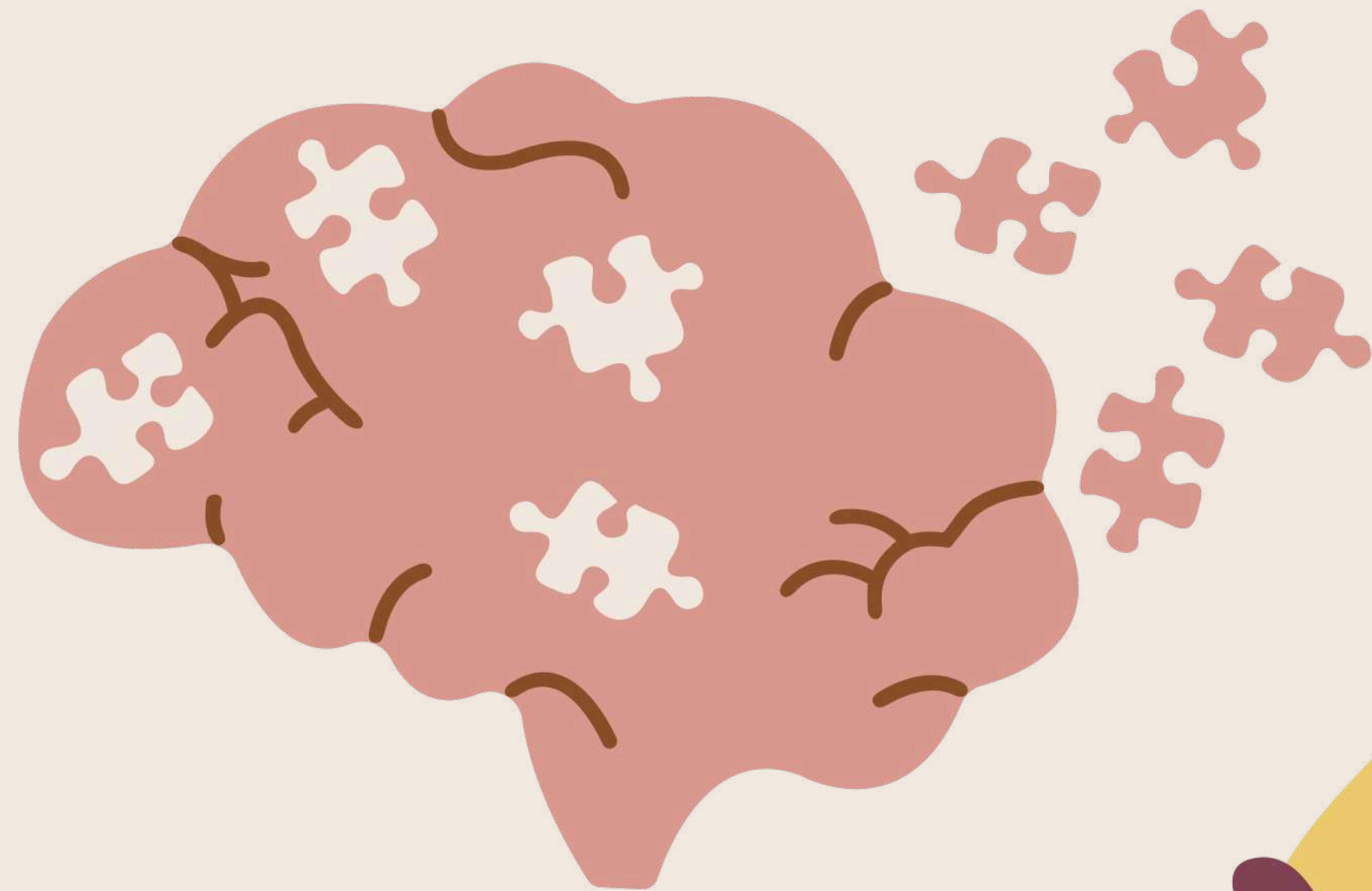
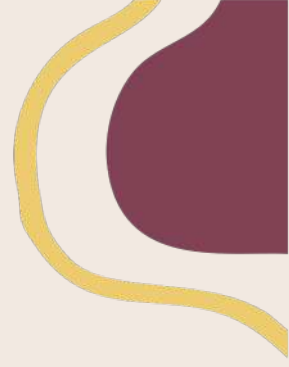
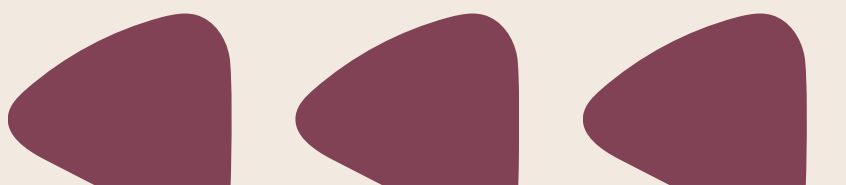


Clinical approach





Objectives

1. History taking in patients with Neurological symptoms
 2. Anatomy of major tracts (e.g. pyramidal tract, spinothalamic tract)
 3. Blood supply of brain and spinal cord.
 4. Signs & symptoms of upper & lower motor neuron lesions
 5. Localization principles of upper motor neuron disorders
 6. Localization principles of lower motor neuron disorders
- 
- 

History taking

As we mention the great Importance of the physical examination in diagnosing neurological symptoms, The history is the key for diagnosing many symptoms as the exam may be normal ,such as headache ,numbness, Loss of consciousness

General approach

Two Key Questions to ask : **what** is the lesion and **where** (in the nervous system) is the lesion?

give additional attention to :

- need of witnesses .
- people with communication difficulties.
- clarification of the patients words .
- fears of and thoughts of patients .

General approach

Time relationships :

onset, duration, pattern over time...

precipitating, exacerbating or relieving factors :

what was the patient doing ?

better or worse ; posture , menstrual cycle ,medication...

Associated symptoms I.e : Headache associated with

1-Nausea, vomiting ,Photophobia may go with migraine

2-neck stiffness,fever and rash more likely to meningitis

common presenting symptoms

Headache :

Migraine ,tension type ..etc.

Transient loss of consciousness(TLOC):
syncope is LOC due to inadequate cerebral perfusion and is the most common cause of (TLOC).

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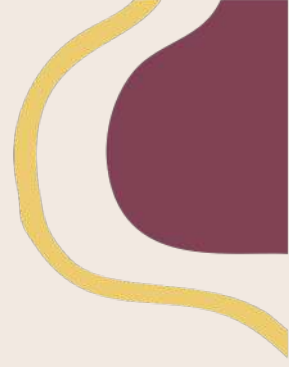
7.1 Clinical characteristics of headache syndromes				
	Onset	Duration/periodicity	Pain location	Associated features
Primary syndromes				
Migraine	Evolves over 30–120 min	Usually last <24 h, recurrent with weeks/months symptom-free	Classically unilateral but may be anywhere including face/neck	Aura (usually visual), nausea/vomiting, photophobia and phonophobia
Cluster headache	Rapid onset, often waking patient from sleep	30–120 min, 1–4 attacks within 24 h, clusters usually last weeks to months, with months to years of remission	Orbital/retro-orbital; always same side during cluster, may switch sides between clusters	Autonomic features, including conjunctival injection, tearing, nasal stuffiness, ptosis, miosis, agitation
Stabbing headache	Abrupt, rarely from sleep	Very brief, seconds or less	Anywhere over head	Common in migraineurs
Secondary syndromes				
Meningitis	Usually evolves over a day or two, can be abrupt	Depends on cause and treatment, usually days to weeks	Global, including neck stiffness	Fever, meningism, rash, false localising signs, signs of raised intracranial pressure
Subarachnoid haemorrhage	Abrupt, immediately maximal, rare from sleep	May be fatal at onset, usually days to weeks	Anywhere, poor localising value	20% isolated headache only; nausea/vomiting, reduced consciousness, false localising signs, III nerve palsies
Temporal arteritis	Gradual onset of temple pain and scalp tenderness	Continuous	Temple and scalp	Usually in those >55 years; unwell, jaw pain on chewing, visual symptoms, tender temporal arteries, elevated erythrocyte sedimentation rate and C-reactive protein

common presenting symptoms

Seizure :

epileptic seizure is caused by paroxysmal electrical discharges, either from the whole brain (**generalized seizure**) or part of the brain (**focal seizure**).

tonic clonic (convulsion) is the most common type of generalized seizure .

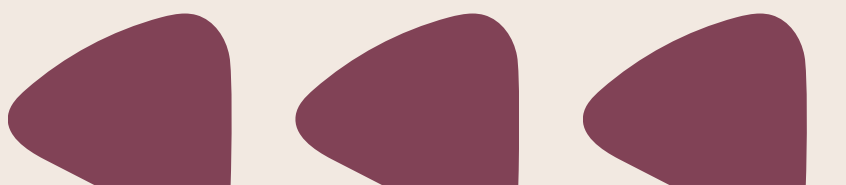


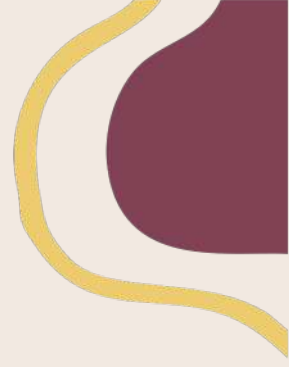
common presenting symptoms

Focal neurological symptoms due to stroke or transient ischemic attack

A stroke is a focal neurological deficit of rapid onset due to a vascular cause. A transient ischemic attack is the same but symptoms resolved within 24 hours.

It demands urgent assessment and treatment.





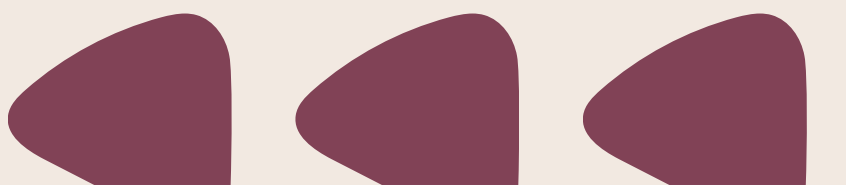
common presenting symptoms

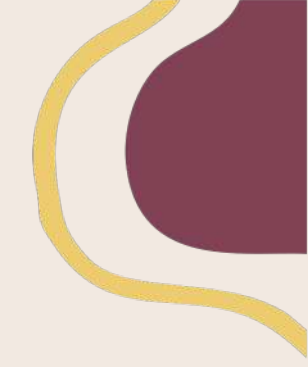
dizziness and vertigo

affects 30% of those over 65 yrs

Common causes are: Postural hypotension,
cerebrovascular disease or cardiac arrhythmia.

functional neurological symptoms.



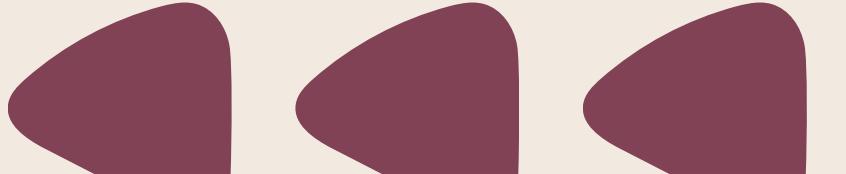


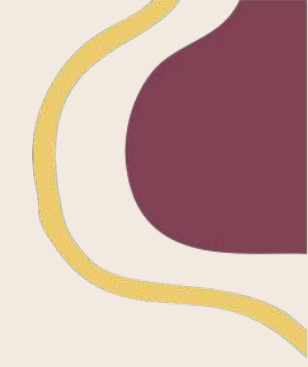
past Medical history :

any previous symptoms could be important ,birth history ,vascular diseases, and Imp. risk factors .

DRUG history:

always inquire about drugs ,vaccinations ,and the absence of vaccinations (polio,measles)



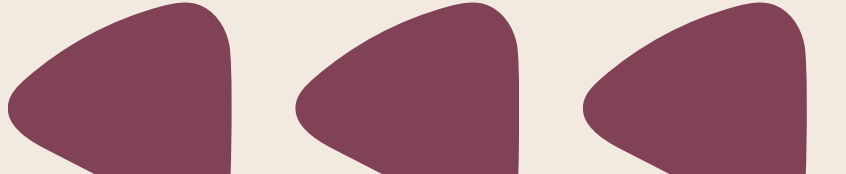


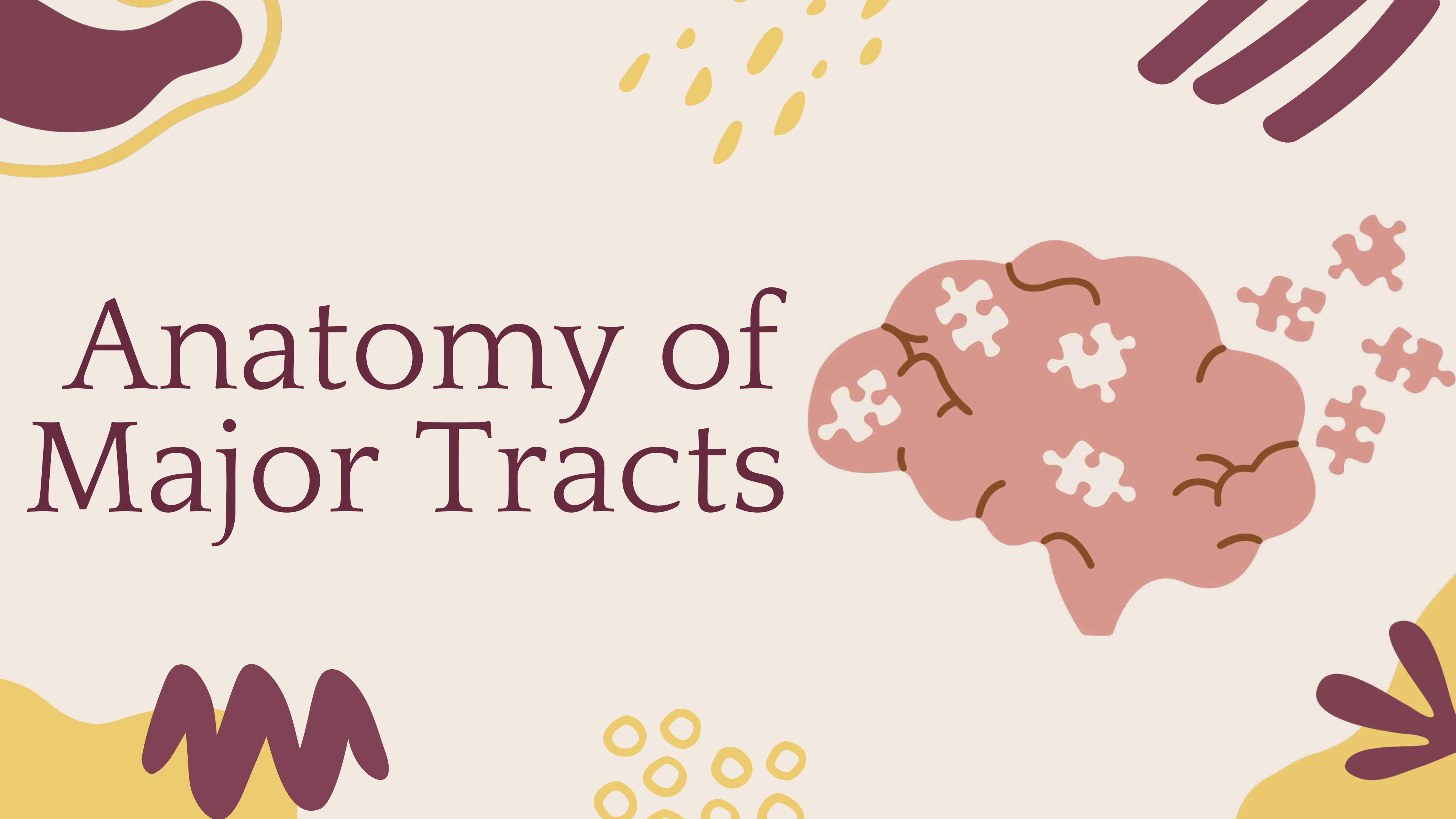
Social history:

Social circumstances are relevant, alcohol is the most common neurological toxin, poor diet, B12 deficiency, sexually transmitted diseases (HIV, syphilis).

occupational history:

occupational factors are relevant to several neurological disorders i.e. Toxic peripheral neuropathy (lead and heavy metals), manganese cause Parkinsonism. affects pt occupation.





Anatomy of Major Tracts

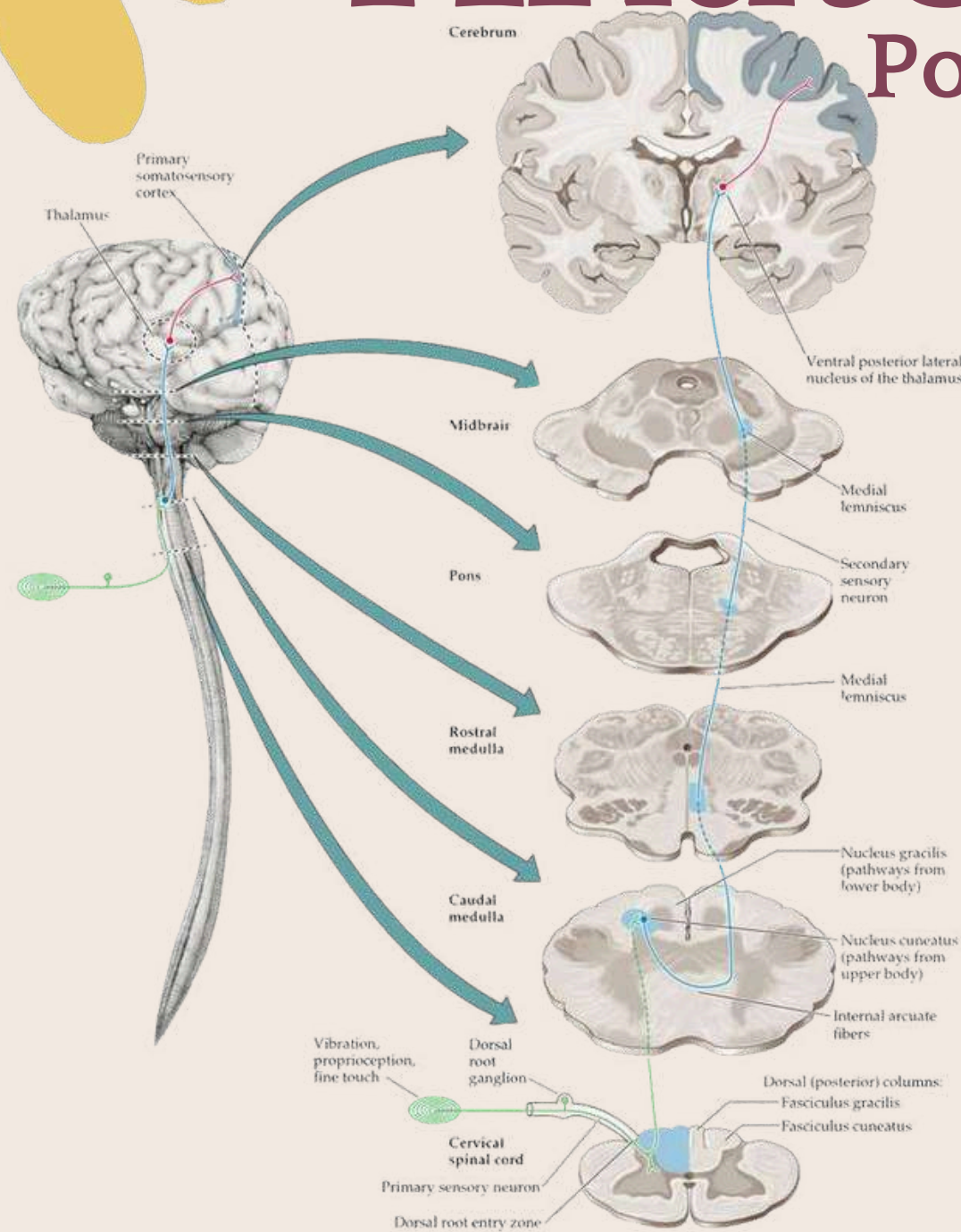
Anatomy of major tracts

Main Somatosensory Pathways:

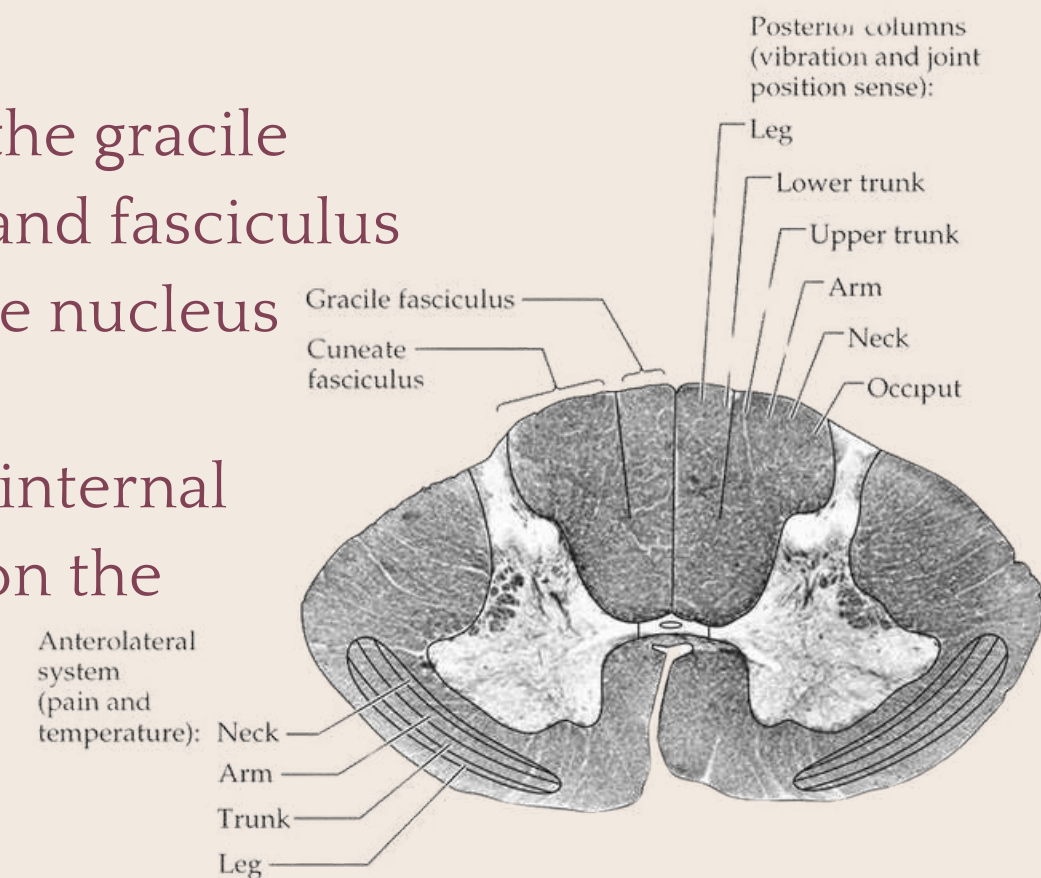
- The posterior column–medial lemniscal pathway conveys proprioception, vibration sense, and fine, discriminative touch.
- The anterolateral pathways include the spinothalamic tract and other associated tracts that convey pain, temperature sense, and crude touch

