CLINICAL NEUROLOGY ESSENTIALS

Robert Coni

Table of contents

5. The spinal cord

6. The peripheral nervous system

Chapter 1

THE NEUROLOGIC METHOD

THE NEUROLOGICAL EXAMINATION

The eight steps

1. Mental status examination
2. Cranial nerve examination
3. Motor examination
4. Reflex examination
5. Special reflex examination
6. Sensory examination
7. Cerebellar examination
8. Gait and station examination

Mental state examination

Assess

- Appearance and mood
- Higher cortical functions
    - speech
    - attention
    - orientation
    - memory
    - calculation
    - perception and praxis

Aphasia—refers to a dysfunction of language, either production and / or comprehension.

Dysarthria—refers to a dysfunction of the mechanical process of speech production, and language processing is normal.

Neglect—a non-dominant hemisphere injury (usually on the right side of the brain) provokes a syndrome characterized by neglect of the opposite side of the body and may involve the visual field, sensory appreciation from an affected region, or non-recognition of one's own body (asomatognosia). The injured individual may extinguish stimuli from the left side of the body when both sides are stimulated simultaneously.

Cranial nerve examination

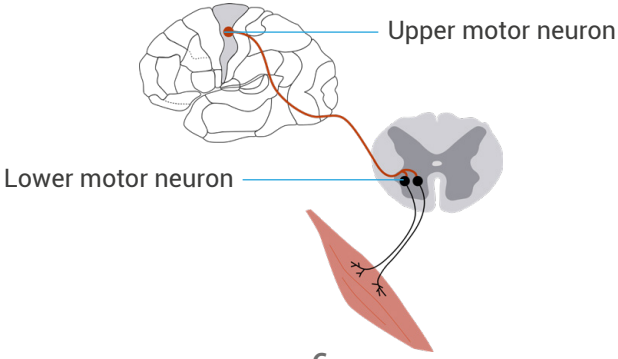
When testing the cranial nerves, it is best to group them by function.

Test the nerves associated with the following functions

- Smell (CN I)
- Eye movements and vision (CN II, III, IV, and VI)
- Facial movement and sensation (CN V and VII)
- Hearing and balance (CN VIII)
- Oromandibular function and sensation (CN IX, X, and XII)
- Sternocleidomastoid and trapezius muscles (CN XI)

Motor examination

It is useful to localize muscle weakness to the upper motor neuron (suggesting a brain or spinal cord lesion) or the lower motor neuron (suggesting a peripheral nerve injury).



Upper motor neuron signs

- Hypertonicity
- Hyperreflexia
- Babinski response (extensor plantar response)
- Clonus

Lower motor neuron signs

- Flaccid tone
- Muscle wasting and atrophy
- Hyporeflexia
- Fasciculations

If time is limited, test extensors of the arms and flexors of the legs.

While observing for bulk and tone, also test the strength of the muscle. **Directed muscle testing** involves having the patient contract the muscle against resistance and then using the universal grading system, determined by the Medical Research Council, applying a numerical value to the strength of that muscle. **(Reference: *O'Brien, 2010*)**

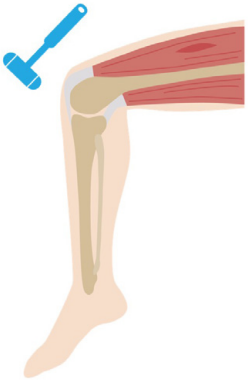
Grading muscle power

0	No power
1	Slight flicker of muscle contraction
2	Movement only when gravity is removed
3	Movement against gravity but not resistance
4	Movement against some resistance
5	Full power against resistance

Reflex examination

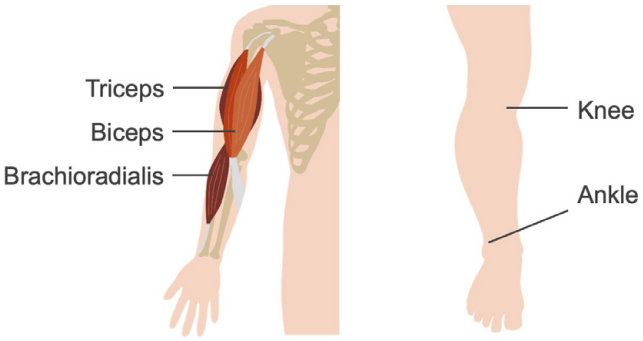
Reflexes represent a window to the motor system and can be helpful in differentiating upper motor neuron weakness from lower motor neuron weakness. In upper motor neuron disorders, reflexes are pathologically brisk, while in lower motor neuron disorders, they are decreased or absent.

