscles below the lesion
- Loss of reflexes and loss of tone (flaccidity)
- Loss of all sensations (anaesthesia) and voluntary movements below the level of the lesion due to interruption of all sensory and motor tracts.
- Loss of muscle tone **(flaccidity),** and vasomotor tone (vasodilation) leading to fall in blood pressure
- Bladder urinary retention with overflow due to paralysis of the wall of the urinary bladder

Cause of spinal shock: sudden withdrawal of supraspinal facilitation on the spinal alpha motor neurons i.e loss of continual tonic discharge transmitted along the excitatory **pontine reticulospinal**, **vestibulospinal** and **corticospinal tracts**.

Paraplegia Stages: B. Return of reflex activity-1

- As the spinal shock ends, spinal reflex activity returns
- The partial recovery may be due :
  - Increased excitability of the spinal cord neurons presumably to compensate for the loss of supraspinal facilitatory influences
  - Disinhibition of motor neurons as a result of absence of inhibitory impulses from higher motor centers
  - Hypersensitivity to excitatory neurotransmitters .

Features of this stage

- Gradual rise of arterial blood pressure due to return of spinal vasomotor activity in the lateral horn cells
- Exaggerated tendon reflexes and spasticity
- Return of visceral reflexes (micturition & defecation)

Paraplegia Stages: B. Return of reflex activity-2

- **Mass reflex:** a minor painful stimulus to the skin of the lower limbs causes
  - Withdrawal and evoke other autonomic reflexes (bladder and rectum emptying, sweating , blood pressure rise) through spread of excitation (by irradiation)
- Voluntary movements and sensations are permanently lost
- Human patients with complete transection never recover fully because effective regeneration never occurs in the human`s CNS.

Paraplegia Stages: C. Extensor Paraplegia

C. Stage of extensor paraplegia

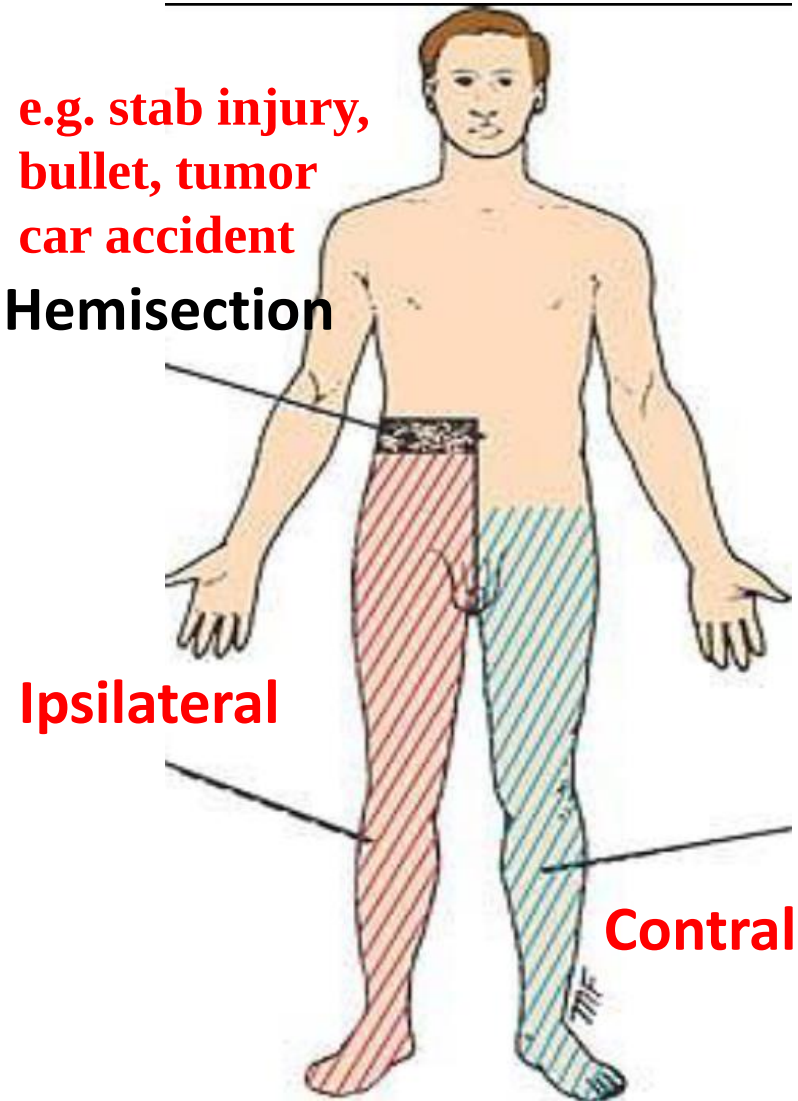
- The tone in extensor muscles returns gradually to exceed that in the flexors
- The lower limbs become spastically extended.
- Extensor reflexes become exaggerated, as shown by tendon jerks and by the appearance of clonus.
- The positive supportive reaction returns and the patient can stand on his feet with appropriate support
- Return of the withdrawal reflex and crossed extensor reflex

B. Hemisection of the Spinal Cord

Brown-Séquard syndrome (hemiparaplegic syndrome)

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)