Anatomy of major tracts

Posterior Column-Medial Lemniscal Pathway:



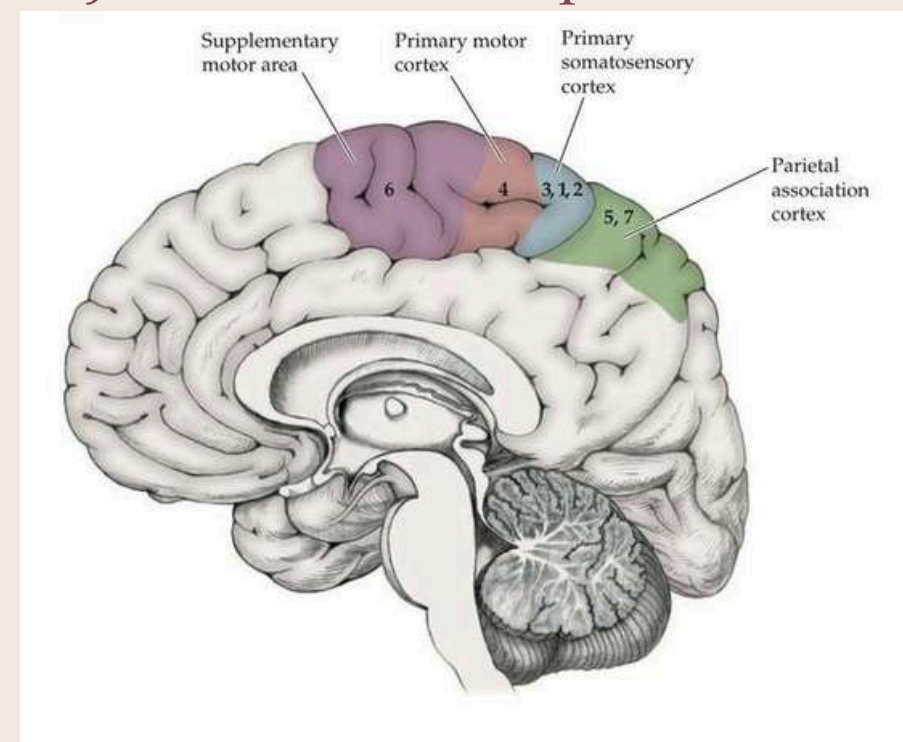
- Large-diameter, myelinated axons carrying information about proprioception, vibration sense, and fine touch enter the spinal cord via the medial portion of the dorsal root entry zone.
- Many of these axons then enter the ipsilateral posterior columns to ascend all the way to the posterior column nuclei in the medulla.
- the **somatotopic** organization of the posterior columns:
- the medial portion, called the gracile fasciculus carries information from the legs and lower trunk. The more lateral cuneate fasciculus carries information from the upper trunk above about T6, and from the arms and neck
- The first-order sensory neurons that have axons in the gracile and cuneate fasciculi (also called fasciculus gracilis and fasciculus cuneatus) synapse onto second-order neurons in the nucleus gracilis and nucleus cuneatus, respectively.
- Axons of these **second-order neurons** decussate as internal arcuate fibers and then form the medial lemniscus on the other side of the medulla



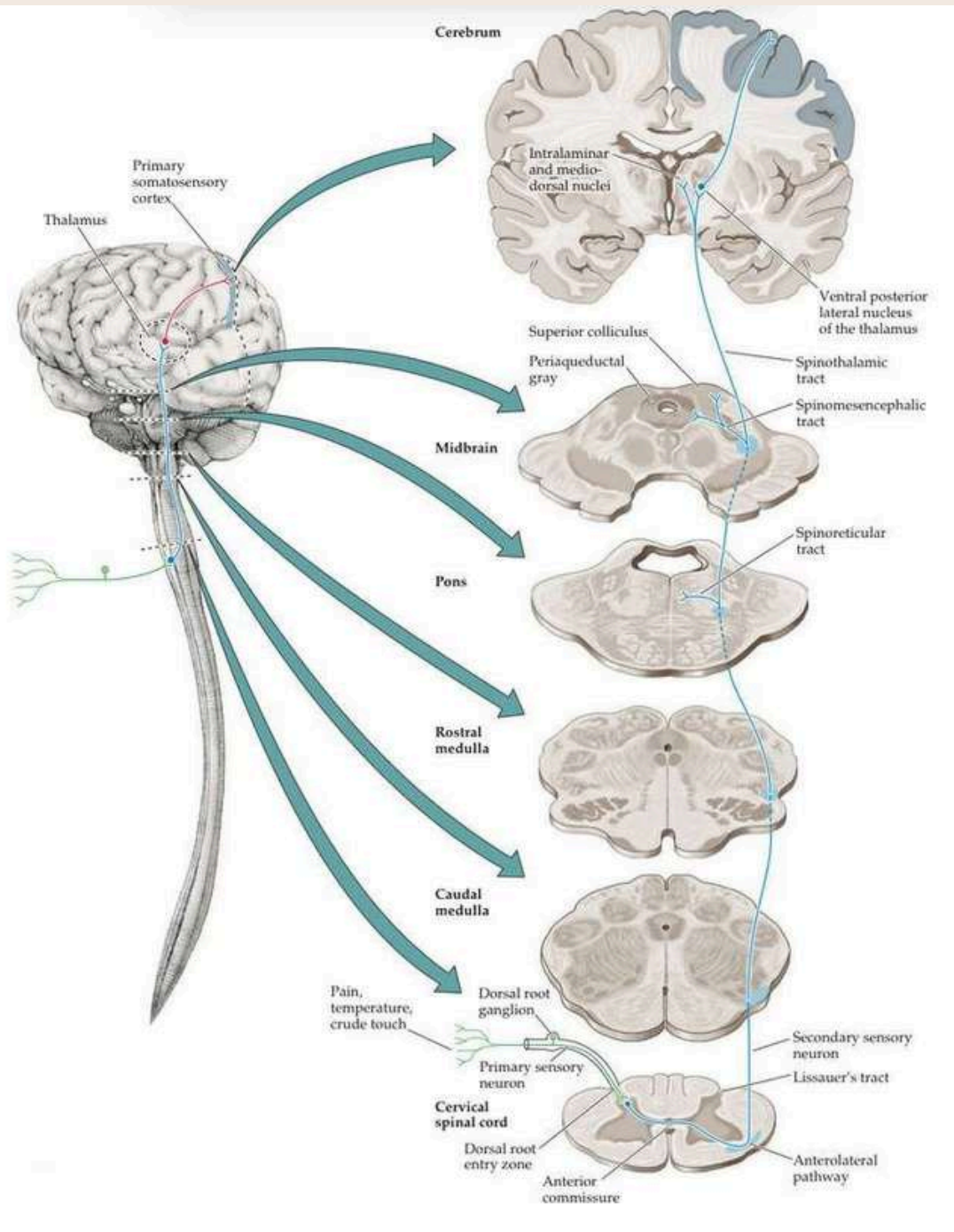
Anatomy of major tracts

Posterior Column–Medial Lemniscal Pathway:

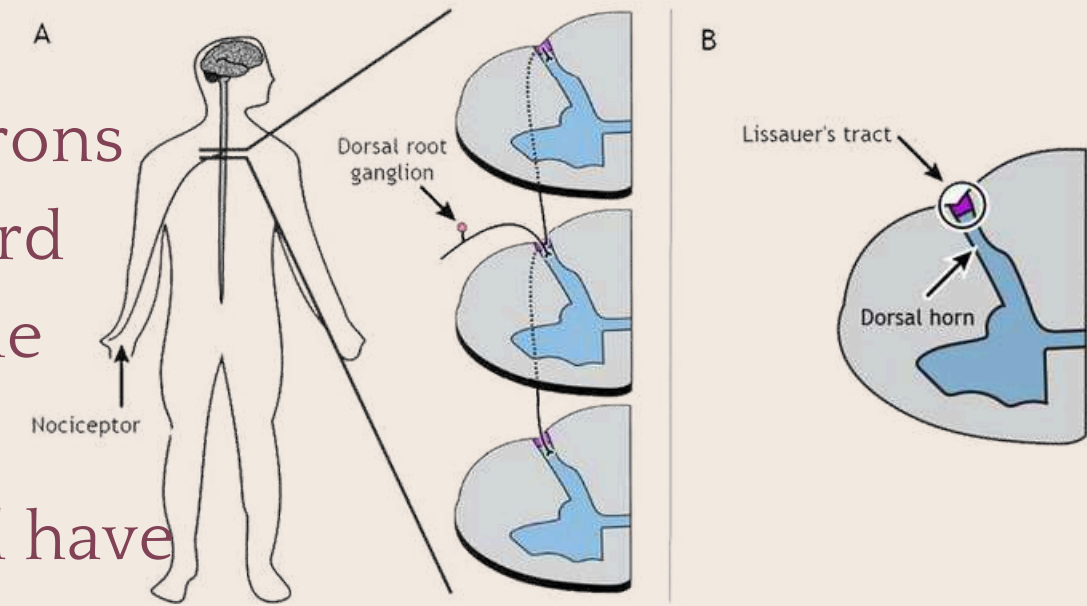
- The medial lemniscus initially has a vertical orientation and then comes to occupy a progressively more lateral and inclined position as it ascends in the brainstem.
- The somatotopic organization of the medial lemniscus assumes a vertical position in the medulla such that the feet are represented more ventrally (“the little person stands up”) and then inclines again in the pons and midbrain such that the arms are represented more medially and legs more laterally (“the little person lies down”)
- **Note:**the medial lemniscus in the pons and midbrain the feet are lateral, while in the posterior columns the feet are medial.
- The next major synapse occurs when the medial lemniscus axons terminate in the ventral posterior lateral nucleus (VPL) of the thalamus.
- The neurons of the VPL then project through the posterior limb of the internal capsule in the thalamic somatosensory radiations to reach the primary somatosensory cortex in the postcentral gyrus



anterolateral pathways:



- Smaller-diameter and unmyelinated axons carrying information about pain and temperature sense also enter the spinal cord via the dorsal root entry zone
 - these axons make their first synapses immediately in the gray matter of the spinal cord, mainly in the dorsal horn marginal zone (lamina I) and deeper in the dorsal horn, in lamina V .
 - Some axon collaterals ascend or descend for a few segments in Lissauer's tract before entering the central gray.
 - Axons from the second-order sensory neurons in the central gray cross over in the spinal cord anterior (ventral) commissure to ascend in the anterolateral white matter.
 - The anterolateral pathways in the spinal cord have a somatotopic organization in which the feet are most laterally represented.
-
- The diagram consists of two parts, A and B. Part A shows a human figure from the back, with a line indicating the pathway of sensory information from the hand (labeled 'Nociceptor') through the 'Dorsal root ganglion' and into the spinal cord. Part B is a magnified view of the dorsal horn, showing the entry of the axon into the dorsal horn and its potential ascent or descent in Lissauer's tract before synapsing.



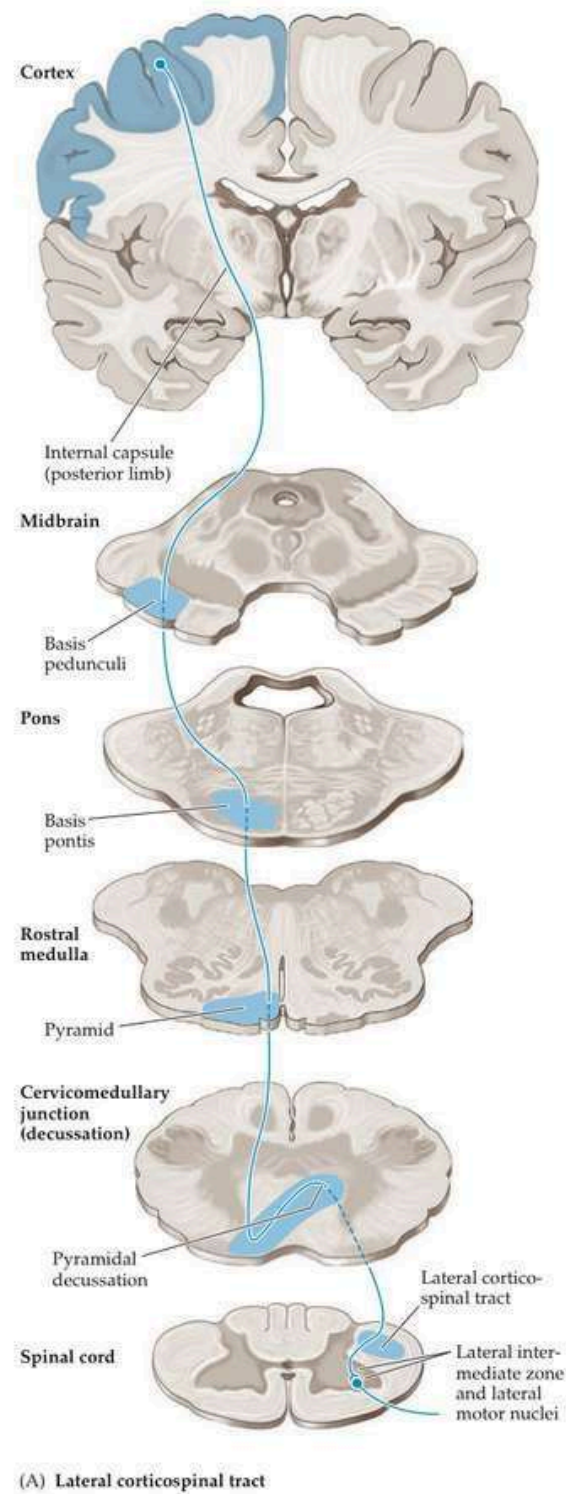
Anatomy of major tracts

anterolateral pathways:

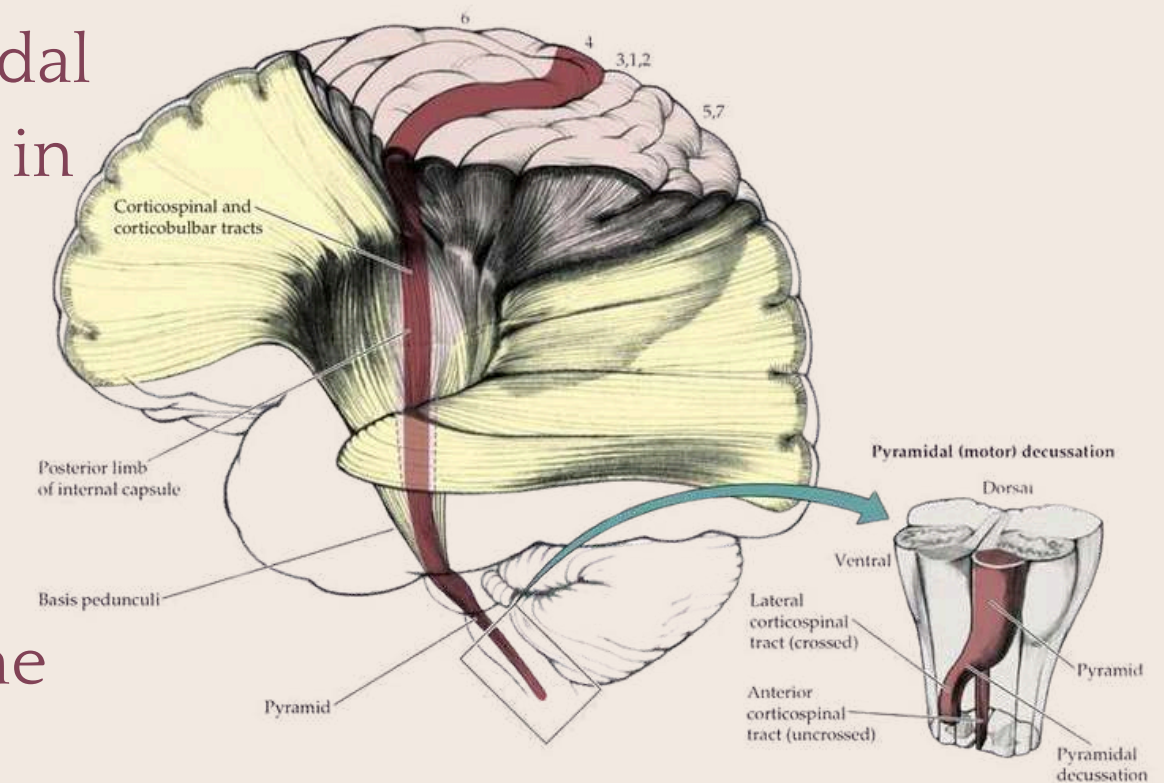
- When they reach the medulla, the anterolateral pathways are located laterally, running in the groove between the inferior olives and the inferior cerebellar peduncles.
- They then enter the pontine tegmentum to lie just lateral to the medial lemniscus in the pons and midbrain .
- Like the posterior column–medial lemniscal pathway, a major relay for the spinothalamic tract is in the ventral posterior lateral nucleus (VPL) of the thalamus .
- However, the terminations of the spinothalamic tract and the posterior column–medial lemniscal pathway reach separate neurons within the VPL
- From the VPL, information travelling in the spinothalamic tract is again conveyed via the thalamic somatosensory radiations to the primary somatosensory cortex in the post- central gyrus.

Anatomy of major tracts

Corticospinal Tract:



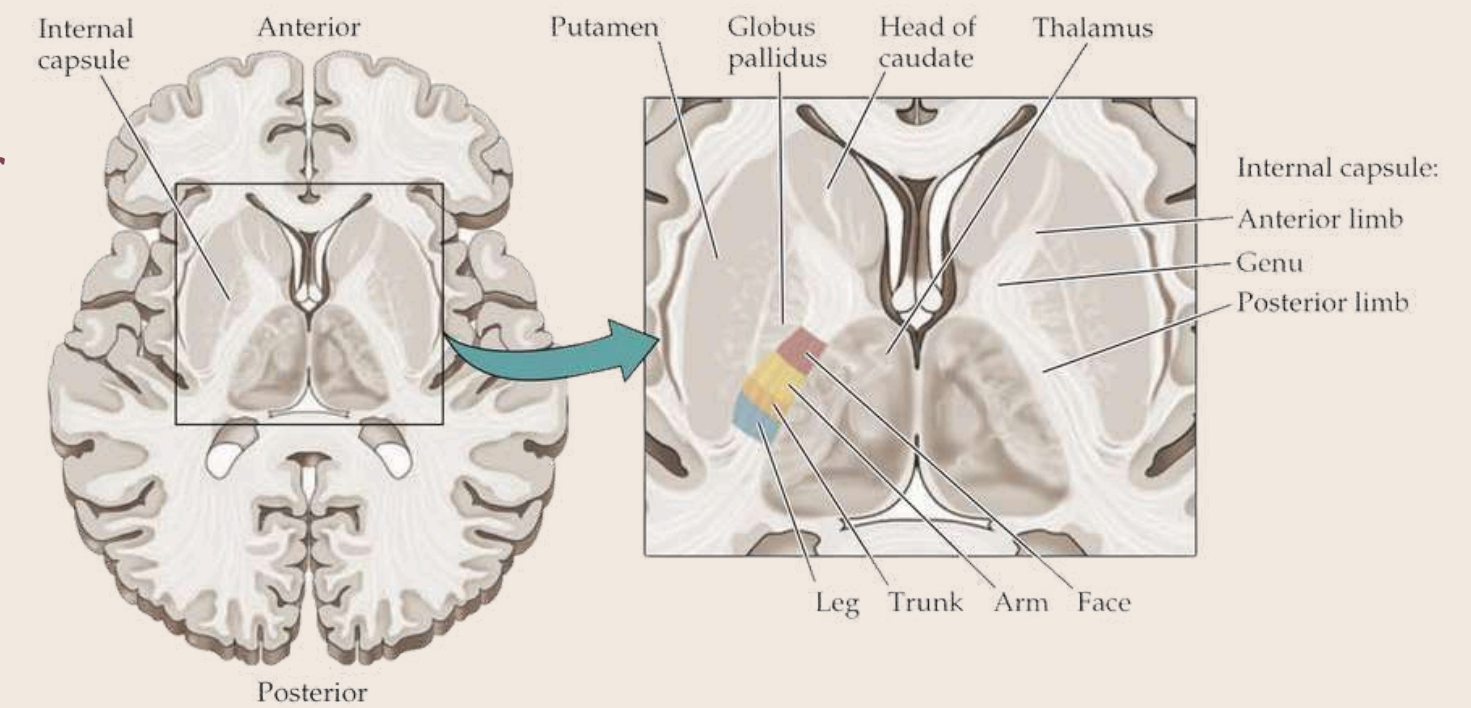
- the lateral corticospinal tract is the most clinically important descending motor pathway in the nervous system.
- Over half of the corticospinal tract fibers originate in the primary motor cortex (Brodmann's area 4) of the precentral gyrus. The remainder arise from the premotor and supplementary motor areas (area 6) or from the parietal lobe (areas 3, 1, 2, 5, and 7)
- About 3% of corticospinal neurons are giant pyramidal cells called Betz cells, which are the largest neurons in the human nervous system.
- Axons from the cerebral cortex enter the upper portions of the cerebral white matter, or corona radiata, and descend toward the internal capsule.
- The corticospinal tract lies in the posterior limb of the internal capsule



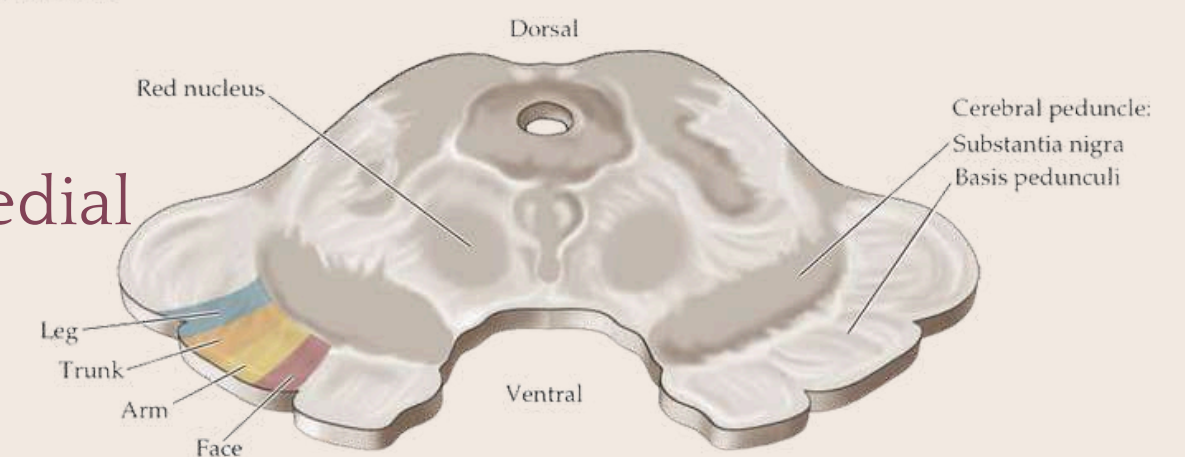
Anatomy of major tracts

Corticospinal Tract:

- The **somatotopic map** is preserved in the internal capsule, so motor fibers for the face are most anterior, and those for the arm and leg are progressively more posterior
- Fibers projecting from the cortex to the brainstem, including motor fibers for the face, are called corticobulbar instead of corticospinal because they project from the cortex to the brainstem,
- The internal capsule continues into the midbrain cerebral peduncles, meaning, “feet of the brain”
- The white matter is located in the ventral portion of the cerebral peduncles and is called the basis pedunculi.
- The middle one-third of the basis pedunculi contains corticobulbar and corti- cospinal fibers with the face, arm, and leg axons arranged from medial to lat- eral, respectively



(B) Midbrain



Anatomy of major tracts

Corticospinal Tract:

- The corticospinal tract fibers next descend through the ventral pons, where they form somewhat scattered fascicles. These collect on the ventral surface of the medulla to form the medullary pyramids. For this reason the corticospinal tract is sometimes referred to as the pyramidal tract.
- The transition from medulla to spinal cord is called the cervicomedullary junction, which occurs at the level of the foramen magnum. At this point about 85% of the pyramidal tract fibers cross over in the pyramidal decussation to enter the lateral white matter columns of the spinal cord, forming the lateral corticospinal tract.
- A somatotopic representation is present in the lateral corticospinal tract, with fibers that control the upper extremity located medial to those that control the lower extremity.