Reflexes are elicited by tapping the tendon with a reflex hammer and looking for muscle contraction.



4	Sustained with clonus
3	Hyperactive
2	Normal
1	Hypoactive
0	Absent

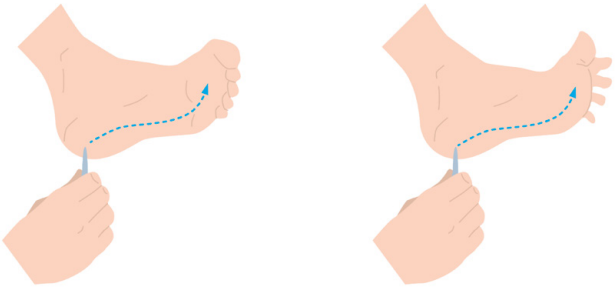
Commonly checked reflexes



Special reflex examination

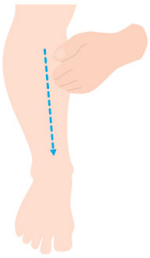
The plantar reflex

The plantar reflex is elicited by using the smallest amount of pressure possible to obtain a response, while moving from the heel toward the large toe, on the outer surface of the sole of the foot. You observe the response at the first metatarsophalangeal joint. In adults, the toe will normally move downward producing a flexor plantar response. If the toe moves upward, producing an extensor plantar response—also called the **Babinski response**—this suggests an **upper motor lesion** in the motor pathways or somewhere along the corticospinal tract.

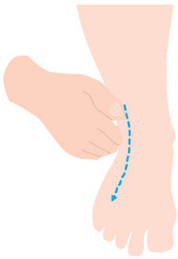


Normal response Babinski response

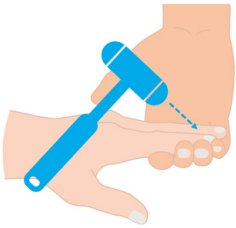
Other special reflexes



Oppenheim reflex

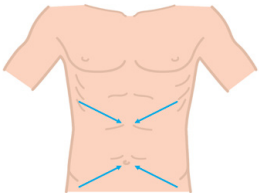


Chaddock's reflex

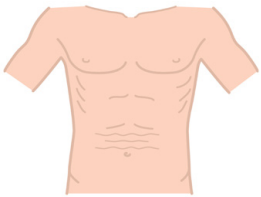


Finger flexor reflex

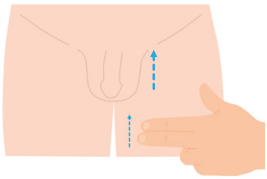
Hoffman's sign



Superficial abdominal reflex



Beevor's sign



Cremasteric reflex

Sensory examination

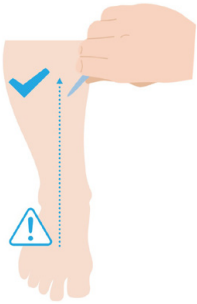
Cutaneous sensibilities

- Pinprick
- Temperature
- Light touch
- Vibration
- Proprioception

Cortical sensibilities

- Two-point discrimination
- Graphesthesia
- Stereognosia
- Double simultaneous stimulation

As you are testing sensation, it is generally best to first test regions with sensory loss, then move towards regions with normal sensation.



Always remember to interpret your findings within the clinical context. Integrating the sensory and motor information will help you make an accurate diagnosis.

Cerebellar examination

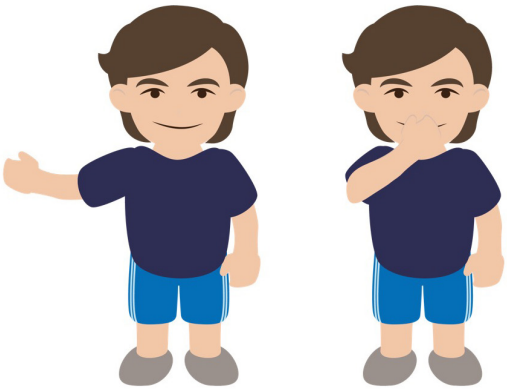
Pronator drift

While testing for pronator drift, a quick downward push of the extremity should produce a very quick reflexive return to the previous posture. If the extremity bounces up and down before achieving return to the beginning posture, this is likely a subtle sign of cerebellar dysfunction.