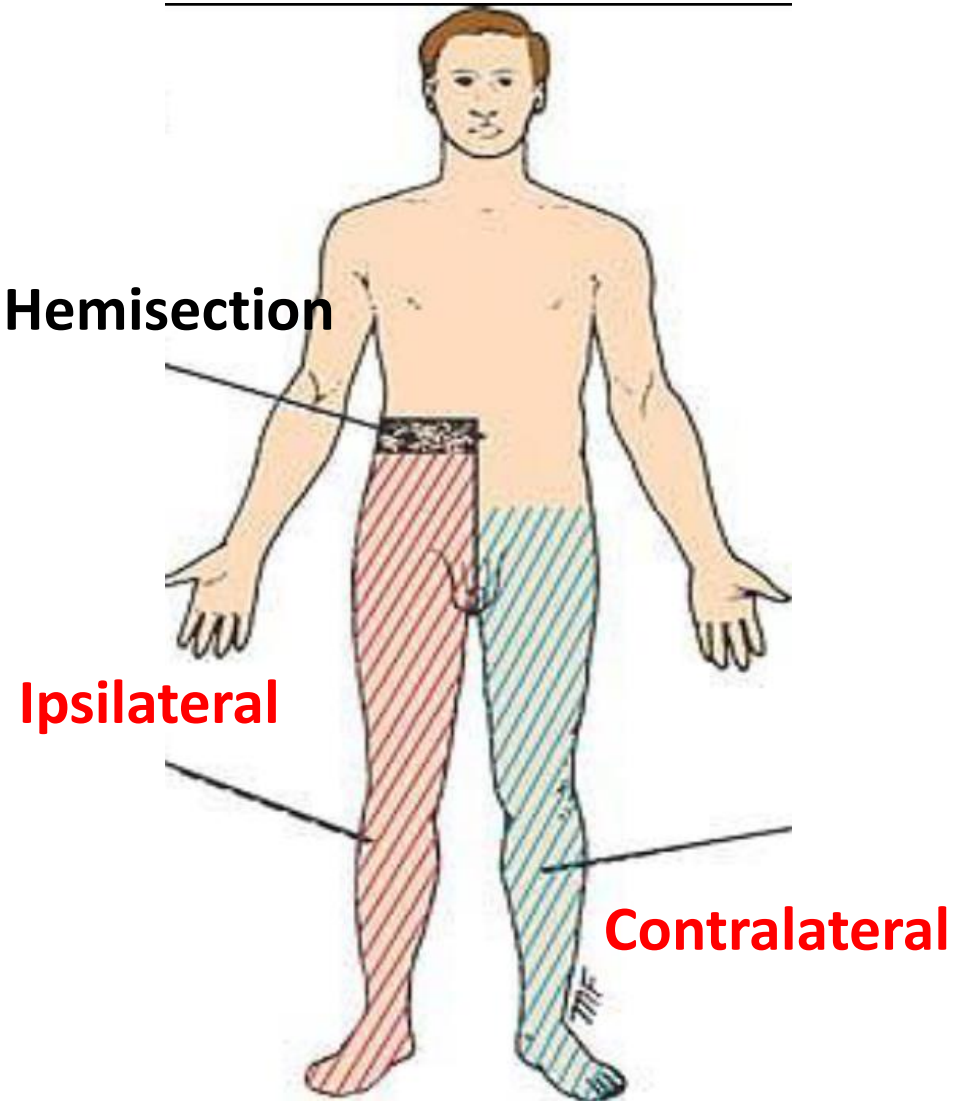


B. Hemisection of the Spinal Cord

Brown-Séquard syndrome (hemiparaplegic syndrome)

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)



Lower Motor Neuron Lesion (LMNL)-1

LMNs constitute part of the reflex arc. The integrity of the reflex arc is essential for maintenance of muscle tone

LMNL is a lesion which affects nerve fibers traveling from the anterior horn of the spinal cord to the relevant muscle(s)

- Associated with areflexia and muscle hypotonicity or atonia
- Leads to flaccid paralysis (paralysis accompanied by muscle loss or atrophy/wasting).
- The denervated muscle fibers depolarize spontaneously causing **fibrillations** potentials (not visible to the naked eye , but detectable only by electromyography, EMG)

Lower Motor Neuron Lesion (LMNL)-2

- Re-innervation of denervated fibers from neighbouring motor units may occur
- This causes spontaneous depolarization of the re-innervated muscle fibers causing **fasciculations** (visible contractions of groups of motor units)
- Fasciculations indicate partial re-innervation.

BULBAR PALSY

- Is a similar disorder as pseudobulbar palsy but is caused by lower motor neuron lesions
- It consists of LMN signs in regions innervated by the facial (VII), glossopharyngeal (IX), Vagus (X) and hypoglossal (XII) cranial nerves

Differences Between Upper & Lower Motor Neuron lesions

Upper Motor Neuron Lesion (UMNL)

- No wasting
- Loss of skilled finger/toe movements
- Increased tone of clasp-knife type
- Weakness mostly in anti-gravity muscles
- Increased reflexes and clonus
- Extensor plantar responses.

Lower Motor Neuron Lesion (LMNL)

- Wasting
- Fasciculation
- Decreased tone (i.e. flaccidity)
- Weakness in body muscles
- Decreased or absent reflexes
- Flexor or absent plantar responses.


Thank You
Any Questions
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