(C) Spinal cord

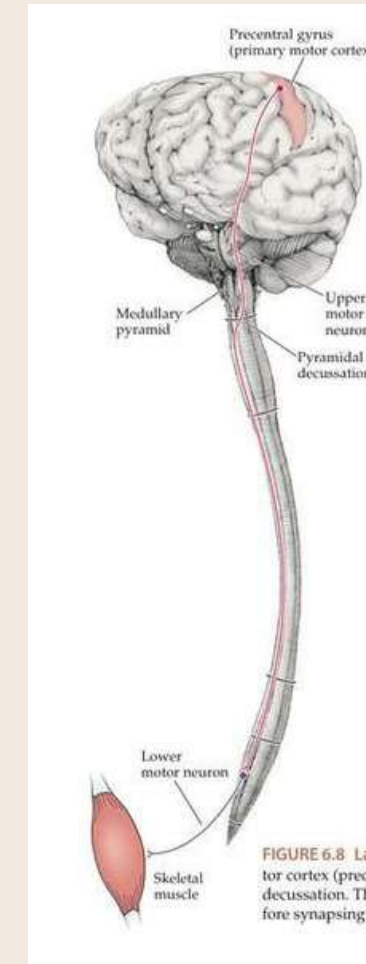
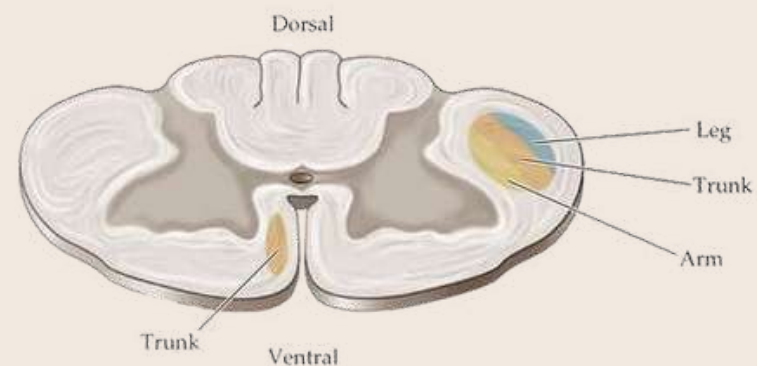
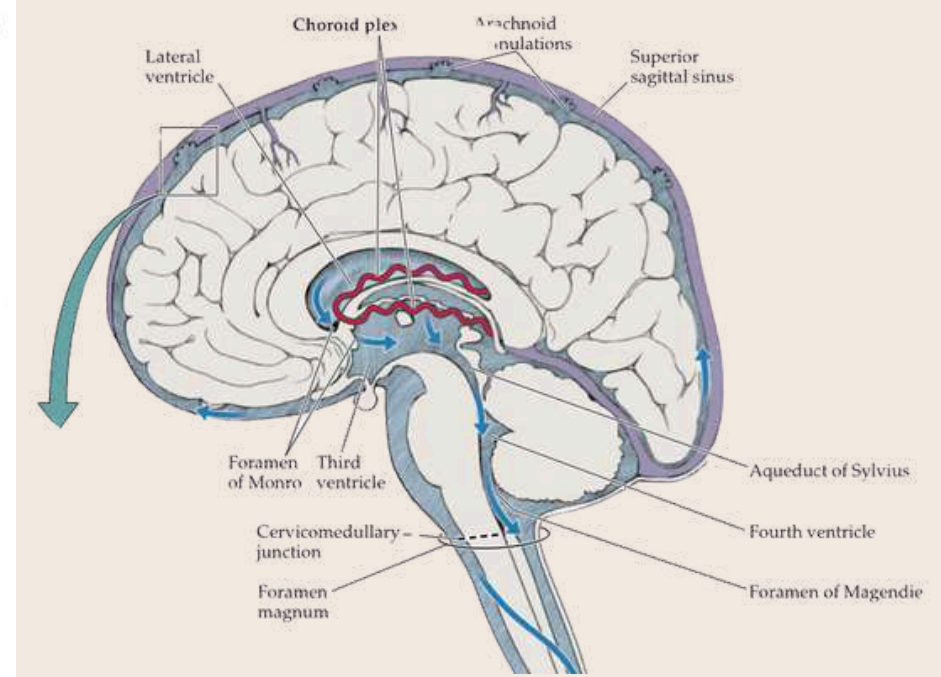


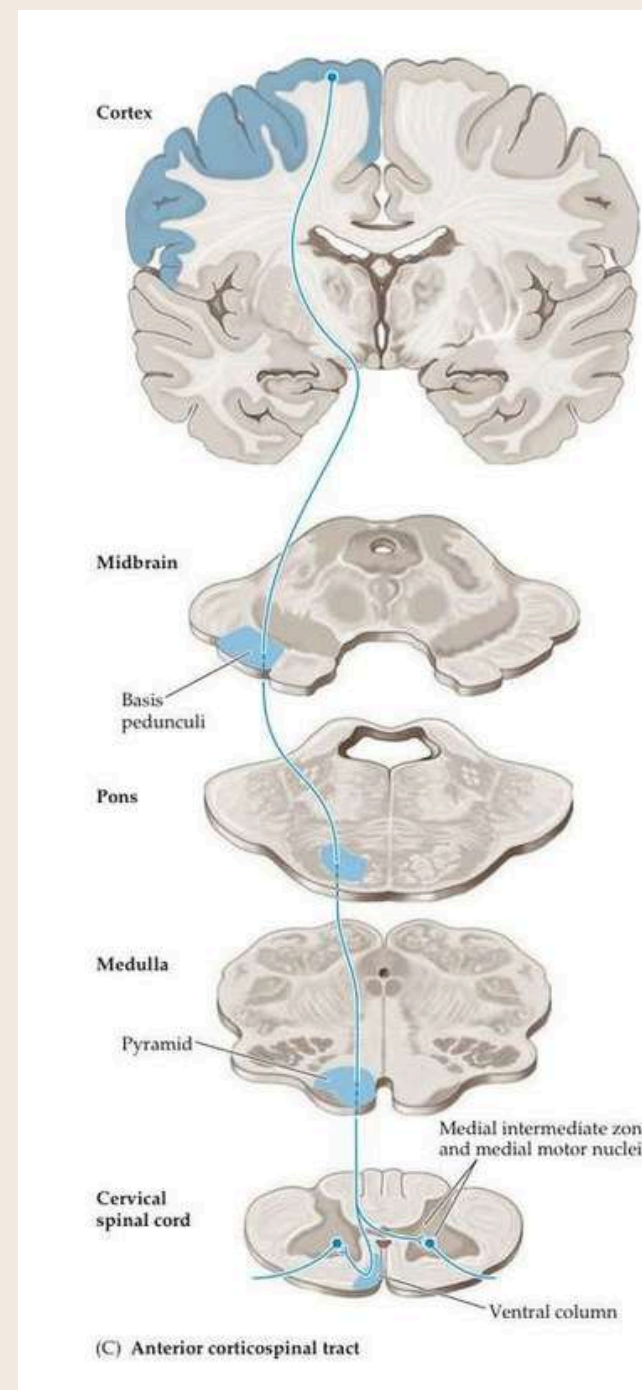
FIGURE 6.8 Lateral corticospinal tract (pyramidal tract). The axons of the upper motor neurons cross over in the pyramidal decussation. The axons of the lower motor neurons cross over in the pyramidal decussation. The axons of the lower motor neurons cross over in the pyramidal decussation.



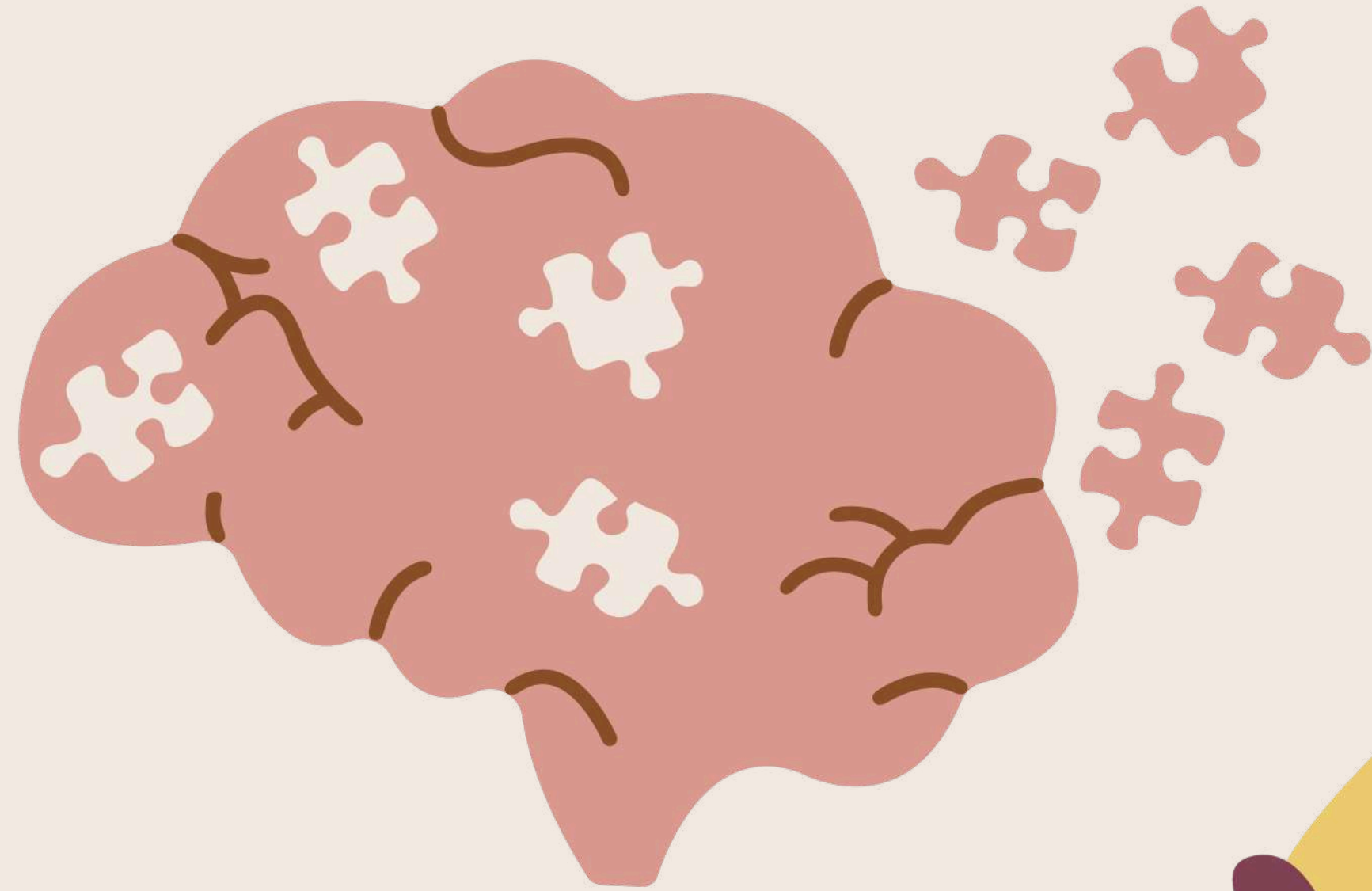
Anatomy of major tracts

Corticospinal Tract:

- Finally, the axons of the lateral corticospinal tract enter the spinal cord central gray matter to synapse onto anterior horn cells .
- The remaining ~15% of corticospinal fibers continue into the spinal cord ipsilaterally, without cross- ing, and enter the anterior white matter columns to form the anterior corti- cospinal tract



Blood supply of brain and spinal cord

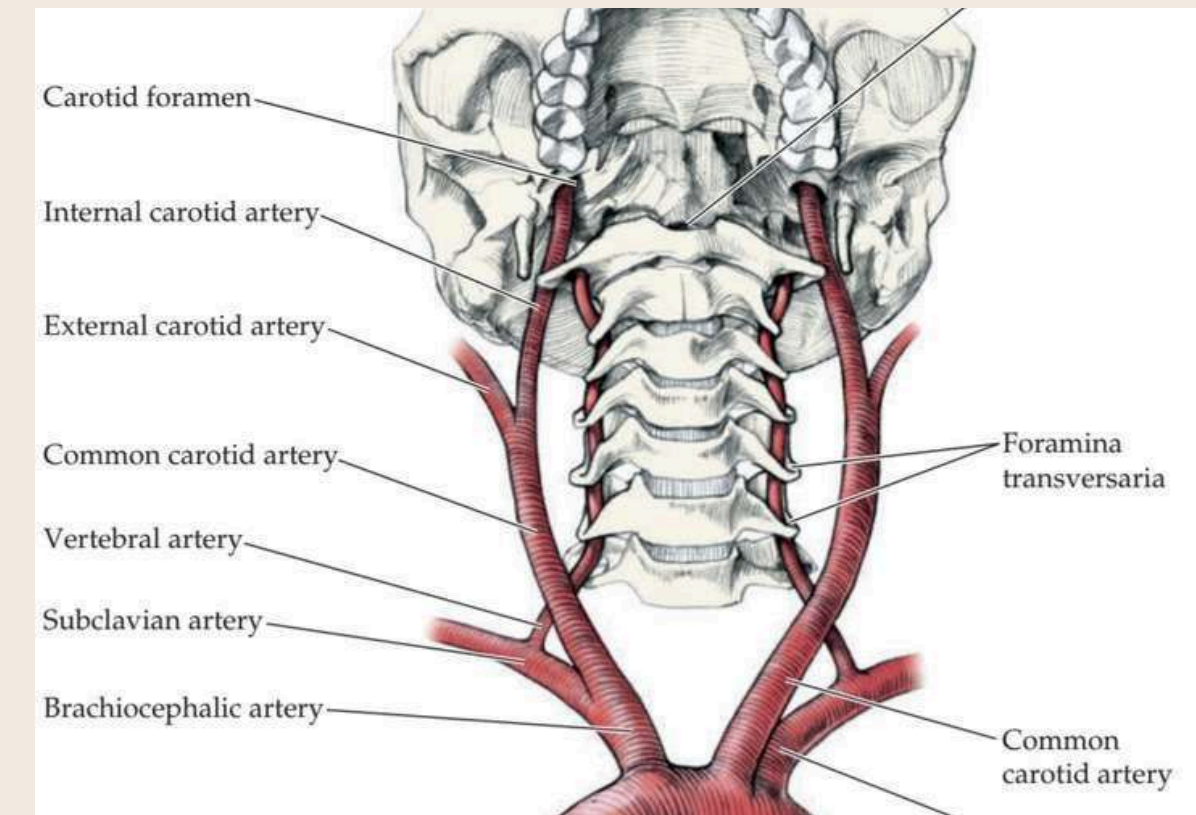


Blood supply of brain



Circle of Willis

The arterial supply to the cerebral hemispheres is derived from the anterior circulation provided by the bilaterally paired internal carotid arteries, as well as by the posterior circulation provided by the bilateral vertebral arteries and joining to form the basilar artery. These anterior and posterior circulations meet in an anastomotic ring called the circle of Willis,

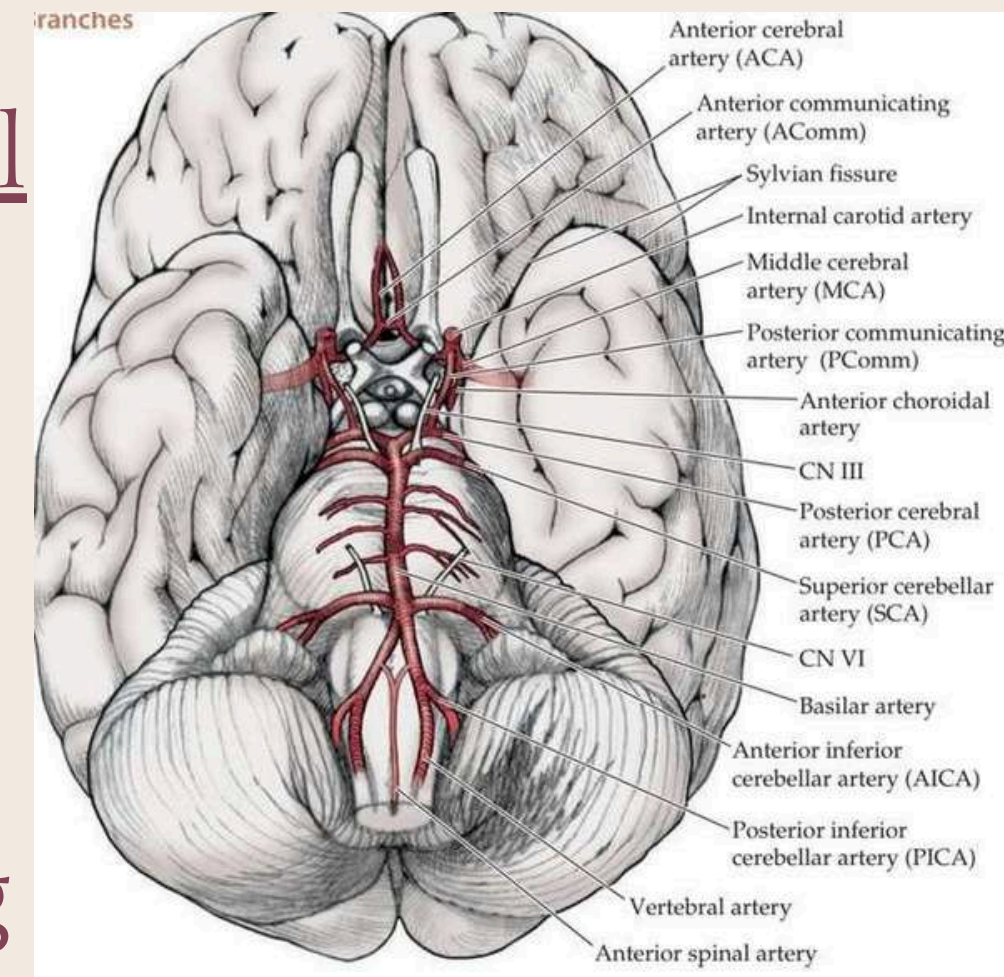


Blood supply of brain



The main arteries supplying the cerebral hemispheres are the anterior, middle, and posterior cerebral arteries. The anterior cerebral arteries (ACAs) and middle cerebral arteries (MCAs) are the terminal branches of the internal carotid arteries The anterior cerebral arteries anastomose anteriorly at the anterior communicating artery (AComm). The anterior and posterior circulations are linked to each other via the posterior communicating arteries (PComms).

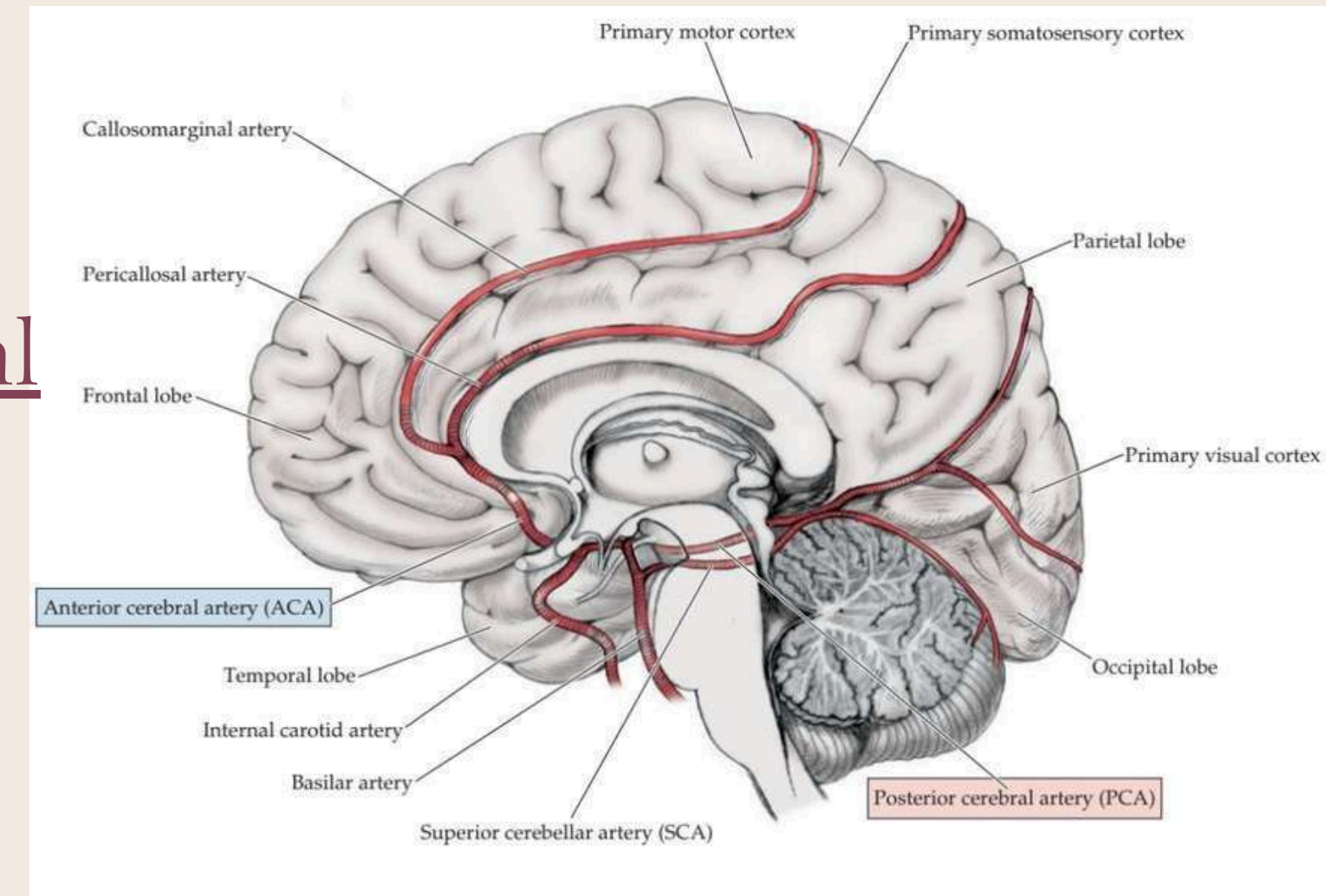
The posterior cerebral arteries (PCAs) arise from the top of the basilar artery



