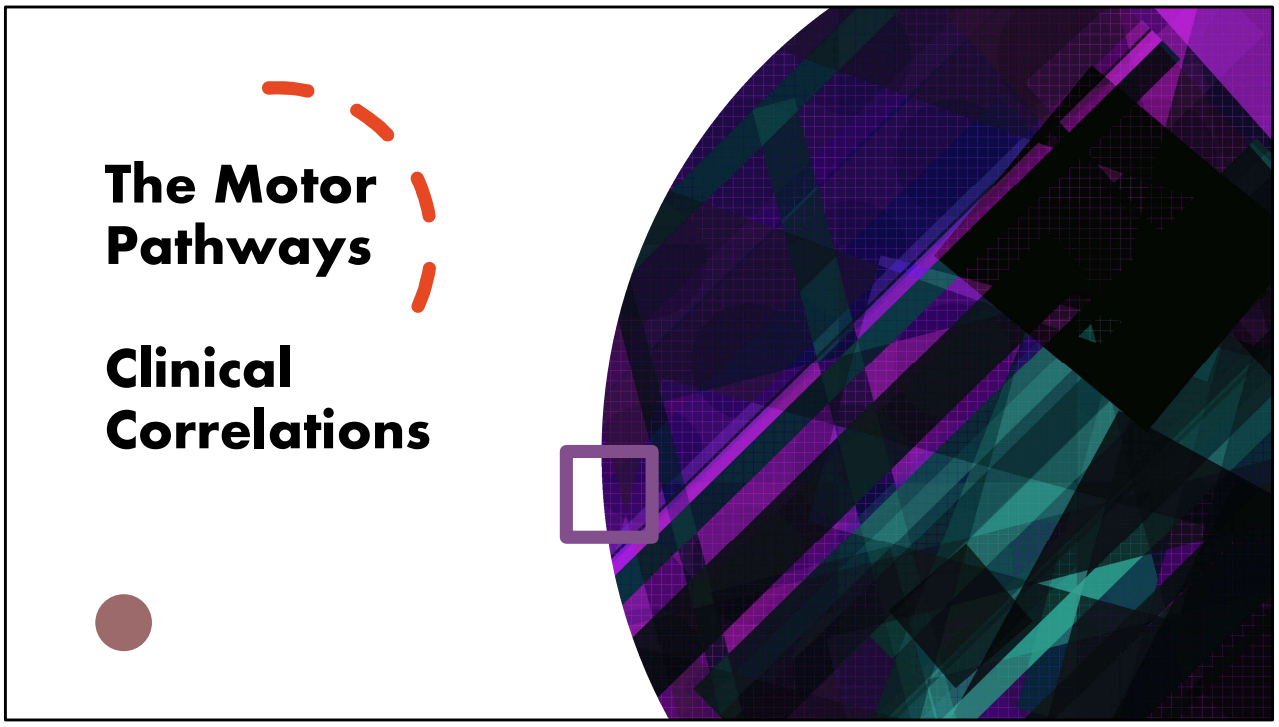
The Motor Pathways

Clinical Correlations



Learning Objectives

1. Explain the concept of upper and lower motor neurons
2. Explain and recognize the signs and symptoms produced by upper and lower motor neuron lesions
3. Apply the acquired knowledge to solve clinical cases.

Upper and Lower Motor Neurons

OBJ. # 1

Upper motor neuron	Lower motor neuron
Cell bodies and fibers controlling the activity of lower motor neurons and interneurons	α-motor neurons in ventral horn of spinal cord and α-motor neurons in the motor nuclei of the cranial nerves. They directly innervate muscle fibers.
most in cortex	last part of path

The concept of an upper and lower motor neuron is vital to your understanding of neurologic deficits. This will be one of a few keys to localizing the site of a lesion within the nervous system. esp when weakness is a consideration

Upper motor neurons refers to the components of the descending motor pathways before they reach the lower motor neurons. This does not include the basal ganglia or the cerebellum.

Upper Motor Neuron Lesion

OBJ. # 1

Damage to the cell bodies of origin and /or axons of UMN. The consequence is a loss of input to and modulation of the function of Lower motor neurons (LMN).

The symptoms and signs of patients affected of UMN deficits include:

- Muscle **weakness** / paralysis
- Increase deep tendon reflex responses: **Hyperreflexia**
- Increase in muscle tone: Hypertonicity, **Spasticity**
- Abnormal reflexes: Babinski sign (upgoing toes)
- Decreased superficial reflexes (superficial abdominal)

Remember that lower motor neurons receive input from much more than just upper motor neurons. When they lose the input of an upper motor neuron, many changes in their output take place. Weakness is common to both upper and lower motor neuron lesions, but the other findings here help to distinguish the two.

