

Motor Pathways – Clinical Correlations

Learning Objectives: After studying this chapter students should be able to:

1. Explain the concept of upper and lower motor neuron
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.

Presentation Notes

Objective #1; Slide 3 - The concept of upper motor neuron and lower motor neuron

Upper motor neurons are in the motor cortex and the brainstem. They project down to control lower motor neurons. They are the origin of the descending motor pathways. They are known as the upper motor neuron pathways or corticospinal tracts. Their function is to modulate the activity of lower motor neurons. Lower motor neurons are α -motor neurons and γ -motor neurons located in the ventral horns of all spinal cord levels and in the motor nuclei of the cranial nerves in the brainstem. Thus, all the motor nuclei of the cranial nerves contain lower motor neurons whose activity is regulated by the upper motor pathways.

Upper and Lower Motor Neurons

OBJ. # 1

Upper motor neuron

Cell bodies and fibers controlling the activity of lower motor neurons and interneurons

Lower motor neuron

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.

Objective # 2; Slides 4, 5

Upper motor neuron (UMN) lesions occur when the neurons of origin of descending pathways in the cerebral cortex or the descending pathways themselves are damaged. This includes the trajectory of these long tracts in the spinal cord. A lesion to the spinal cord that damages these descending motor fibers produces upper motor neuron deficits.

Damage to the corticobulbar tracts innervating the lower motor neurons of the motor cranial nerves produces upper motor neuron deficits of the cranial nerves.

Upper motor neuron damage produces weakness with increased muscle tone, hyperreflexia, and pathologic reflexes. The combination of hypertonia and hyperreflexia is known as spasticity. It is characterized by resistance to rapid stretching of the muscle. Spasticity manifests differently in the upper and lower extremities. Extensor muscles are more affected in the legs and flexor muscles are more affected in the arms. This produces a characteristic pattern of hyperextension of the affected lower extremity with hyperflexion of the affected upper extremity. Pathologic reflexes can also become present

It is important clinically to differentiate the 2 main types of hypertonia: spasticity and rigidity. Spasticity is the hypertonia of UMN damage; rigidity occurs mostly due to damage of basal ganglia (nuclei) pathways as in Parkinson's disease. Rigidity is characterized by hypertonia that affects all muscle groups; the resistance to movements is independent of the velocity of the movement.



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
- **Fasciculation and fibrillation.** These are spontaneous twitches due to abnormal activity of damaged motor neurons or denervated muscle fibers respectively.

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)

Objective # 2; Slide 6

Lower motor neuron lesions occur when the α -motor neurons in the ventral horns, the ventral roots, or the motor axons in a spinal nerve are damaged. Damage to the α -motor neurons in the motor nuclei of the cranial nerves also produces lower motor neuron deficits. A lower motor neuron deficit is characterized by weakness with decreased muscle tone, depressed reflexes, and atrophy (chronic finding.) Fasciculations are abnormal twitches due to spontaneous activity of groups of muscle cells. Fibrillations occur due to denervation of the muscle fibers.



OBJ. # 1

Lower Motor Neuron Lesion

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Objective 3; Slides 7, 8

Damage to the corticospinal tracts above the decussation of the fibers at the spinomedullary junction produces upper motor neuron deficits on the contralateral side of the body.

OBJ. 2 & 3

Case Study

A 75-year-old man with a history of diabetes and coronary artery disease presents with **sudden weakness** of the **left arm** and **slurred speech**. Evaluation of the patient demonstrates an obstruction of the **right middle cerebral artery**

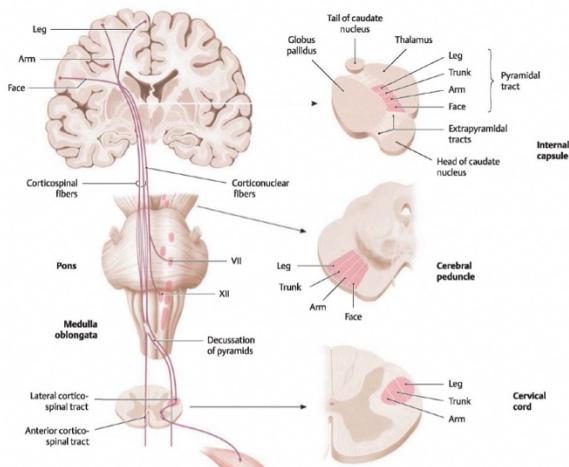
Motor Pathways

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

Illustrator: Markus Voll
Schuenke et al. THIEME Atlas of Anatomy • Head and Neuroanatomy
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Objective #3; Slides 9, 10, 11

Damage to the corticospinal fibers below decussation (in the spinal cord) produces an ipsilateral deficit. Damage at any spinal cord level produces no deficit above the lesion and an upper motor neuron deficit below the level of lesion. A lower motor neuron deficit may occur at the lesion level.