Blood supply of brain



anterior cerebral arteries :
passes forward to travel in the interhemispheric fissure as it sweeps back and over the corpus callosum. Two major branches commonly seen are the pericallosal and callosomarginal arteries. The anterior cerebral artery thus supplies **most of the cortex on the anterior medial surface of the brain, from the frontal to the anterior parietal lobes, usually including the medial sensorimotor cortex**



Blood supply of brain

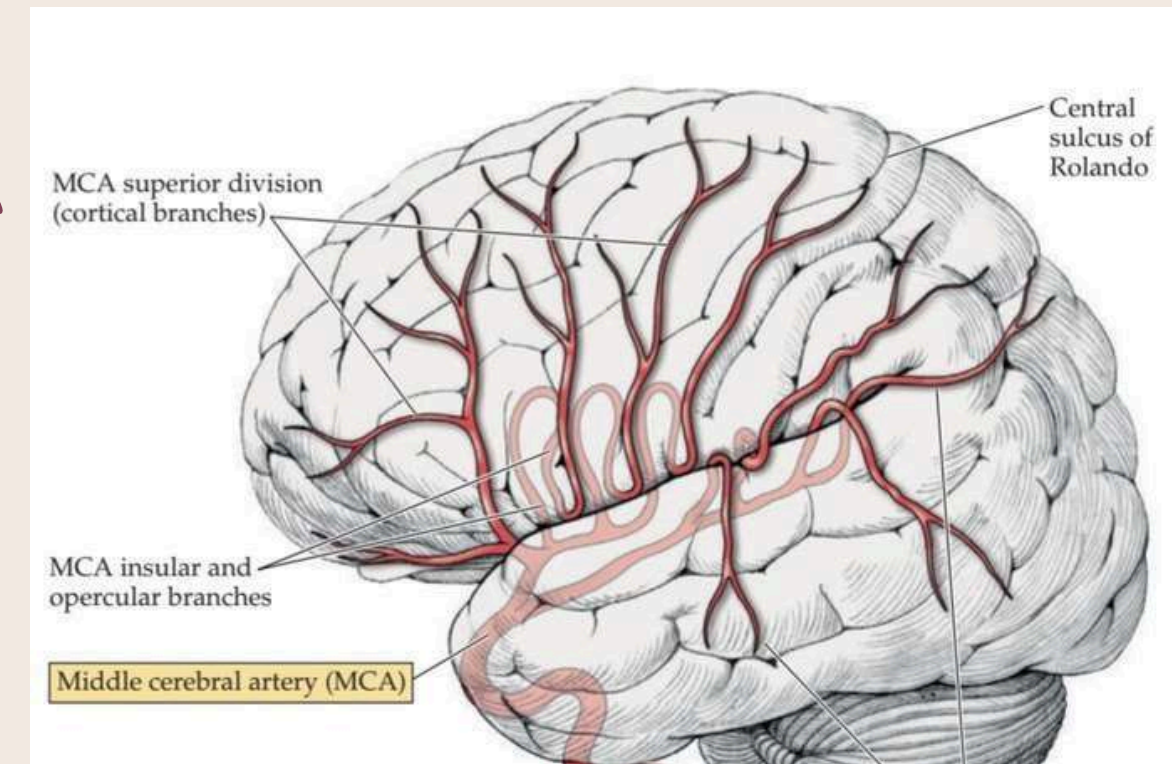


middle cerebral artery :

turns laterally to enter the depths of the Sylvian fissure. Within the Sylvian fissure it usually

bifurcates into the superior division and the inferior division.

- The superior division supplies the cortex above the Sylvian fissure, including the lateral frontal lobe and usually including the peri-Rolandic cortex
- The inferior division supplies the cortex below the Sylvian fissure, including the lateral temporal lobe and a variable portion of the parietal lobe



Blood supply of brain

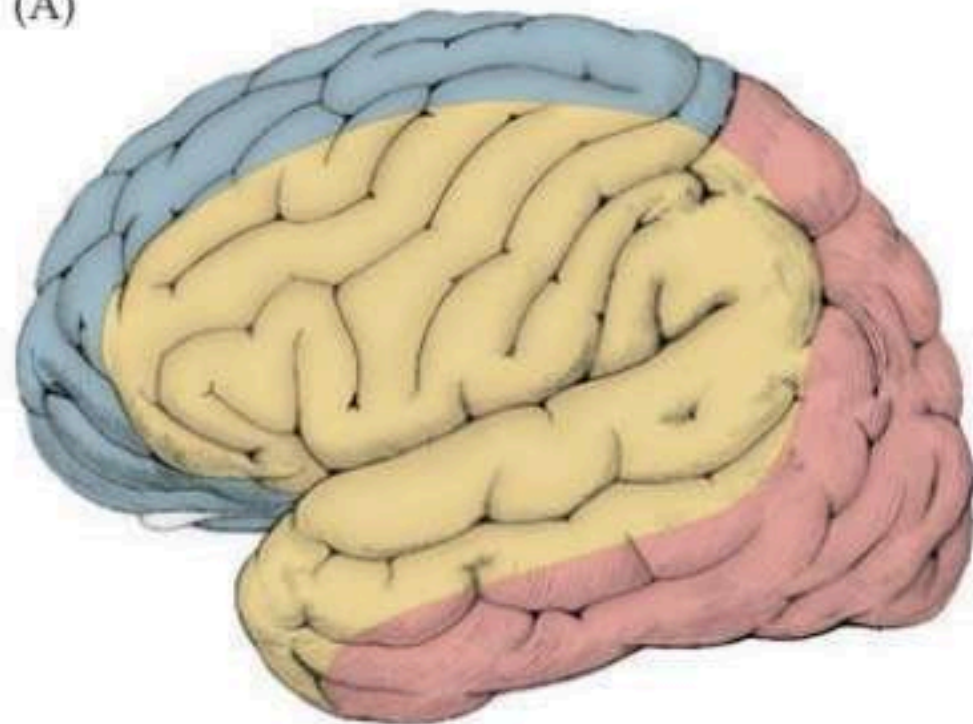


posterior cerebral artery :

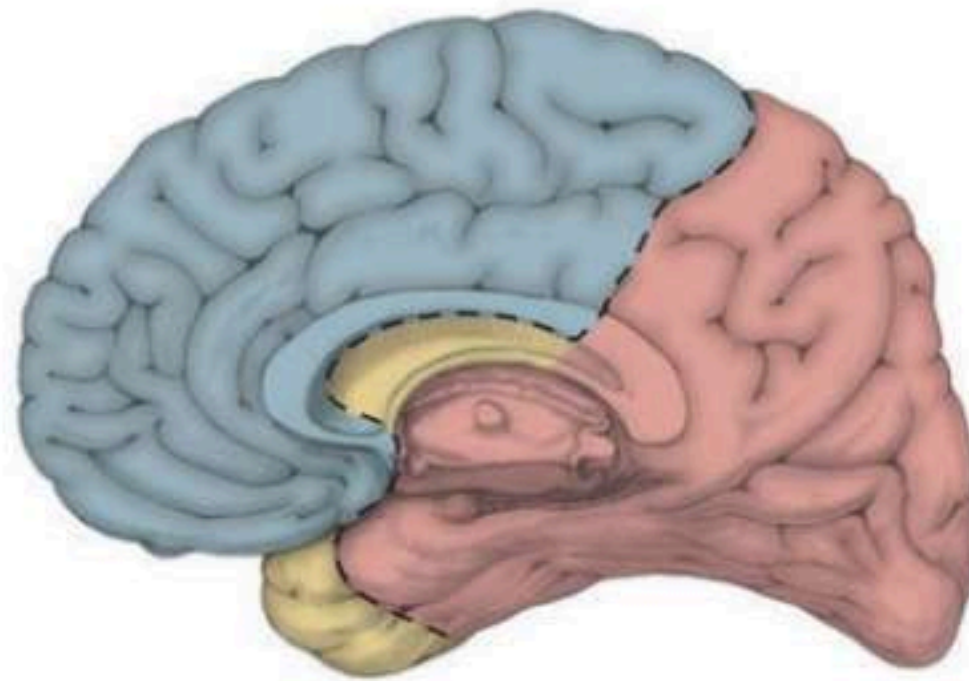
curves back after arising from the top of the basilar and sends branches over the inferior and medial temporal lobes and over the medial occipital cortex. The posterior cerebral artery territory therefore includes the inferior and medial temporal and occipital cortex

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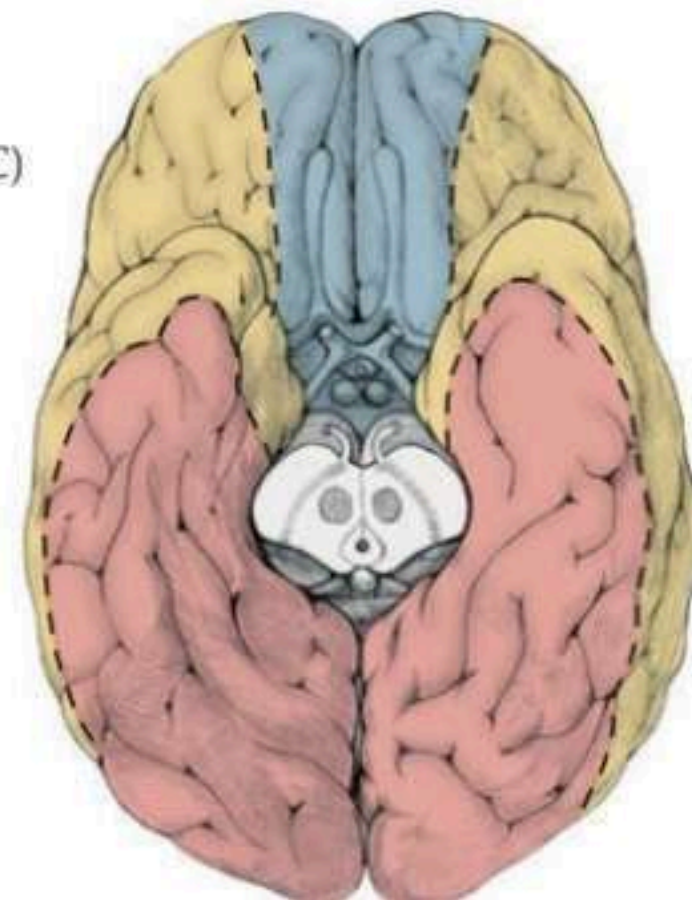
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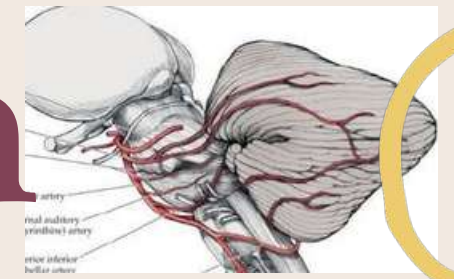
(B)



(C)

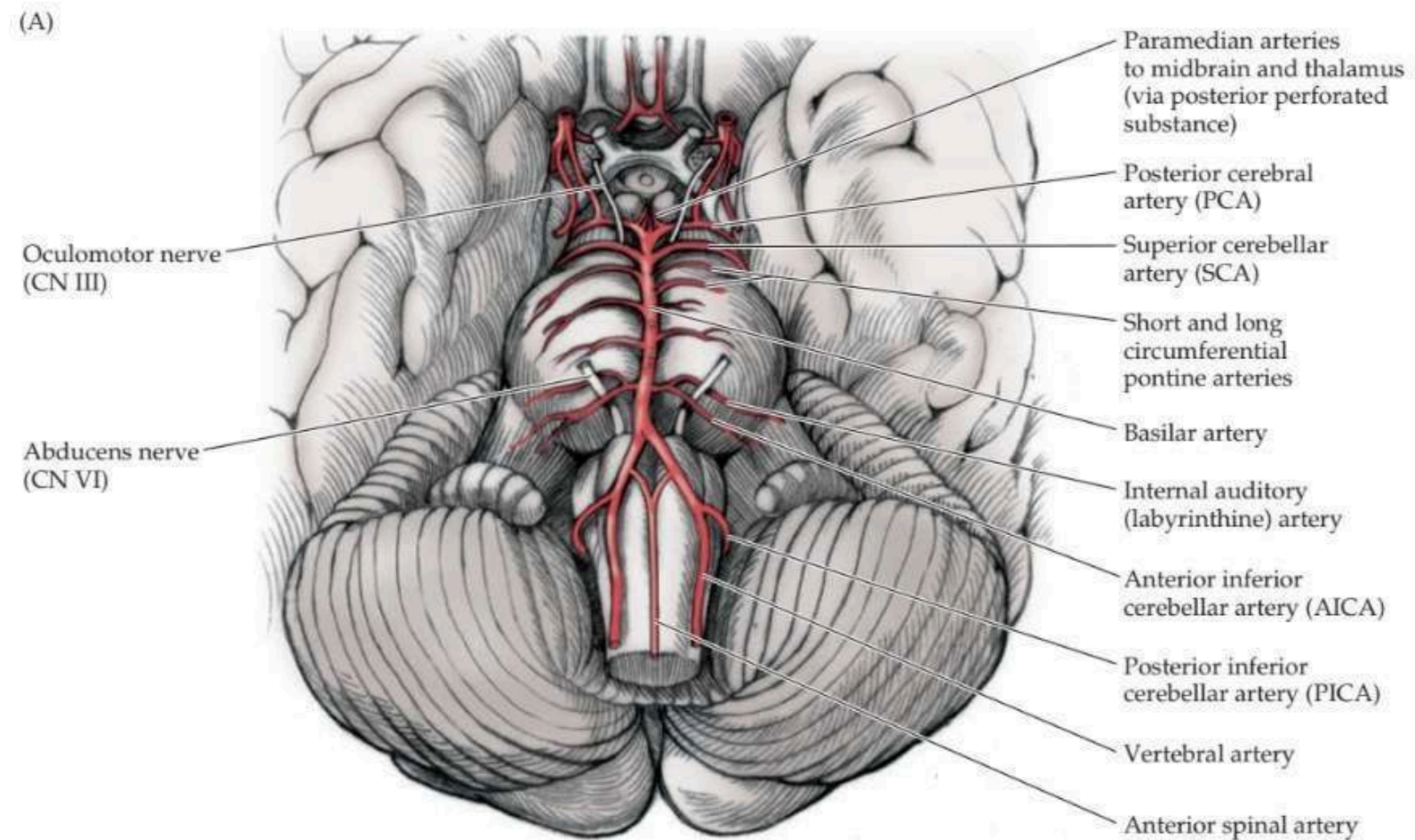
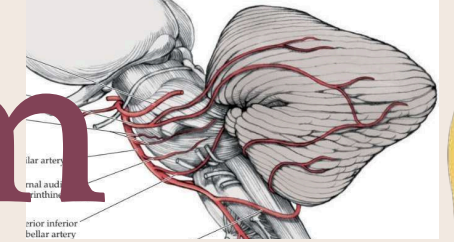


Blood supply of brainstem

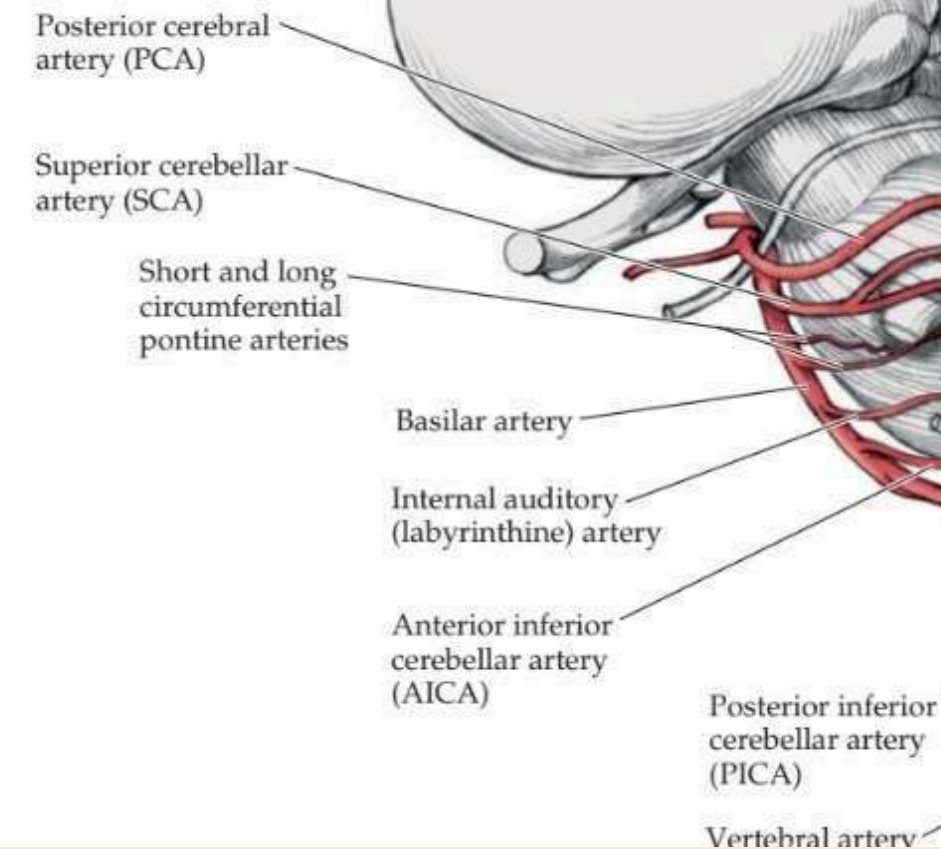


The vertebrobasilar system gives rise to multiple branches to provide the blood supply to the brainstem and cerebellum. In addition, most parts of the thalamus, as well as the inferior-medial occipital and temporal lobes, are supplied by the posterior cerebral arteries arising from the top of the basilar artery. The largest branches of the vertebrobasilar system are the posterior inferior cerebellar artery (PICA), anterior inferior cerebellar artery (AICA), superior cerebellar artery (SCA), and posterior cerebral artery (PCA)

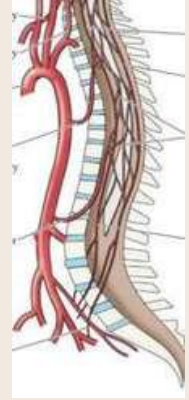
Blood supply of brainstem



(B)



Blood supply of spinal cord



The blood supply to the spinal cord arises from branches of the vertebral arteries and spinal radicular arteries.

1. The **vertebral arteries** give rise to the **anterior spinal artery** that runs along the ventral surface of the spinal cord (**supplies approximately the anterior two-thirds of the cord**) In addition, two **posterior spinal arteries** arise from the **vertebral** or **posterior inferior cerebellar arteries** and supply the dorsal surface of the cord
2. Thirty-one segmental arterial branches enter the spinal canal along its length; most of the branches arise from the aorta and supply the meninges. **Only six to ten of these reach the spinal cord as radicular arteries, arising at variable levels**

Blood supply of spinal cord

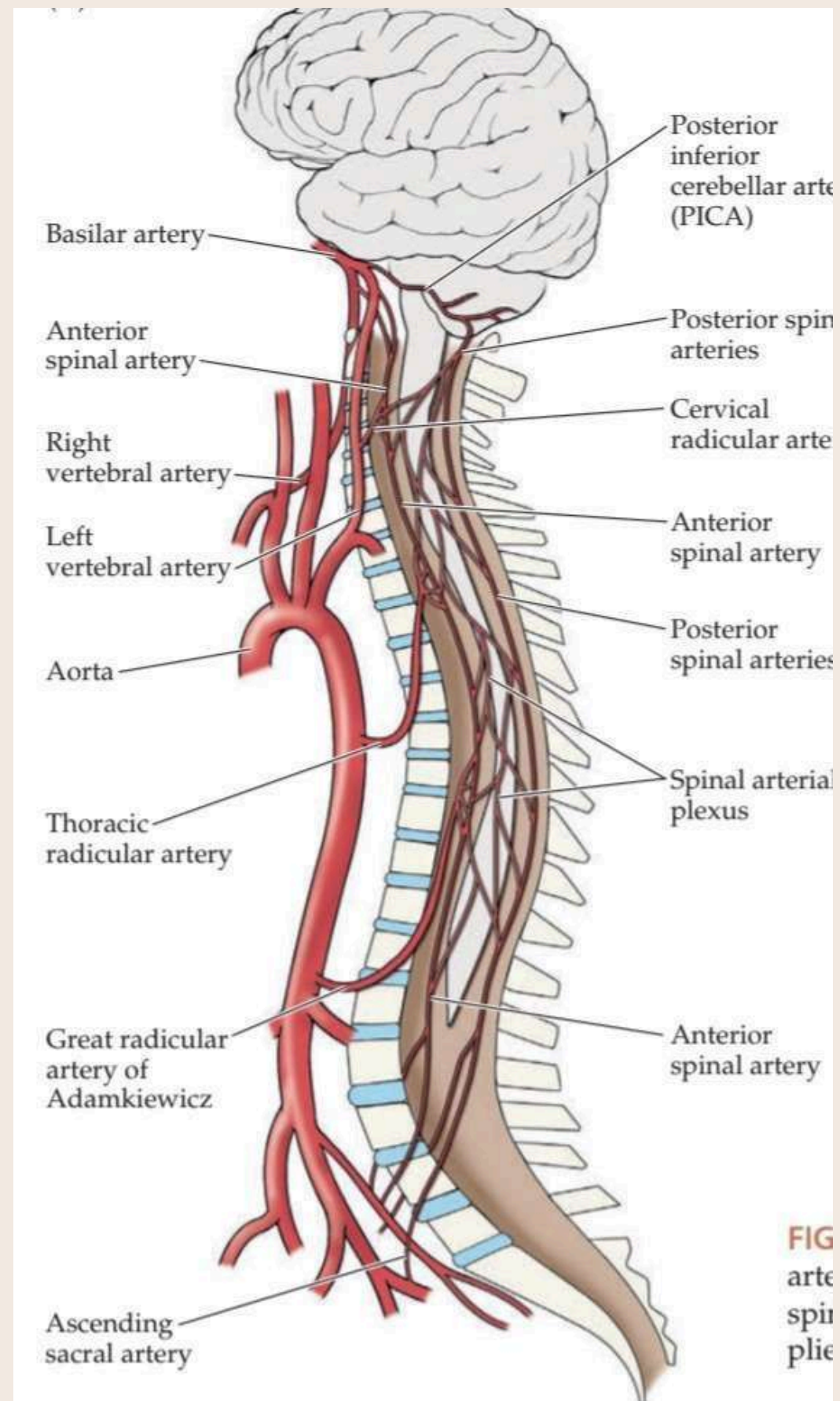
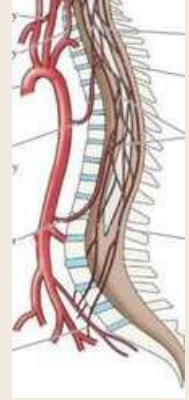
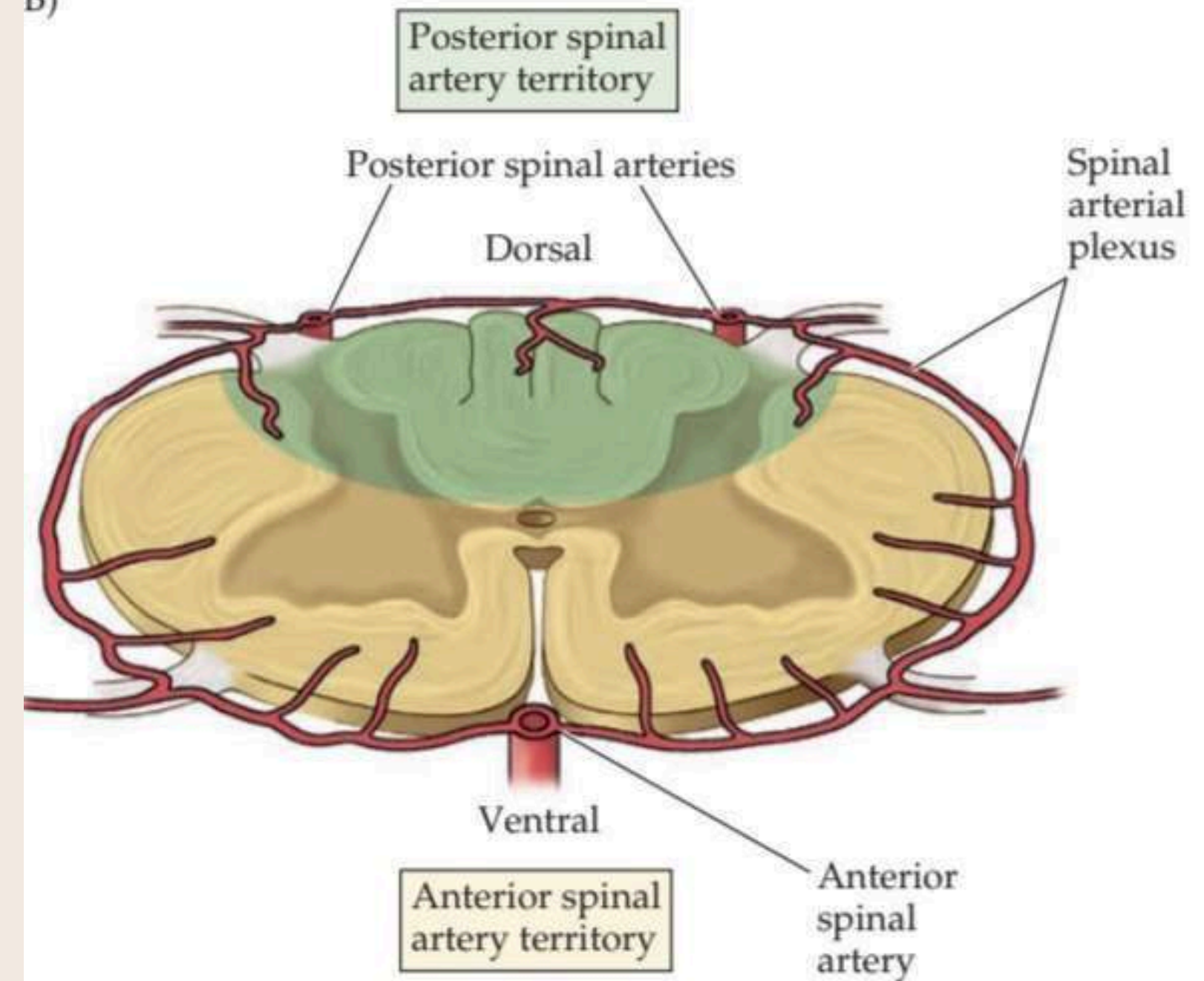
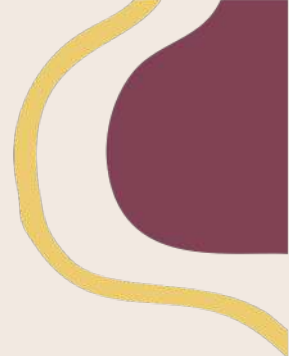