UMN lesions associated with hyperreflexia, spasticity, and up going toes

OBJ. # 1

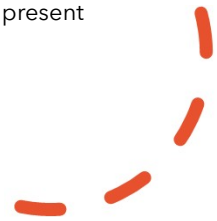
Muscle tone

- Muscle tone represents the resting level of tension in a muscle
- Depends on the resting level of firing of alpha motor neurons
- Which depends on inputs to alpha motor neurons from:
  - Descending motor pathways
  - Afferent limb of the reflex arcs
- Increase muscle tone is called **hypertonia**
- Decreased muscle tone is known as **hypotonia / flaccidity**

Spasticity

- Characterized by a velocity dependent increase in resistance to passive limb movements (stretch stimulus)
- Upper limb flexor muscles are more affected than extensors
- Lower limb extensor muscles are more affected than flexors
- The clasp-knife phenomenon is present

push push push collapse



OBJ. # 1

Lower Motor Neuron Lesion

Damage to α-motor neurons in the brainstem or ventral horn of the spinal cord, ventral roots or motor fibers in a spinal or cranial nerve. Patients with LMN deficits present with the following symptoms and signs:

- Muscle **weakness** / paralysis
- Decrease tone: **Hypotonia**
- Decrease reflexes: **Hyporeflexia**
- Muscle atrophy due to denervation and disuse (chronic finding)
- **Fasciculation and fibrillation**. These are spontaneous twitches due to abnormal activity of damaged motor neurons or denervated muscle fibers respectively.

Again, weakness is present in both types of lesions, though complete paralysis is more common with a lower motor lesion. more redundancy in umn

OBJ. 2 & 3

Case Study

A 75-year-old man with atrial fibrillation presents with **left upper extremity weakness,** and **slurred speech** that began about 3 weeks prior. On examination you find 3/5 weakness throughout the left upper extremity, brisk reflexes at the left biceps and brachioradialis, and densely dysarthric speech. Where would you localize this lesion?