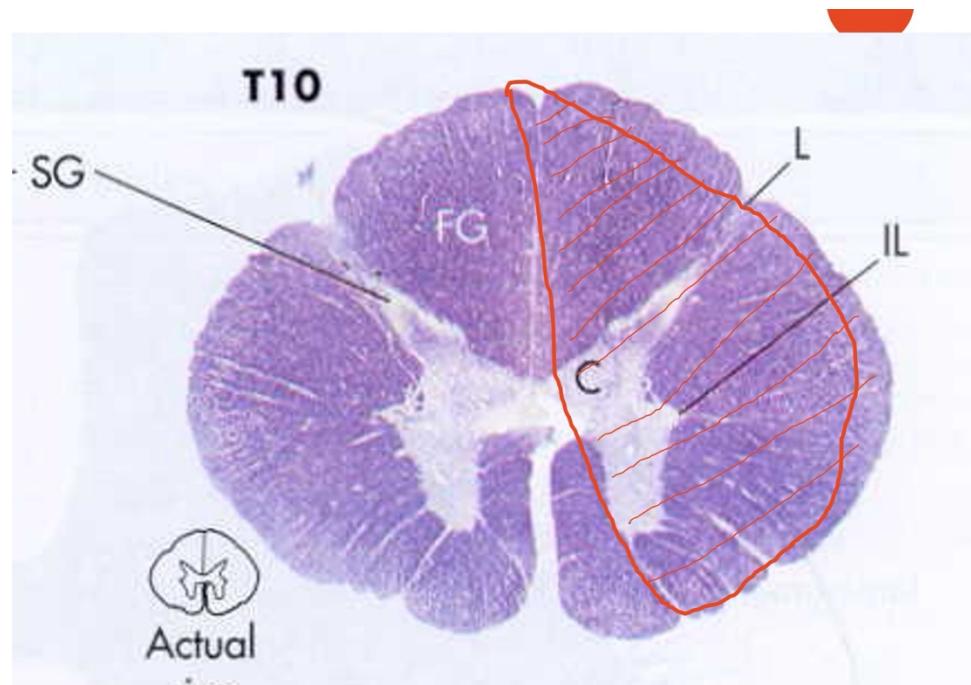
OBJ. 2 & 3

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.

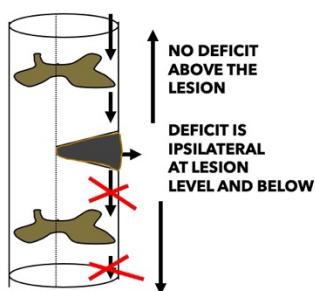
OBJ. 2 & 3



OBJ. 2 & 3

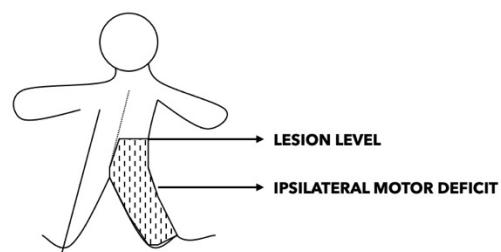
Motor Pathways

SPINAL CORD
MOTOR PATHWAY



Lesion level

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



Objective #3; Slides 12, 13

Upper motor neuron deficits can also occur due to damage of the corticobulbar tracts that regulate the activity of neurons in the cranial nerve nuclei. In these cases, there would be an upper motor neuron deficit on the contralateral face, associated with upper motor neuron deficits of the cranial nerves that innervate the facial muscles, palate and tongue. In most individuals, information from corticobulbar fibers to the cranial nerve motor nuclei come predominantly from the contralateral cortex, through the contralateral corticobulbar fibers.

One clinically important exception is the motor nucleus of the facial nerve. This nucleus is split into two portions, one containing lower motor neurons to the upper face and the other containing lower motor neurons to the lower face. Only the part which contains the lower motor neurons to the upper face receives bilateral innervation from the corticobulbar fibers. The result of this split is that patients with upper motor neuron facial weakness have a characteristic “forehead-sparing” pattern of facial weakness in which they have a facial droop but can lift the forehead on the affected side. Patients with Bell’s palsy, a lower motor neuron disease of the facial nerve, will have weakness of the entire affected side of the face.