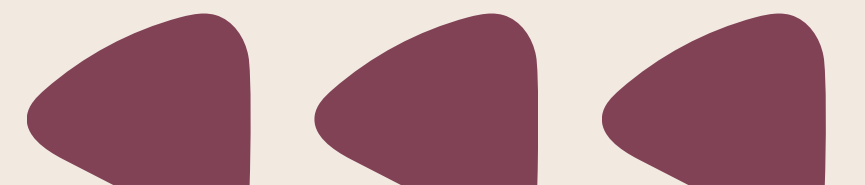
FIG
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B)



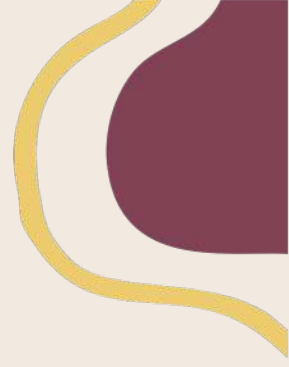


Upper and lower neuron lesions



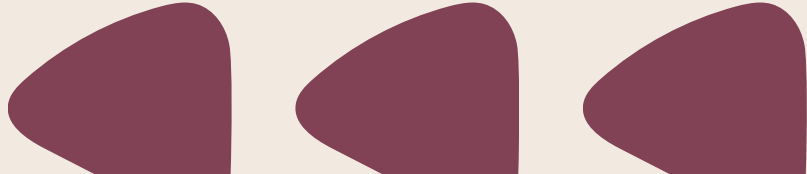


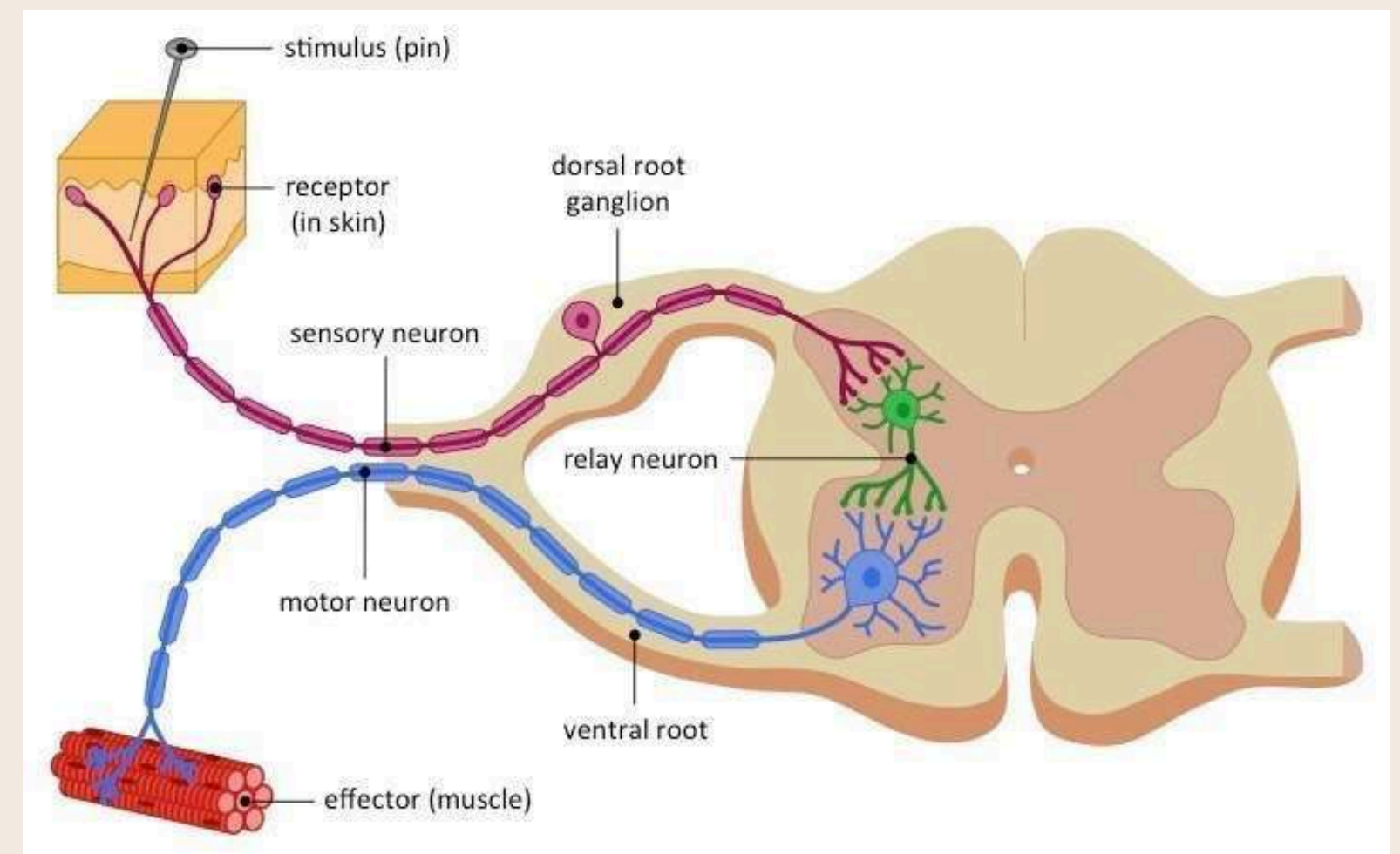
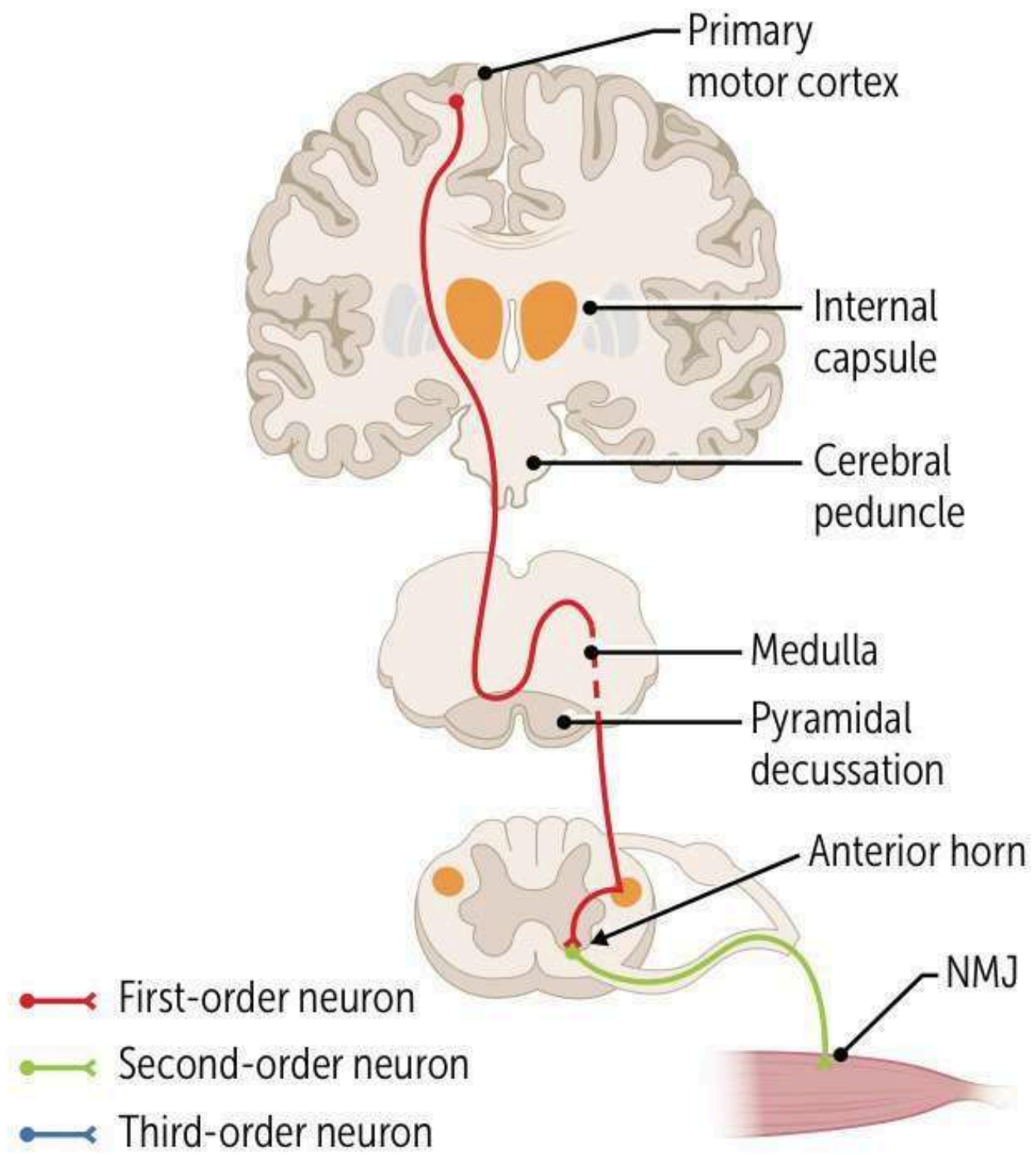
Upper and lower motor neuron lesion definitions:



Upper motor neuron lesion : is a lesion of the neural pathway above the anterior horn of the spinal cord or motor nuclei of the cranial nerves.

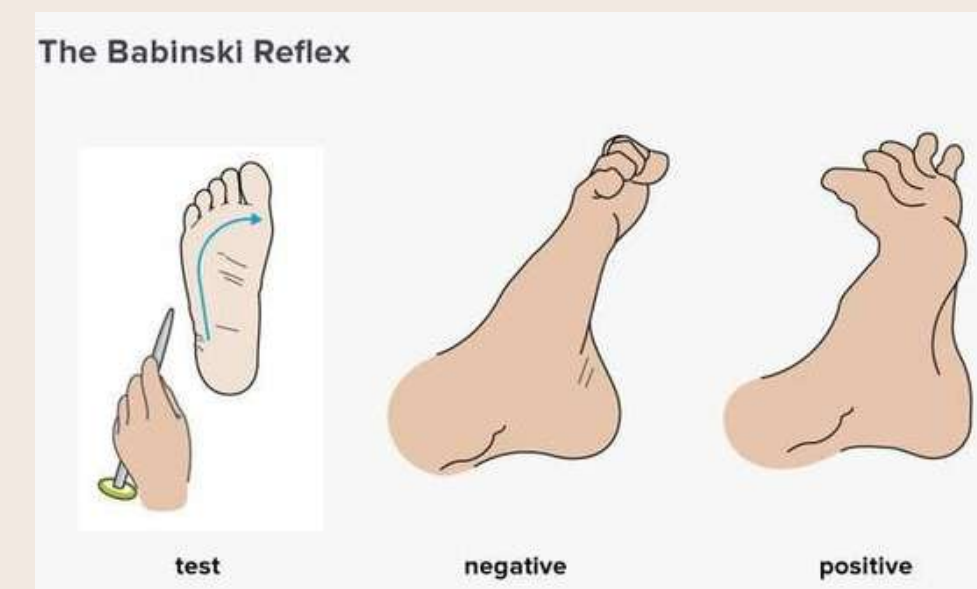
Lower motor neuron lesion : which originate in the brainstem (cranial nerve motor nuclei) and spinal cord (anterior horn cells) and directly innervate skeletal muscles

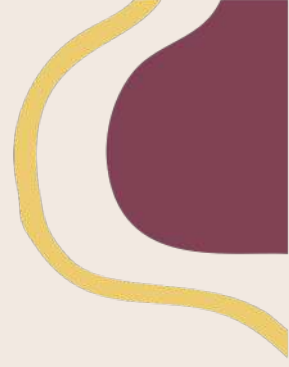




Upper motor lesion signs and symptoms :

- **Weakness:** Often affects large muscle groups but less severe than lower motor neuron lesions.
- **Spasticity:** Increased muscle tone (hypertonia) due to loss of inhibition.
- **Hyperreflexia:** Exaggerated deep tendon reflexes.
- **Clonus:** Rhythmic, involuntary contractions and relaxations of muscles.
- **Babinski Sign:** Upgoing big toe with fanning of other toes when the sole of the foot is stimulated.
- **No Muscle Atrophy:** Early on, though disuse atrophy may develop over time.
- **No Fasciculations:** Muscle twitching is generally absent.

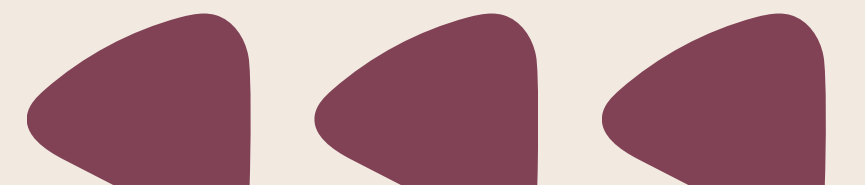




Lower motor lesion signs and symptoms:

- **Weakness:** Often severe and affects individual muscles or small muscle groups.
- **Flaccidity:** Decreased muscle tone (hypotonia) due to the loss of motor innervation.
- **Hyporeflexia:** Decreased or absent deep tendon reflexes.
- **Muscle Atrophy:** Significant loss of muscle mass over time due to denervation.
- **Fasciculations:** Visible, spontaneous contractions of muscle fibers, leading to twitching.

The reflex will be normal or absent.



UPPER MOTOR NEURON VERSUS LOWER MOTOR NEURON

UPPER MOTOR NEURON

A motor neuron that originates from the motor region of the cerebral cortex or from the brainstem

Located near the central nervous system

Transmit nerve impulses from the brain to the synapses of lower motor neurons

Form synapses with lower motor neurons

Found in cerebral cortex or in the brainstem

Cell bodies are bigger; located in the brain cortex

Classified based on the pathway they travel in

Has six pathways

Increased muscle tone and hyperactive deep reflexes are symptoms of damage

LOWER MOTOR NEURON

A motor neuron that transmits nerve impulses from the upper motor neurons to the effector muscles

Located near muscles

Transmit nerve impulses from upper motor neurons to the muscles

Form synapses with muscles

Found in brainstem and in the spinal cord

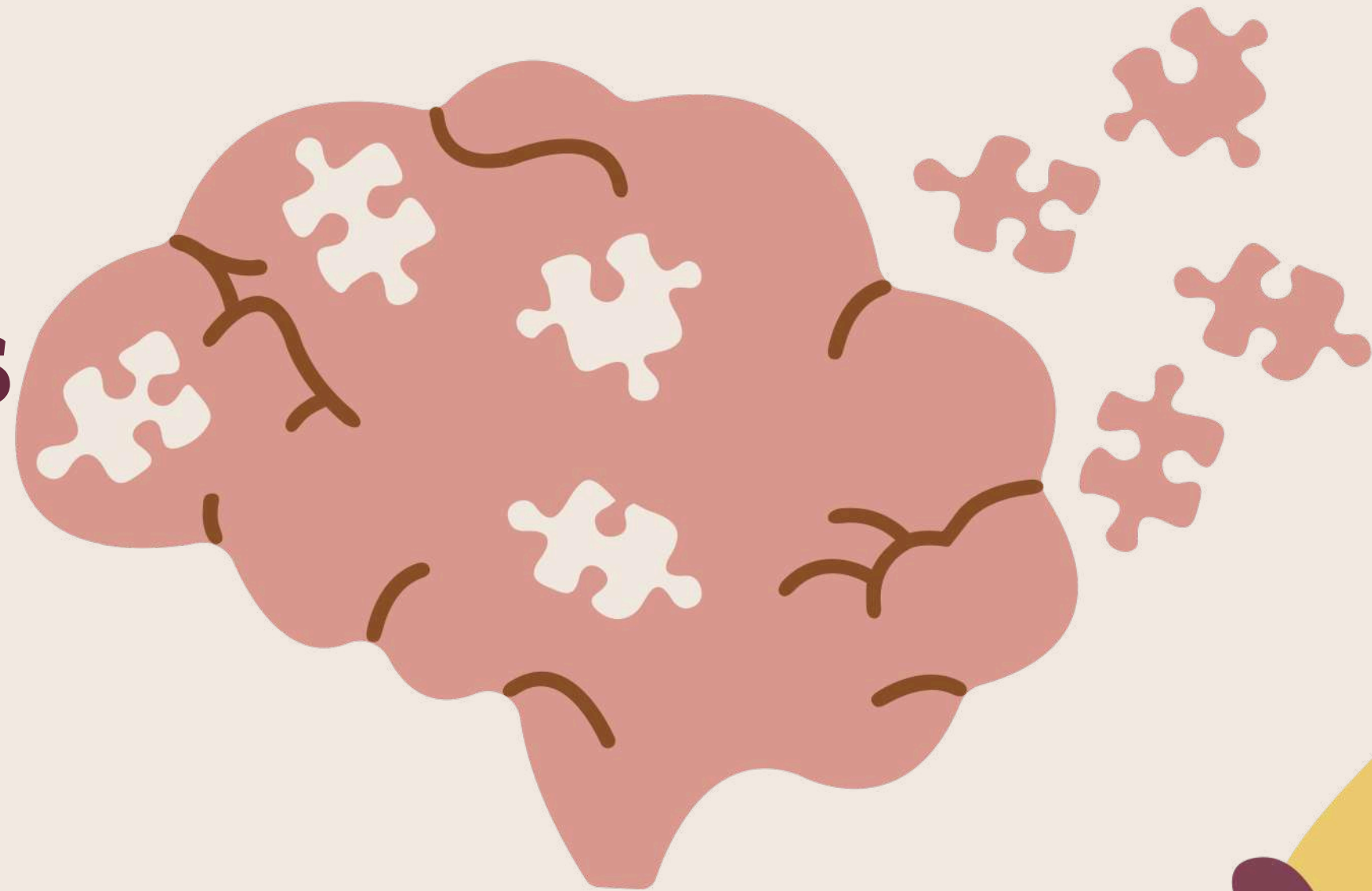
Cell bodies are small; located in the gray matter of the spinal cord and the brainstem

Classified based on the type of muscle fiber they innervate

Include cranial and spinal nerves

Decreased muscle tone, hyperactive deep reflexes, and muscle atrophy are symptoms of damage

Localization Principles of UMN Diseases



Upper motor neurons Overview

Defenition :

neurones whose cell bodies are in the motor cortex and whose axons run in the corticospinal (pyramidal) tracts to synapse with anterior horn cells.

Pathways involved :

Corticospinal Tract: Controls voluntary movements of limbs and trunk.

Corticobulbar Tract: Controls facial, jaw, and neck muscles via cranial nerves.

Key functions :

they play critical role in the control of voluntary movements , regulation of muscle posture and tone by inhibiting reflexes .

UMN Lesions Localization

UMN disorders can be localized based on the clinical presentation.