always check dtrs b/l to see asymmetry

UMN lesion

Motor Pathways

Damage to the corticospinal tracts at any level above decussation

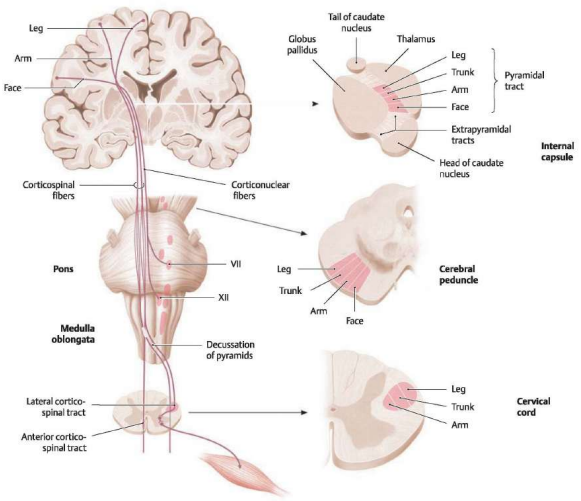
↓

Upper motor neuron deficits on the contralateral side of the body

↓

Contralateral Hemiplegia / Hemiparesis

OBJ. # 2 & 3



A Course of the pyramidal (corticospinal) tract

above CN - bc those cause dysarthria/slurred speech

somewhere between the pons and cortex on the contralateral side

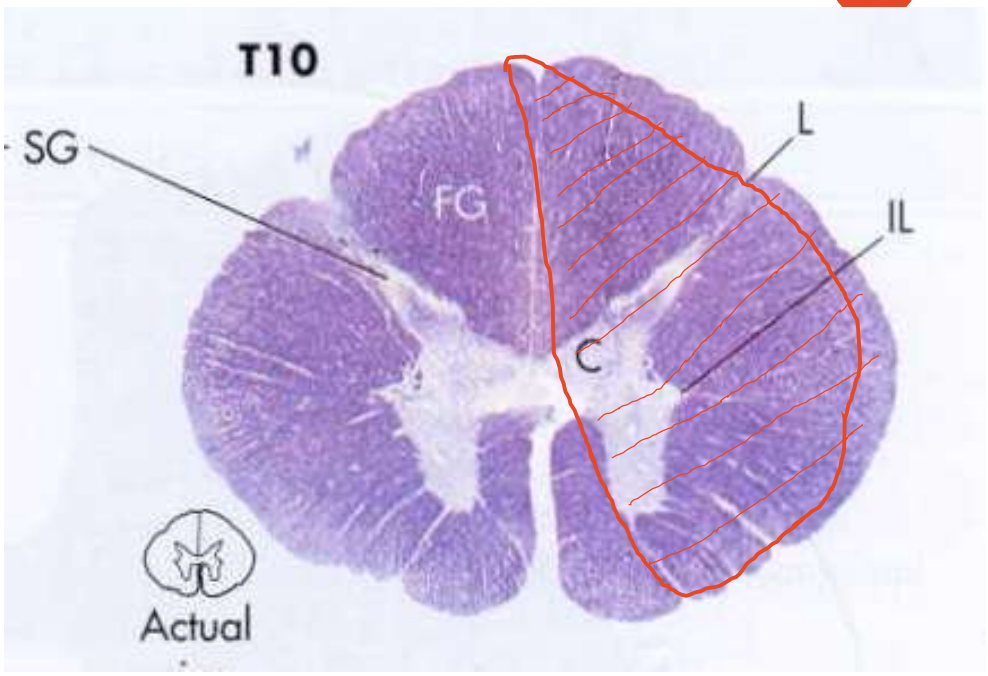
OBJ. 2 & 3

Case Study

A 30-year-old man was in a motorcycle accident and comes to the ER with trauma to the left lower dorsolateral **thoracic area**. Evaluation of the patient shows damage of the **lateral spinal cord at T10 vertebral level.**

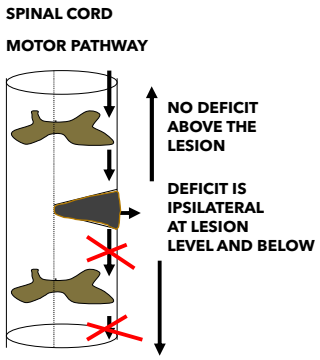
The patient presents with **paresis of the left side of the body and left lower limb** below the damaged area.

OBJ. 2 & 3



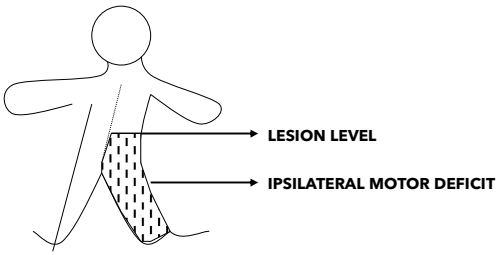
OBJ. 2 & 3

Motor Pathways



Lesion level

- At thoracic or lumbar levels - Lower Limb Paralysis
- At cervical levels - Hemiplegia sparing the face



below decussation = IPSI issues

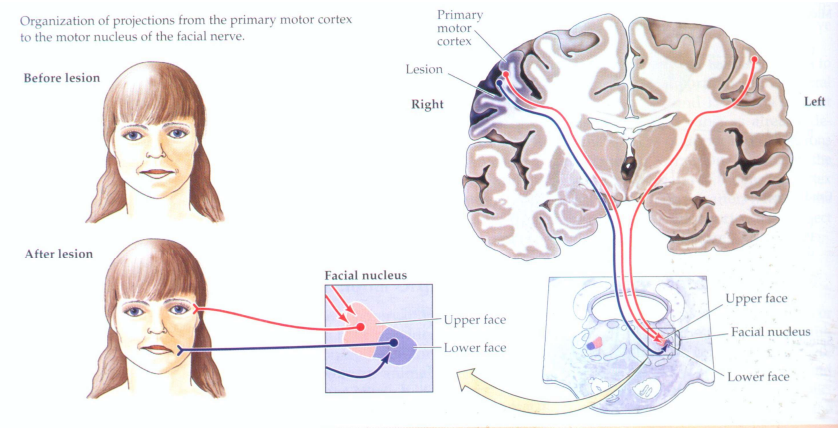
OBJ. 2 & 3

Case Study

A 79-year-old woman with a history of diabetes presents with sudden **weakness of the left arm and the lower part of the left side of the face. The forehead is spared.** Evaluation of the patient shows an obstruction of the **right middle cerebral artery** affecting the lateral aspect of the motor cortex.

OBJ. 2 & 3

Descending Pathways to Facial Motor Nucleus



forehead receives bilateral innervation - can move with either side of face only move with contralateral part of brain

forehead sparing = UMN not bells Palsy more stroke

fav of boards and Massa likes too

OBJ. 2 & 3

Case Study

When the patient was asked to open her mouth and say aaa…, a **flat palate on the left side** could be observed. The **uvula was tilted towards the right.** The **tongue deviated towards the left side.**

only contralateral innervation
weakness of palate it lowers - causing uvula to deviate toward non affected side
sticking tongue out is contraction - deviates toward weaker side = difficult to asse
facial droop

most massa exam questions are clinical scenarios