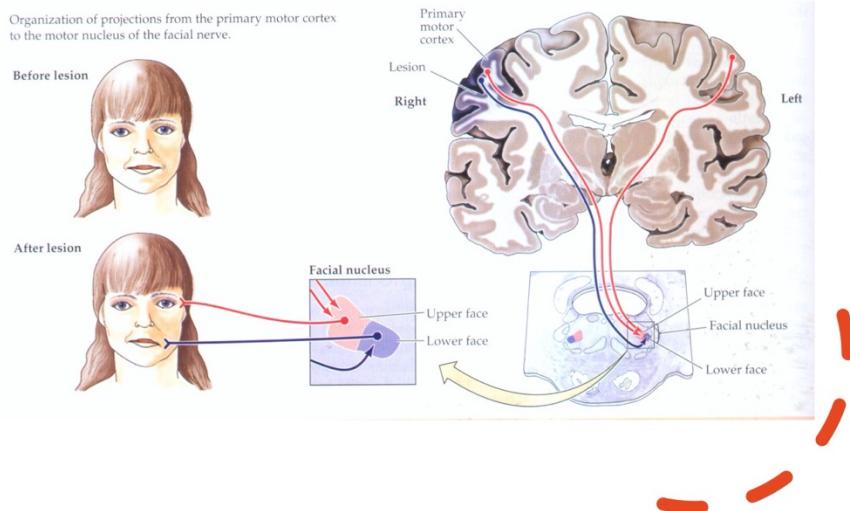
OBJ. 2 & 3

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



Objective #3; Slide 14

Cortical supply to the nucleus ambiguus (motor nucleus for CN IX and X) is mostly contralateral in most people. Damage to corticobulbar fibers to the nucleus ambiguus produces upper motor neuron deficits and these patients show a flat palate contralateral to the lesion side with the uvula tilted towards the side of the lesion and against the palate deficit.

The same corticobulbar fiber distribution is true for the hypoglossal nucleus. Upper motor neuron lesions or central lesions affecting the innervation of the hypoglossal nucleus produce deviation of the tongue away from the lesion side and towards the deficit side. Remember that all this is variable, and some patients will not present any palate or tongue deficits with corticobulbar lesions.

A lesion to the motor nucleus of a cranial nerve or its fibers produces a lower motor neuron deficit. In these cases, the deficit of function is on the same side of the lesion.

Damage to the facial nerve fibers in the periphery is known as Bell's palsy. It is characterized by a flaccid paralysis of the hemi-face including the forehead on the side of the lesion.

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**.



A lesion of the hypoglossal nucleus or its fibers produces deviation of the tongue towards the side of the lesion. Damage of the accessory nucleus or its fibers produces an ipsilateral lower motor neuron deficit.

Damage of the cranial nerve motor nuclei produces a LMN lesion, and the patient will experience LMN deficits.

Some diseases can affect lower motor neurons, upper motor neurons, or both producing motor deficits without sensory deficits. One of these motor neuron diseases is a degenerative disease named amyotrophic lateral sclerosis (ALS) or Lou Gehrig's disease. It produces progressive degeneration of both lower and upper motor neurons and will be the subject of a different lecture. See Blumenfeld chapter 6, page 241, Key Clinical Concept.

When analyzing a case, you need to think first about what type of lesion the patient presents: is it a UMN or a LMN deficit?

The second step in the analysis of a case is the localization of the lesion. What elements can help us with localization?

If the patient has an UMN deficit on the same side of both body and face simultaneously, the lesion probably is of the corticospinal and corticobulbar tracts above the pons – the involvement of facial nucleus is very important in determining the location here. The lesion could be in the cerebral peduncles, in the internal capsule, in the corona radiata or directly in the cortex. We will see later in the course, that there are other elements associated with those places that help us better define the location. If the patient has an UMN deficit on one side of the body that includes an UMN deficit of the palate and tongue on the same side, but without facial muscle deficits, the lesion is of the corticospinal and corticobulbar tracts below the nucleus of VII, probably at the level of the upper medulla. If the patient has an UMN deficit on one side of the body, and face deficits that are contralateral to the body deficit, it is likely that the lesion is in the brainstem and the face deficit (the facial muscles, palate, or tongue) is a LMN deficit. In this case the LMN deficit indicates the lesion site. If the patient has an UMN deficit of the body without involvement of the face, (facial muscles, palate or tongue) it indicates that the deficit is in the spinal cord.

The third step in the analysis of a case is the etiology: vascular, infectious, neoplasia, demyelination, immunological etc.

Finally, if you suspect a vascular lesion, try to identify the artery involved.

Terms used to describe weakness - See Blumenfeld, chapter 6, table 6.5