Clinical presentation: UMNL VS. LMNL symptoms

Key points

	LMN	UMN
Wasting	Present (neurogenic wasting)	Disuse atrophy only
Fasciculations	May be present	Absent
Tone	Normal or decreased (flaccidity)	Increased (spasticity)
Posture	–	Drift of outstretched arm (eyes shut)
Power	Focal weakness, e.g. distribution of individual nerves or roots	Movement-based Pyramidal distribution
Tendon reflexes	Depressed or absent	Brisk
Clonus	Absent	May be present
Plantar response	Downgoing or absent	Upgoing (positive Babinski)
Superficial abdominal responses	Present	May be absent
Gait	May be high-stepping	Spastic, scissoring, circumduction

UMN Lesions Localization

Also , UMN disorders can be localized based on the associated symptoms, which vary according to the lesion site:

Upper motor neuron disorders occur in 4 regions along the Central Nervous System (CNS) which are :

1. Cortical region (motor cortex)
2. Subcortical region (Internal Capsule)
3. Brain Stem
4. Spinal Cord

UMN Lesions Localization

A. Cortical Lesions

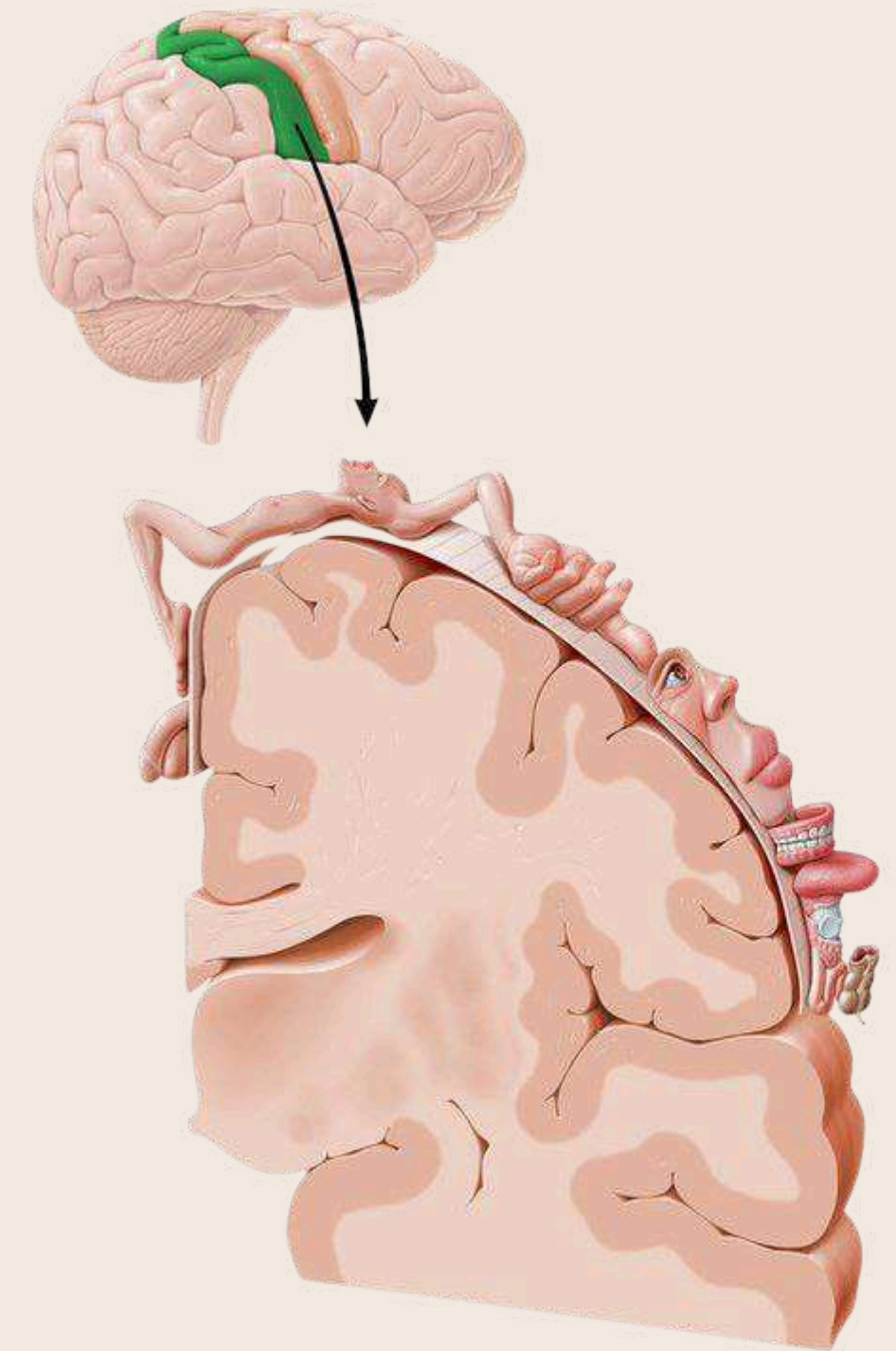
Anatomy.

The motor cortex (precentral gyrus) controls movement via a somatotopic representation known as the motor homunculus.

Key Symptoms

Contralateral Weakness: Lesions in the motor cortex result in weakness on the opposite side of the body.

Specific Localization: A lesion in the medial motor cortex affects the lower limb (leg), while a lateral cortex lesion affects the face and upper limb (hand, arm).



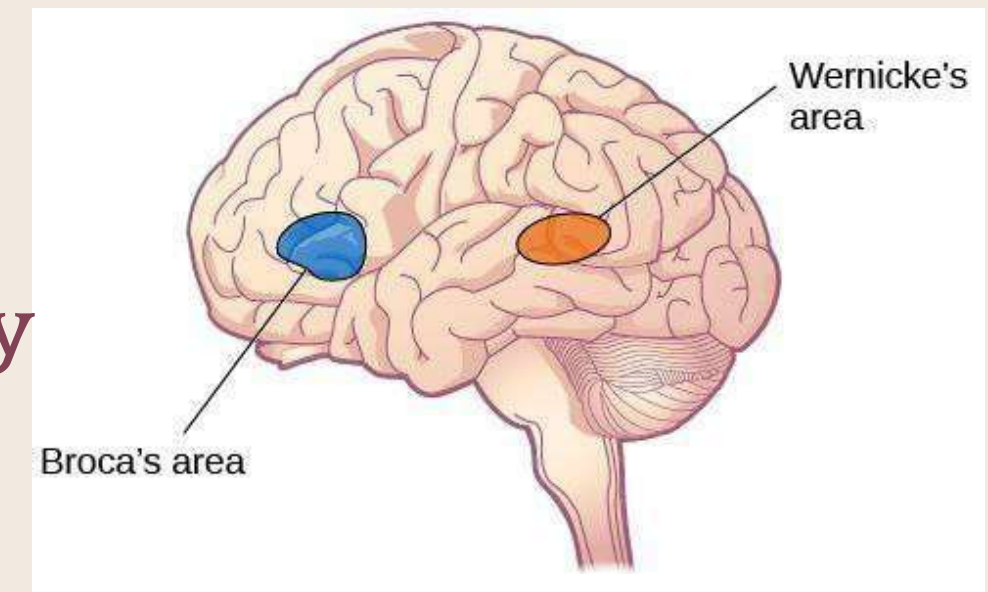
UMN Lesions Localization

Associated Symptoms:

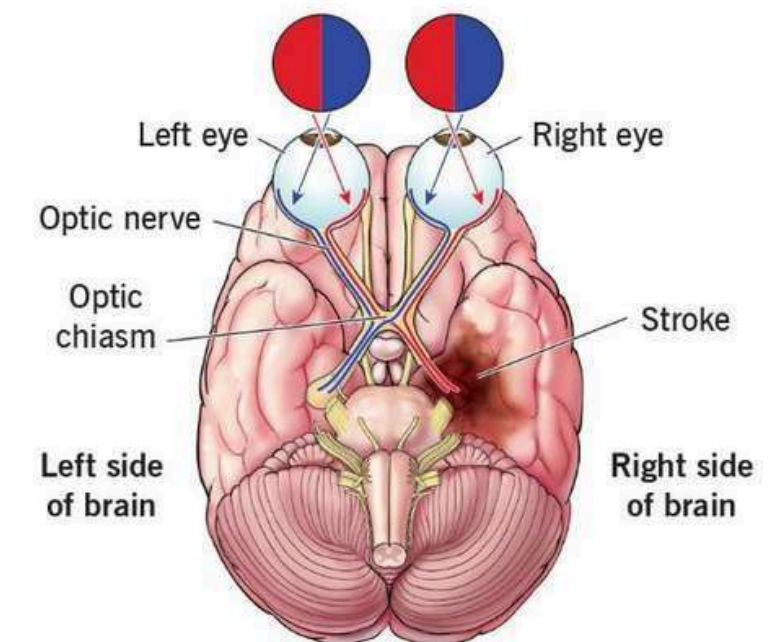
-Aphasia: If the dominant hemisphere is affected, particularly Broca's or Wernicke's areas.

-Hemineglect: Common in non-dominant parietal lesions, leading to the inability to perceive one side of space (usually the left).

-Contralateral Visual Field Defects: Damage to the occipital cortex or optic radiations can cause homonymous hemianopia.



Homonymous hemianopia



Left side homonymous hemianopia



UMN Lesions Localization

B.Subcortical Lesions(Internal Capsule)

Anatomy:

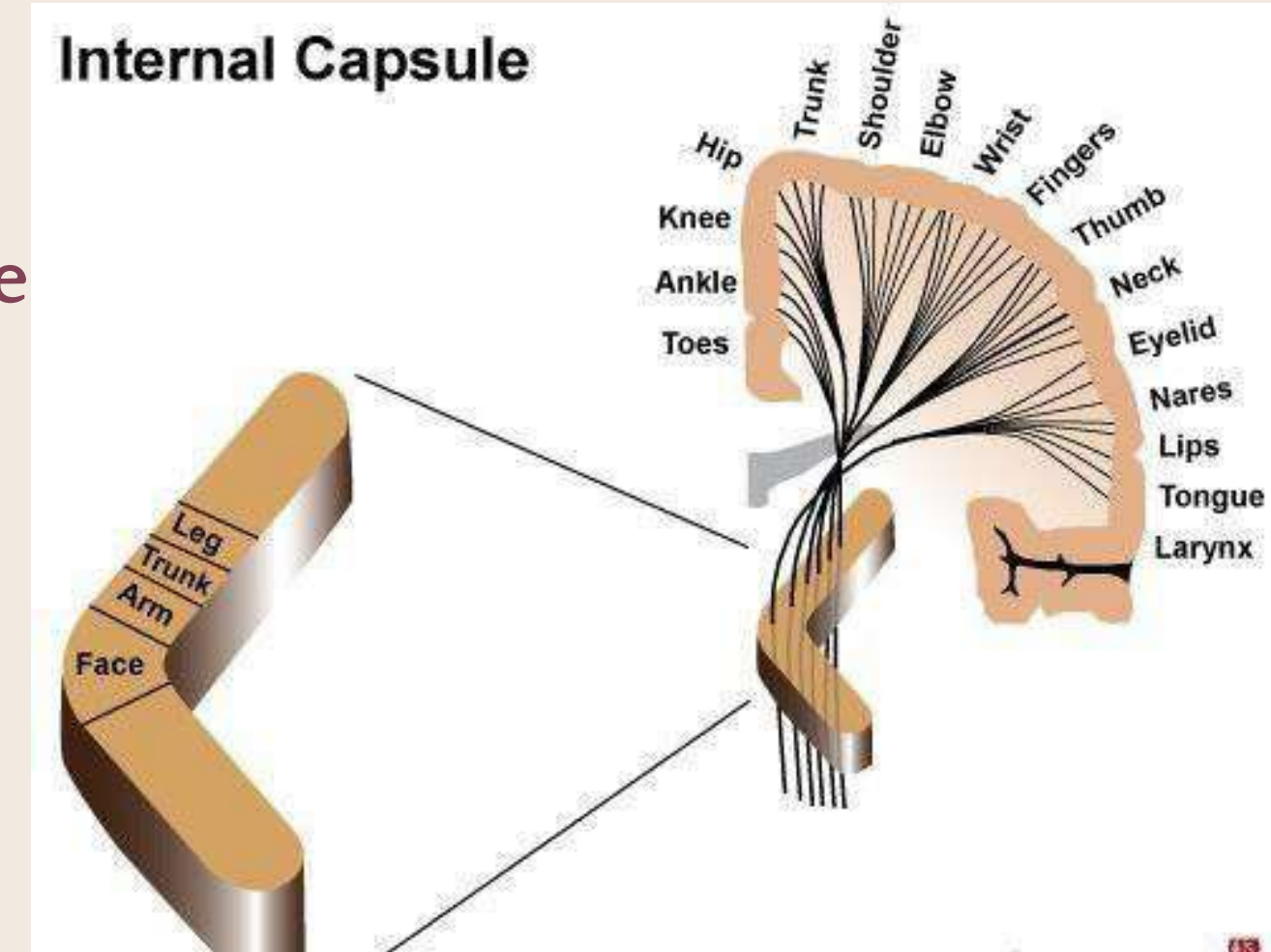
The internal capsule contains dense corticospinal and corticobulbar fibers.

Key Symptoms:

Contralateral Hemiparesis: A small lesion in the internal capsule can result in profound weakness involving the face, arm, and leg.

Minimal Sensory Involvement: Pure motor hemiparesis is common, with little or no sensory loss.

Example: A stroke in the posterior limb of the internal capsule causes contralateral weakness without other significant deficits.



UMN Lesions Localization

C. Brainstem Lesions

Anatomy:

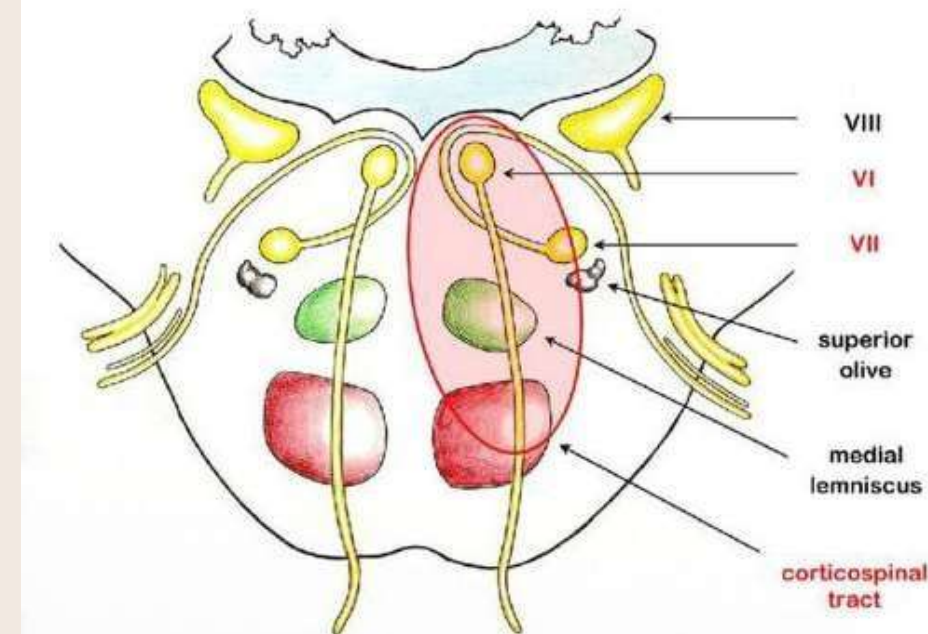
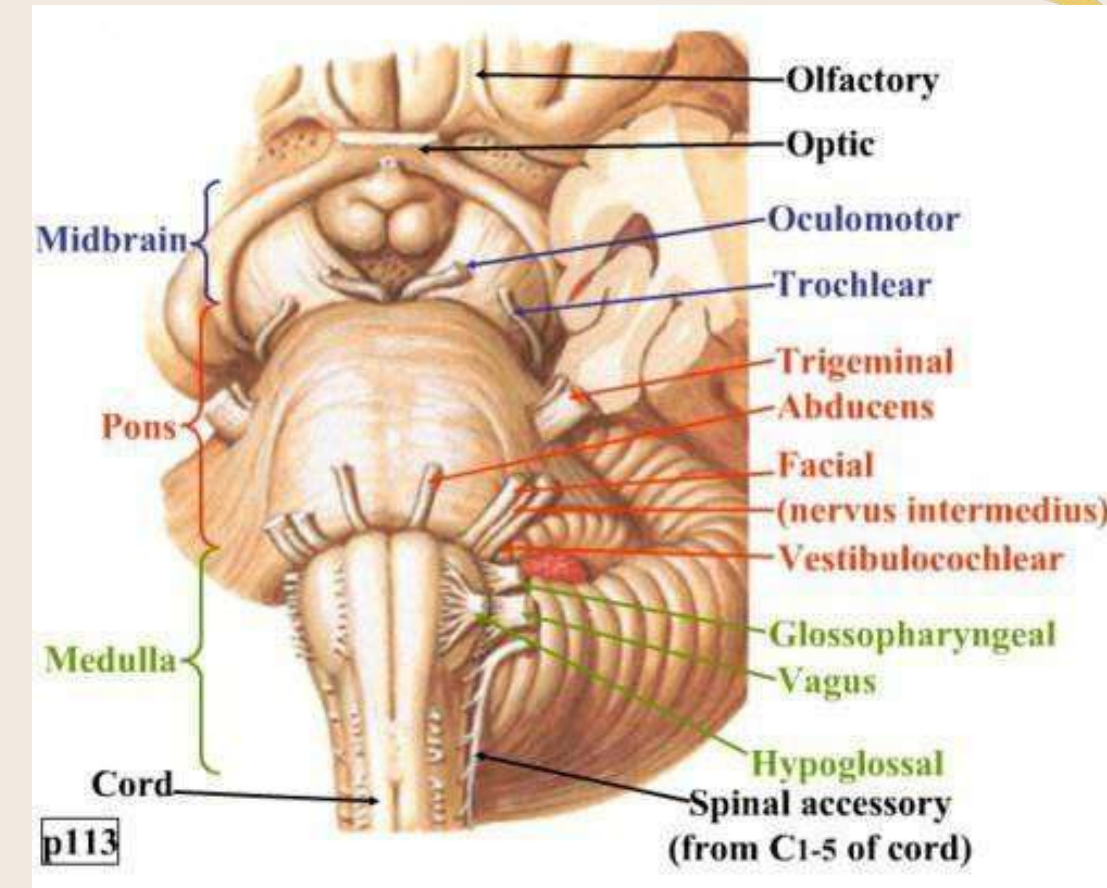
The brainstem contains descending corticospinal and corticobulbar tracts, as well as cranial nerve nuclei.

Key Symptoms:

Crossed Syndromes: UMN signs affecting one side of the body (contralateral) and cranial nerve dysfunction on the opposite side (ipsilateral).

Example: A lesion in the midbrain (Weber's syndrome) may cause ipsilateral oculomotor (CN III) palsy and contralateral hemiparesis.

Cranial Nerve Deficits: Cranial nerve involvement helps localize the lesion to a specific part of the brainstem (e.g., CN V, VII, IX, X, XII).



UMN Lesions Localization

D. Spinal Cord Lesions:

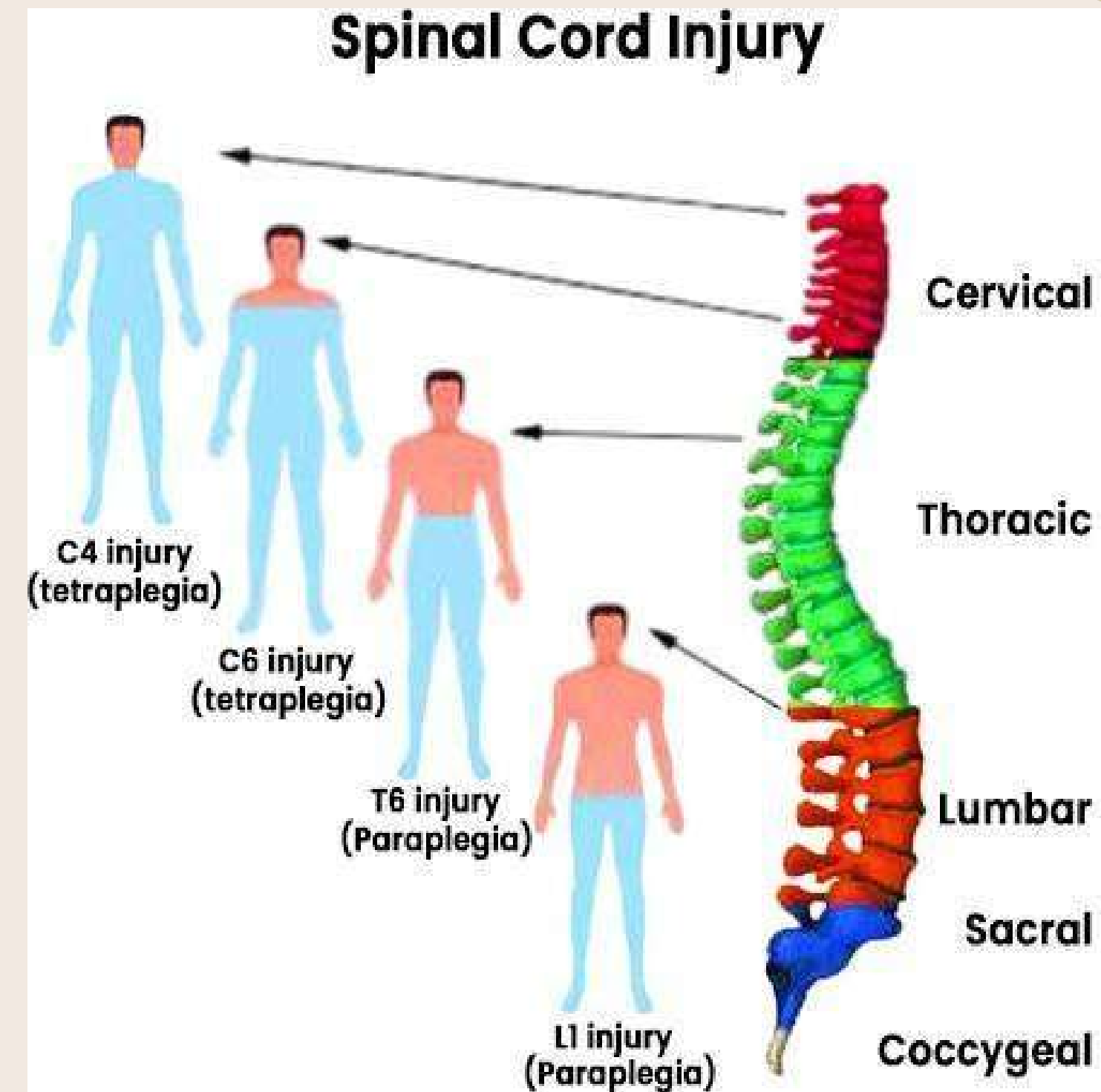
Anatomy:

UMN tracts travel down through the spinal cord and synapse onto lower motor neurons.

Key Symptoms:

1. Cervical Cord Lesions: Cause weakness or paralysis in both upper and lower limbs (quadriparesis) if both sides are affected, often with sensory level changes.

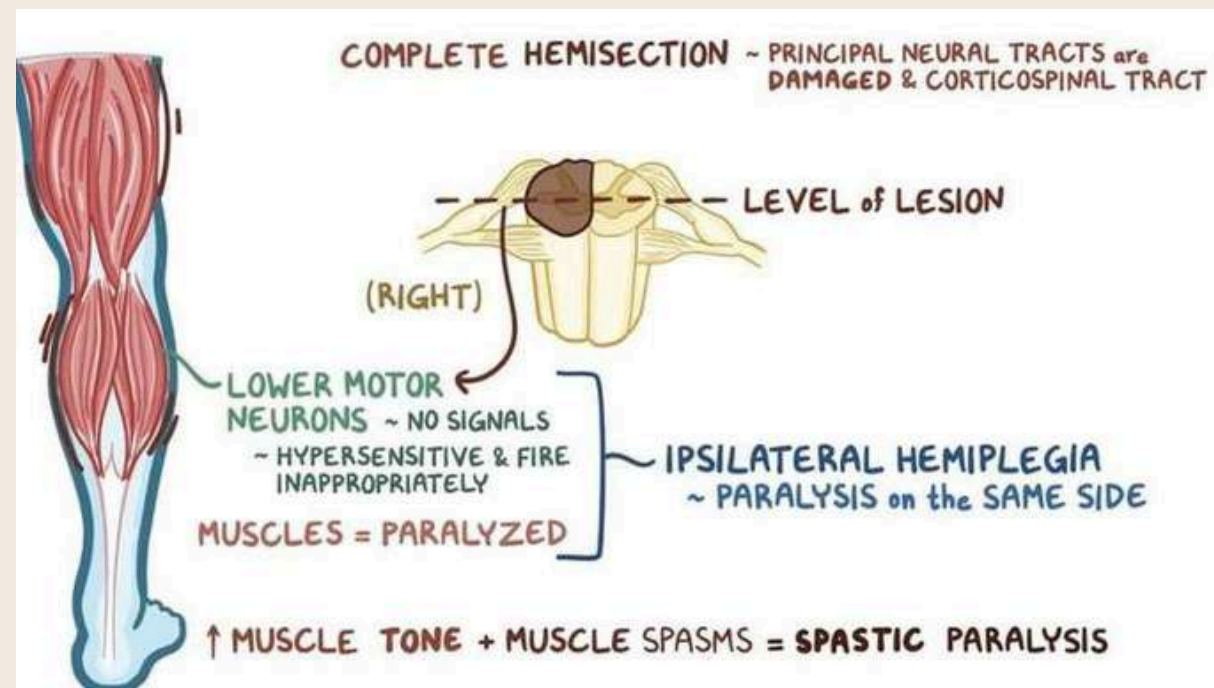
2. Thoracic or Lumbar Cord Lesions: Affect the lower limbs (paraparesis or paraplegia).



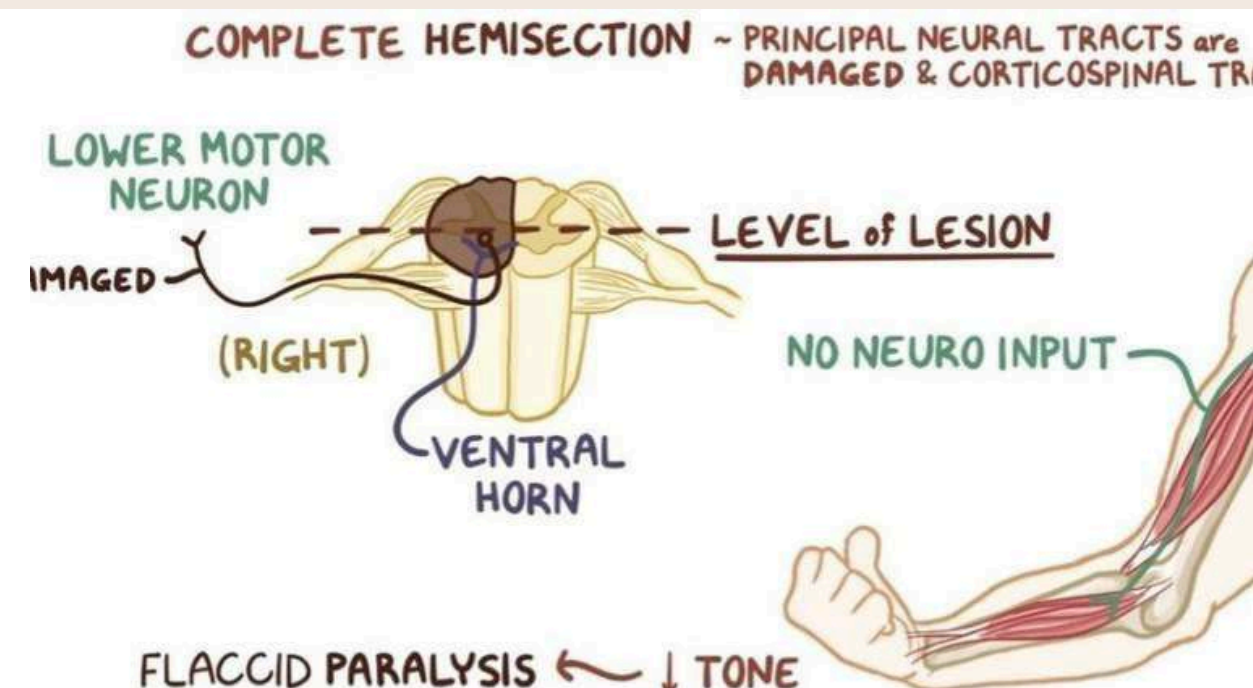
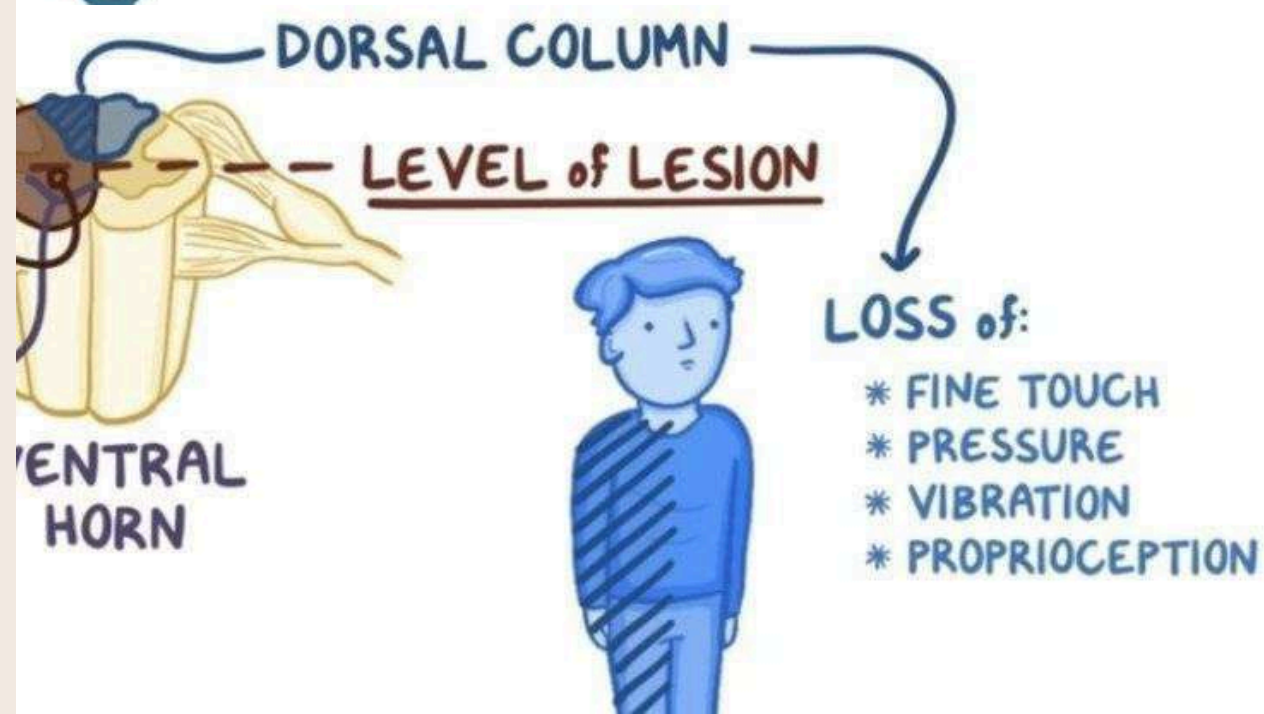
UMN Lesions Localization

3. Hemisection (Brown-Séquard Syndrome): Causes ipsilateral motor deficits (and proprioception loss) and contralateral pain/temperature sensation loss below the level of the lesion.

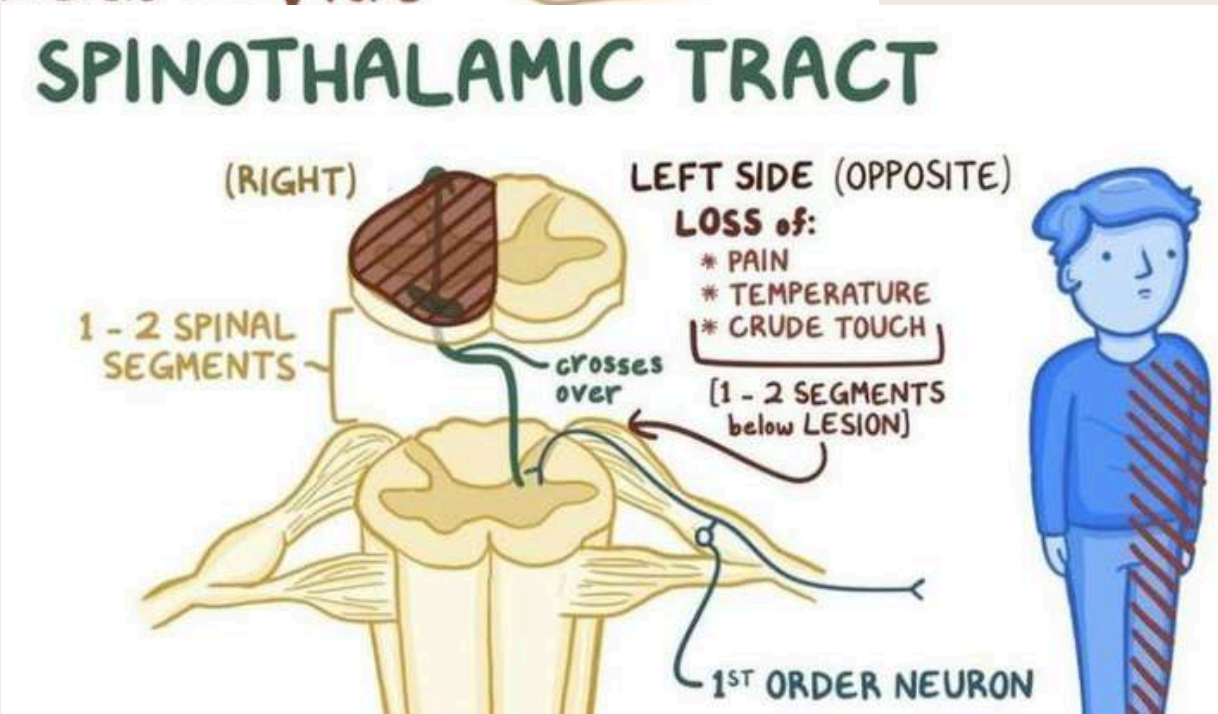
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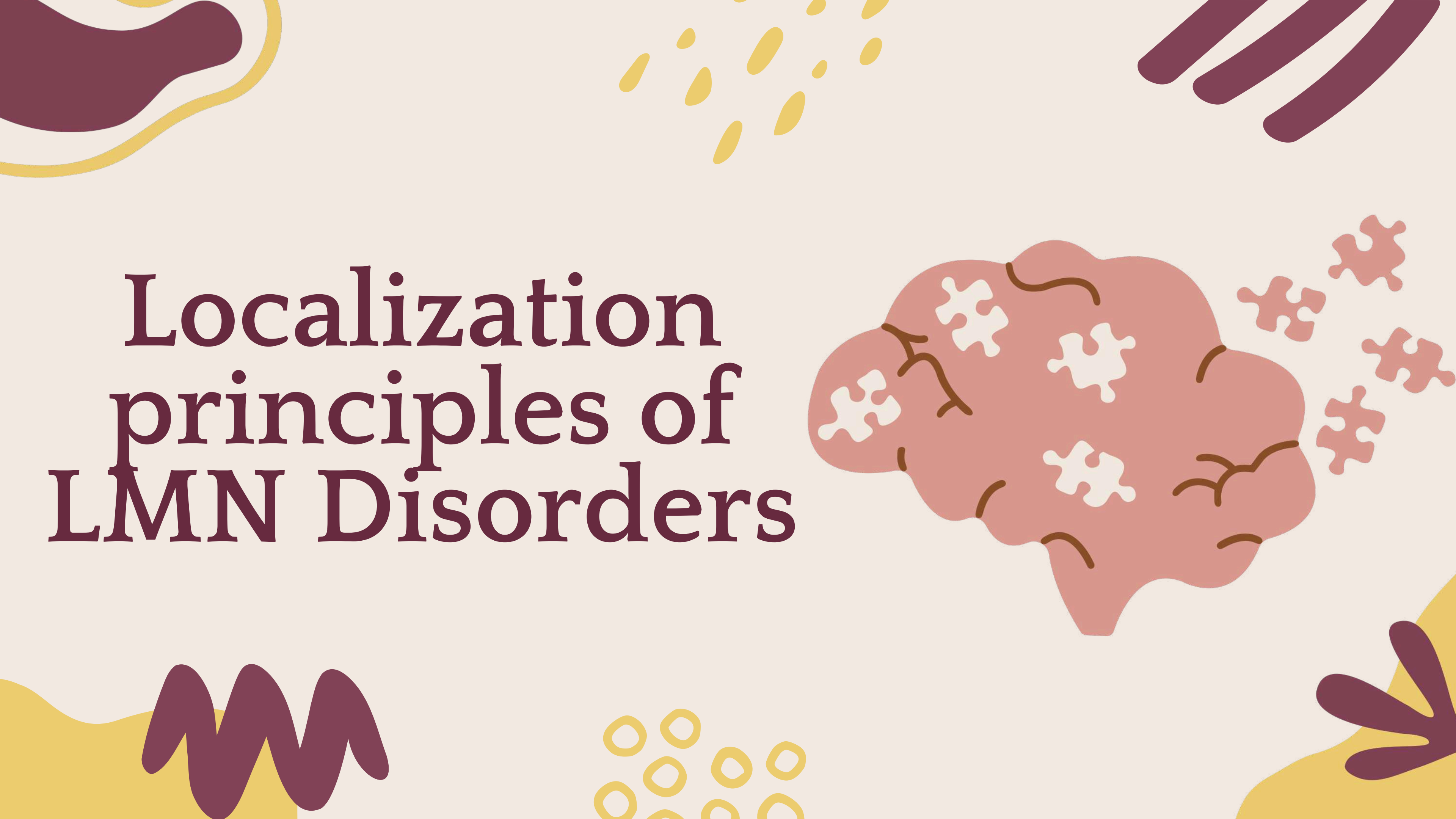


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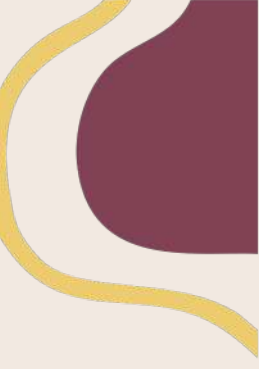




Localization principles of LMN Disorders



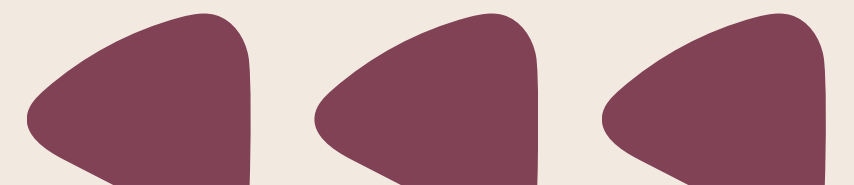
Lower Motor Neuron Overview



Definition : it constitutes of anterior horn cells and its projecting axon all the way to root and nerve to the neuromuscular junction of the innervated muscle

Key Function :

They play critical role in Muscle Activation , Reflex Action, Muscle tone regulation , coordination of Movement along with UMN



Localization principles of lower motor neuron disorders

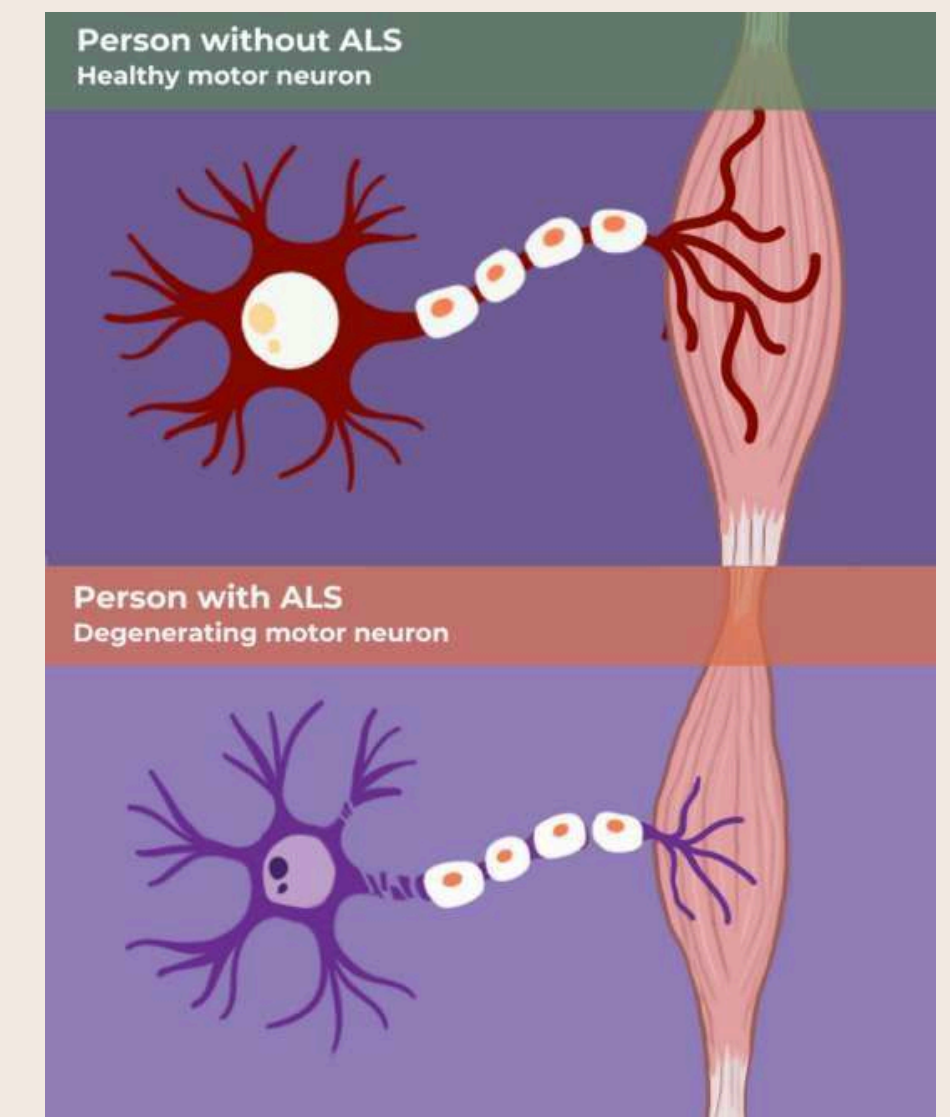
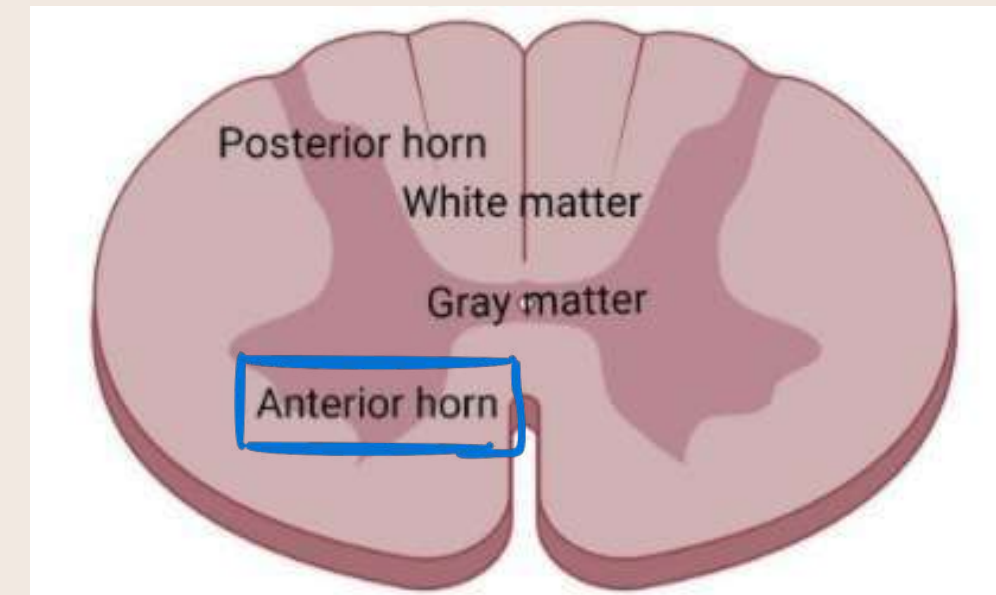
Understanding the location of Lesions helps in diagnosing any specific type of LMN disorder and guiding appropriate treatment.

Lower motor neuron (LMN) disorders can occur in various locations within the nervous system including :

1. Anterior Horn of the Spinal Cord
2. Spinal Nerve Roots
3. Peripheral Nerves
4. Neuromuscular Junction
5. Brainstem

Anterior Horn of the Spinal Cord

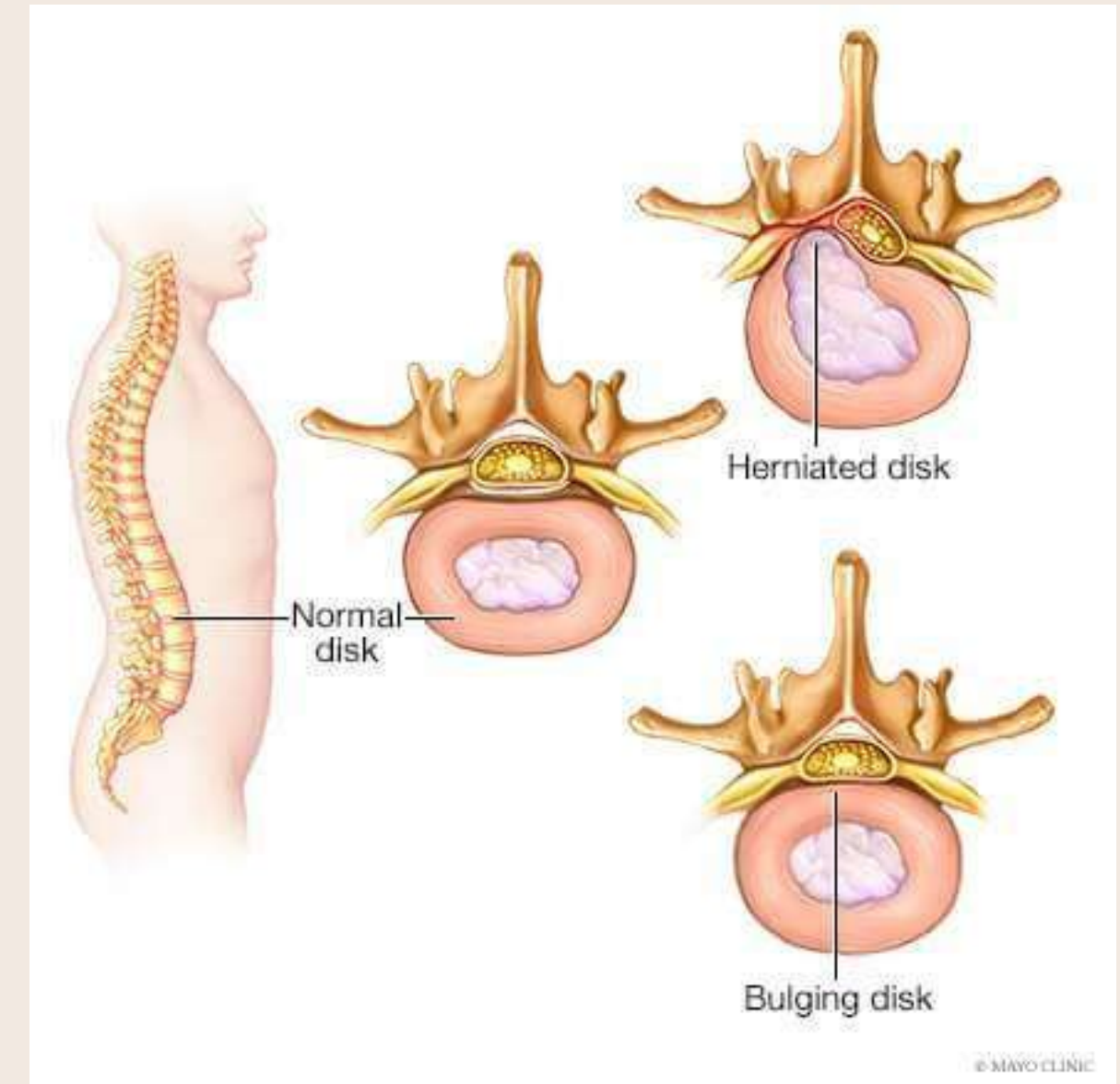
The cell bodies of lower motor neurons reside here. Damage can lead to conditions like spinal muscular atrophy or amyotrophic lateral sclerosis (ALS), Motor neuron disease (alternatively known as amyotrophic lateral sclerosis) is a progressive degenerative disorder of cortical, brainstem and spinal motor neurones (i.e., both UMNs and LMNs).



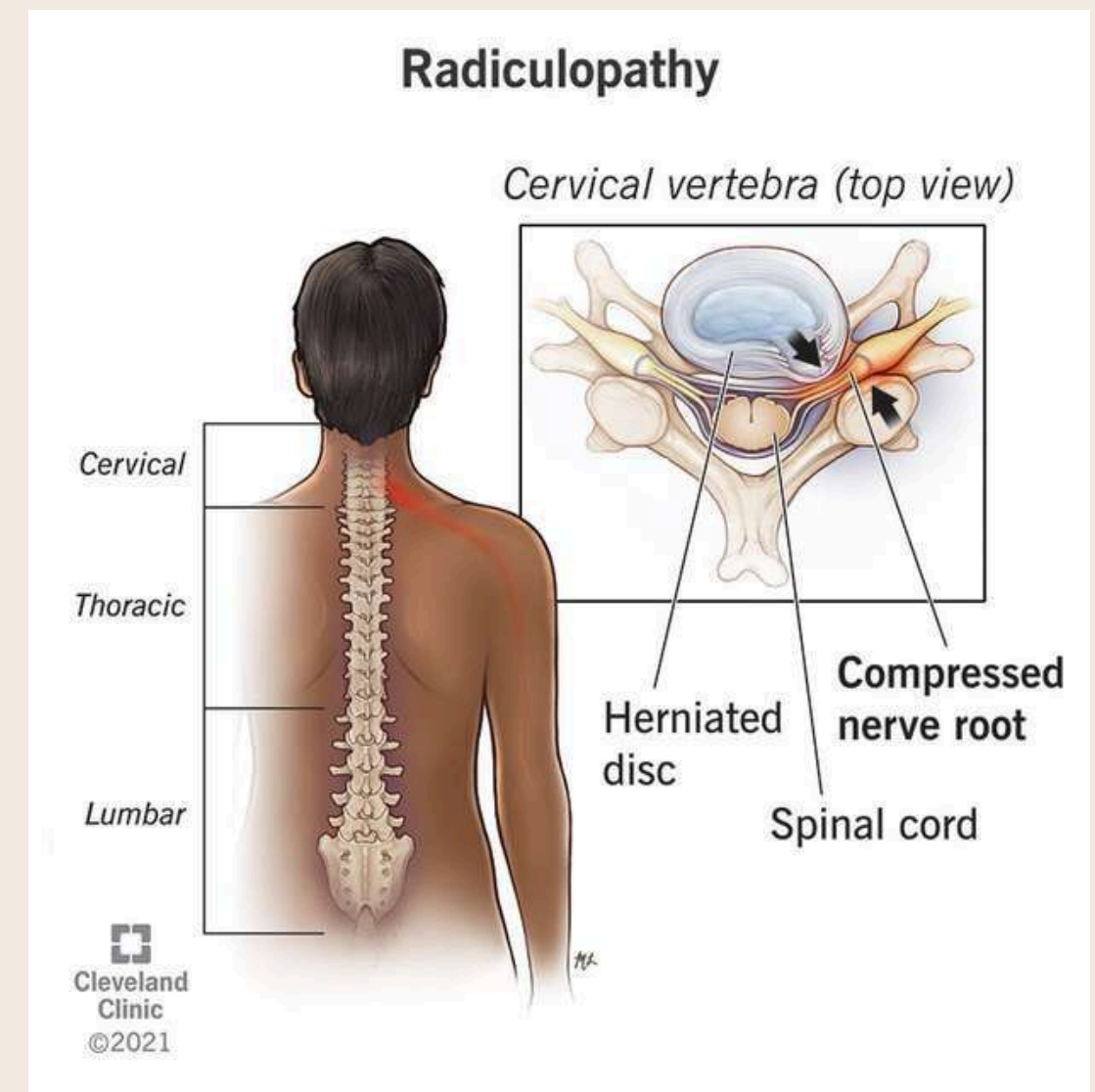
Spinal Nerve Root

LMNs exit the spinal cord through nerve roots. Injury or compression (e.g., herniated discs) can result in radiculopathy, causing weakness in specific muscle groups.

Radiculopathy refers to symptoms that develop when there is compression of a spinal nerve root. Most commonly, the nerve compression is related to a disc herniation or spondylosis (degenerative changes in the spine) and may occur with or without trauma

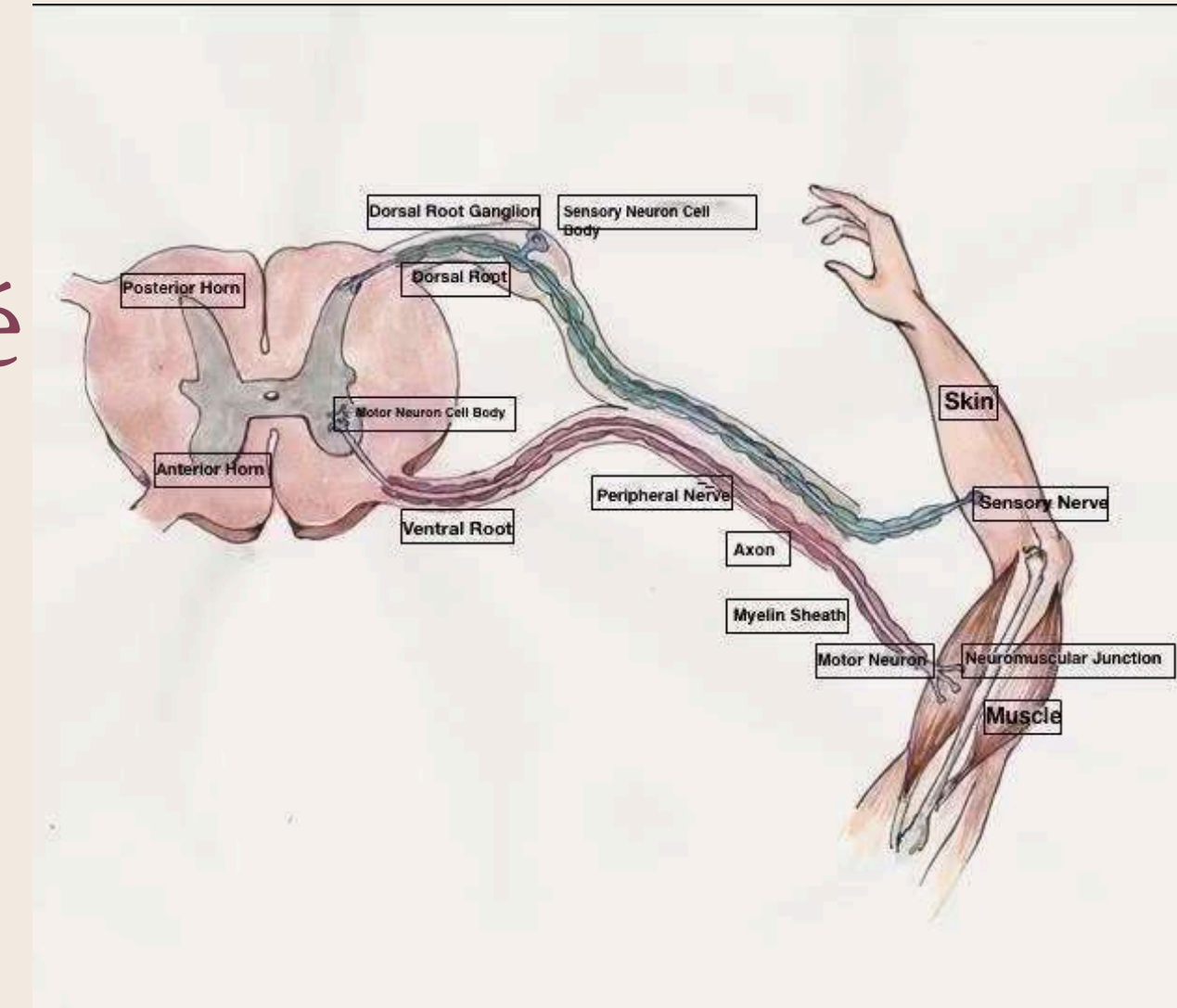


It is important to note that not all disc herniations cause nerve compression or pain. While any nerve root can be affected, the lower cervical (neck) and lower lumbar (low back) levels are the most common. Because the fibers of one spinal nerve root provide sensation and strength in a specific region of the body, arm, or leg, a compressed nerve will cause symptoms in the region where the nerve provides strength and sensation, there is a lot of Symptoms that can be felt including : Pain , Numbness or Tingling , Weakness and more



Peripheral Nerves

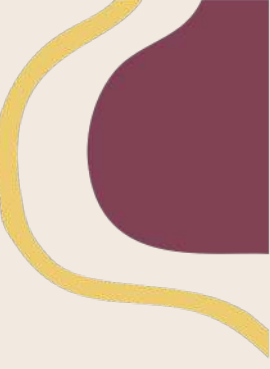
There are many conditions that could affect our peripheral nerves (like: diabetic Neuropathy and Guillain-Barré syndrome) which leads to muscle weakness and atrophy in the areas innervated by the damaged nerves



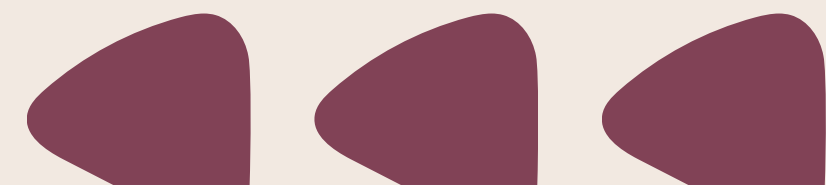
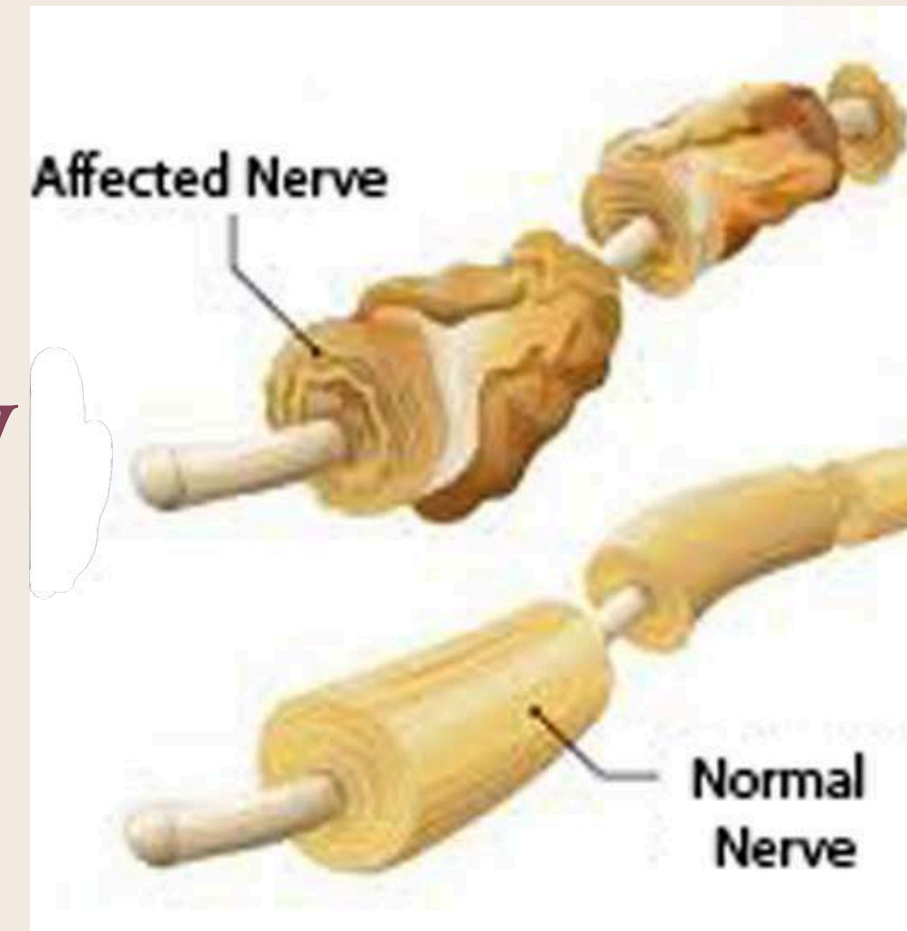
but the question is What is Guillain-Barre syndrome????



Guillain–Barré syndrome



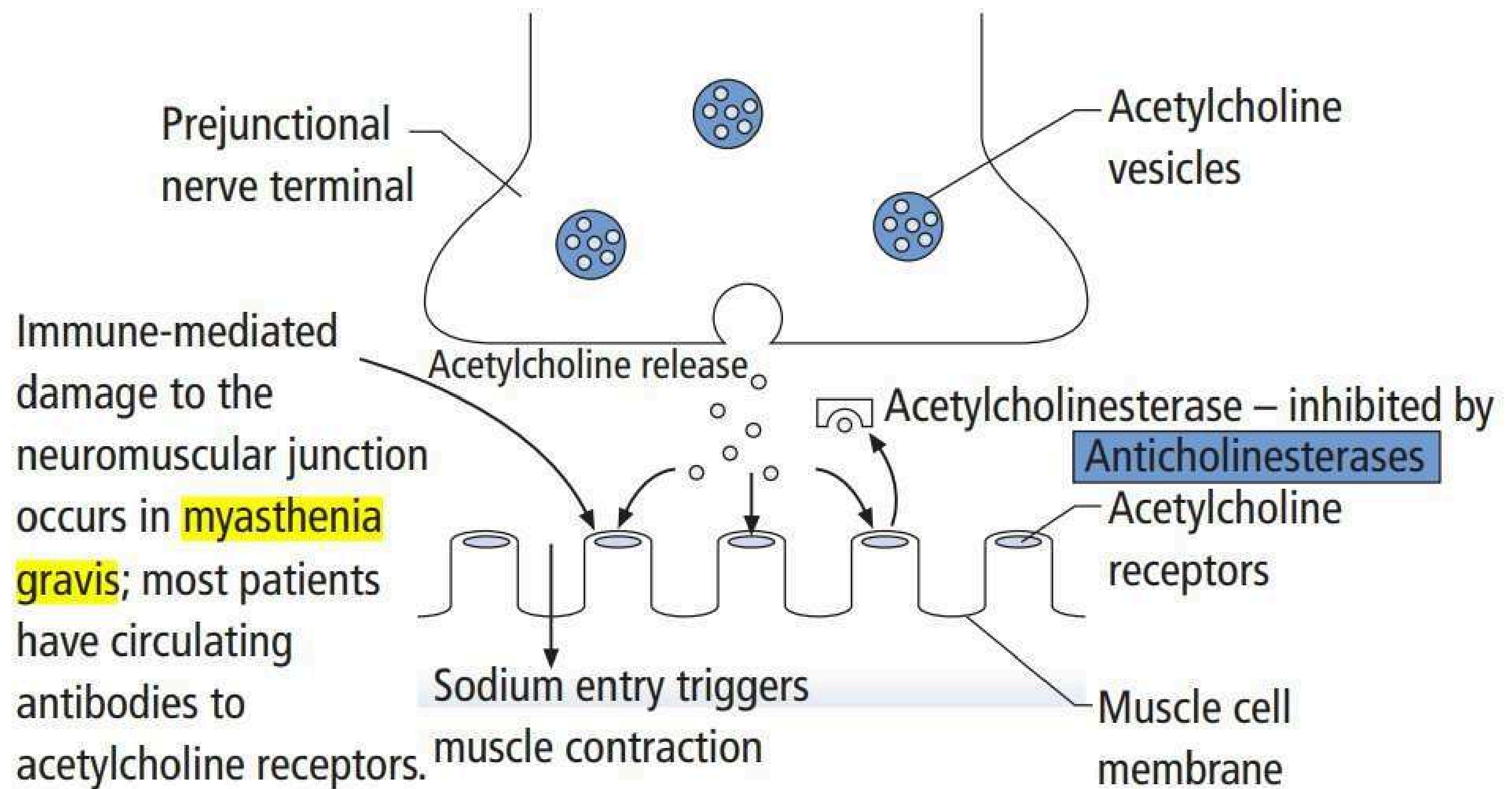
Guillain–Barre syndrome is an acute inflammatory demyelinating polyneuropathy the cause is incompletely understood, but the pathogenetic mechanism involves inflammatory demyelination with variable axonal damage in the peripheral nervous system , it is assumed that it is an autoimmune process that is triggered by many agents like Viruses like CMV , Bacteria etc.



Neuromuscular Junction

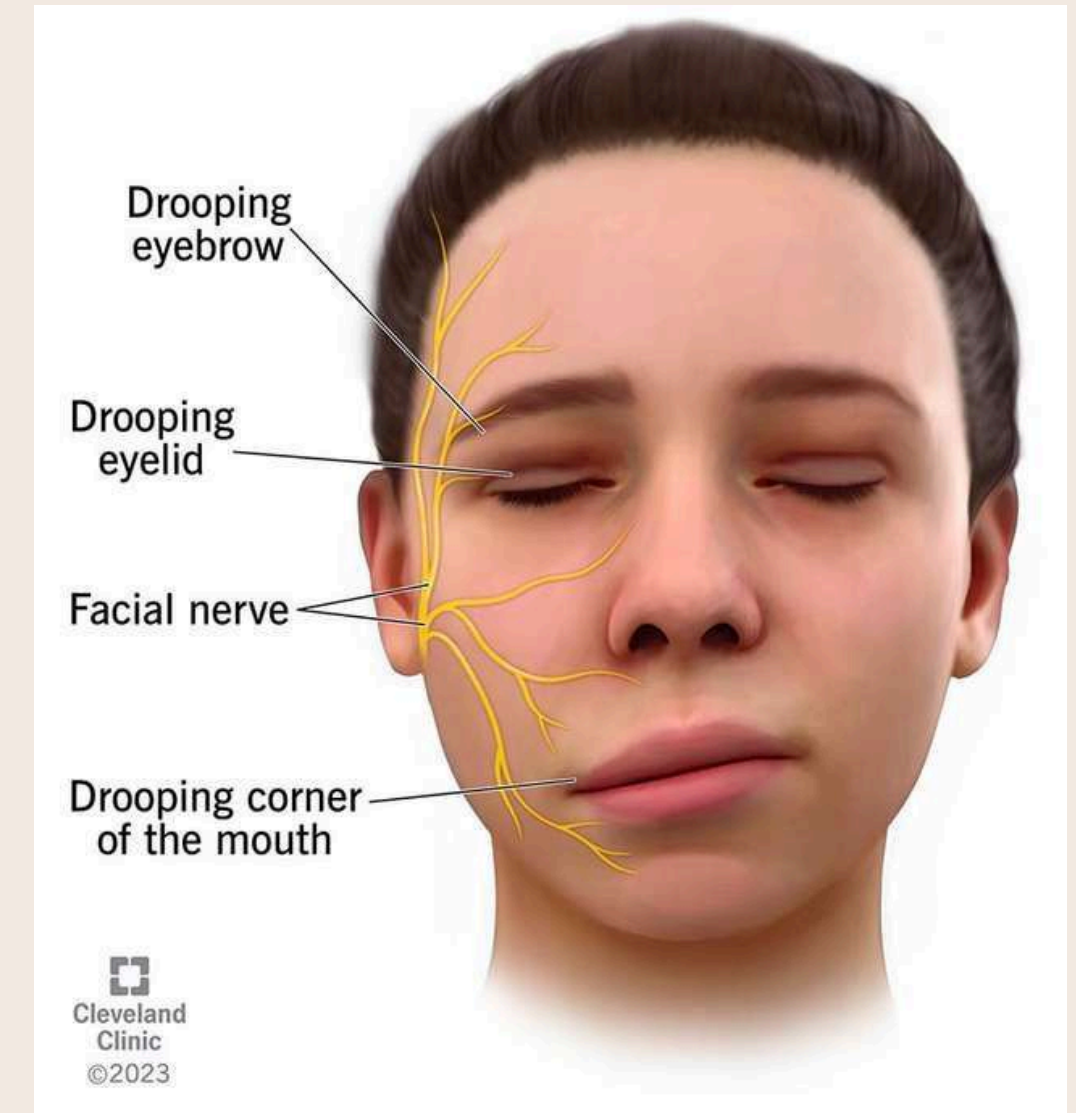
A Damage could happen to our neuromuscular junction as it can affect either presynaptic mechanisms or postsynaptic mechanisms, preventing the junction from functioning normally , and on the disorders that could affect it is Myasthenia Gravis which does affect the transmission of Signals from LMNs to our Muscles

Myasthenia gravis is an autoimmune disorder in which most patients have circulating antibodies to acetylcholine receptors at the neuromuscular junction, The disease is rare, annual incidence is approximately 0.4 in 100,000, but with most patients surviving long term



Brainstem

Lower motor neurons that innervate facial muscles and muscles of the throat reside in the brainstem (e.g., cranial nerves VII and IX). Damage here can lead to specific deficits like facial droop or dysphagia (difficulty in Swallowing).



Thank you for Listening

- References: •
- •
- Mayo Clinic •
- Neurology Lecture notes 9E •
- Neuroanatomy through Clinical cases •
- 2nd edition •
- MCU Blueprint Neurology 5E
- Macleod's Clinical Examination 15E