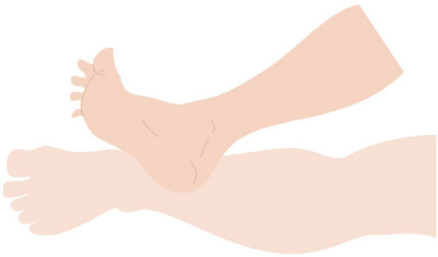
Coordination

Overall, look for smooth and accurate movement. When observing the gait of the patient, you might see swaying and veering off in a particular direction with a lack of movement fluidity, indicating ataxia, which could be due to cerebellar disease.

In the **upper extremities**, test cerebellar function by performing the finger to nose to finger test.



In the **lower extremities**, have the patient perform the heel to shin maneuver to assess the fluidity, and accuracy of movement.



Remember, muscle tone is also decreased in cerebellar disease.

Gait and station examination

What to look for when assessing gait

- Symmetrical movement
- Pain
- Stooping
- Circumduction
- Hiking up of the hip
- Steppage gait
- Gait speed
- Step width

Some common gait types

Parkinsonian gait—characterized by festination (hesitancy and the stopping and starting of movement).

Ataxic gait—associated with imbalance causing the patient to veer off line, either due to sensation loss, or some effect of cerebellar dysfunction.

Apraxic gait—the patient appears to have forgotten how to walk. This can be seen with frontal lobe pathology or normal pressure hydrocephalus.

Hemiplegic gait—there is swinging out of the leg to advance it on one side. This commonly occurs after a stroke.

Common tests for gait

Tandem walk

Heel walk

Toe walk

Romberg test

Pull test

Further reading

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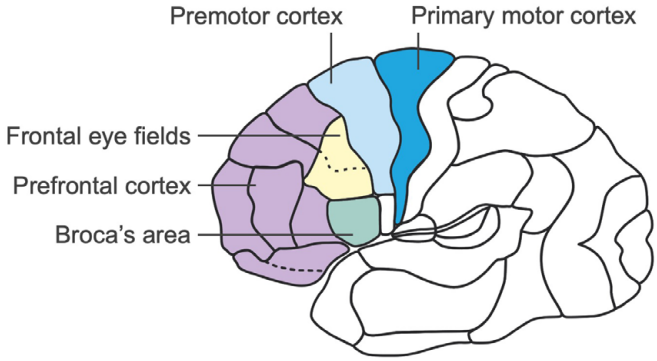
Chapter 2

THE CEREBRAL HEMISPHERES

FRONTAL LOBE

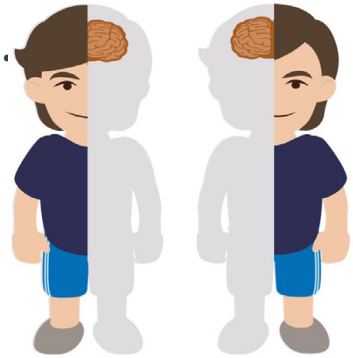
Important regions

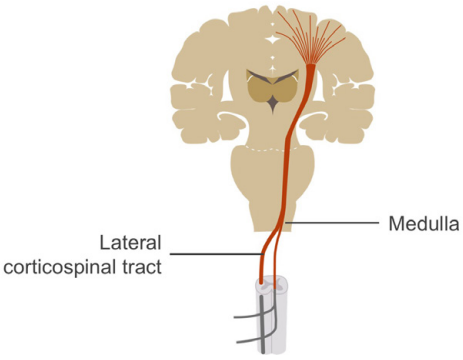


Clinicoanatomic correlation

Motor function

- Controlled by motor cortex
- Corticospinal tract
  - origin of the primary motor tract from the brain to the spinal cord
- Left side of brain controls right side of body and vice versa





Executive functions

- Controlled by prefrontal cortex
- Involves focus, concentration, regulation of motor and sensory input
- Planning
- Initiating
- Maintaining
- Self-checking

Learned motor patterns

- Controlled by premotor cortex (e.g., climbing stairs)

Volitional eye movements

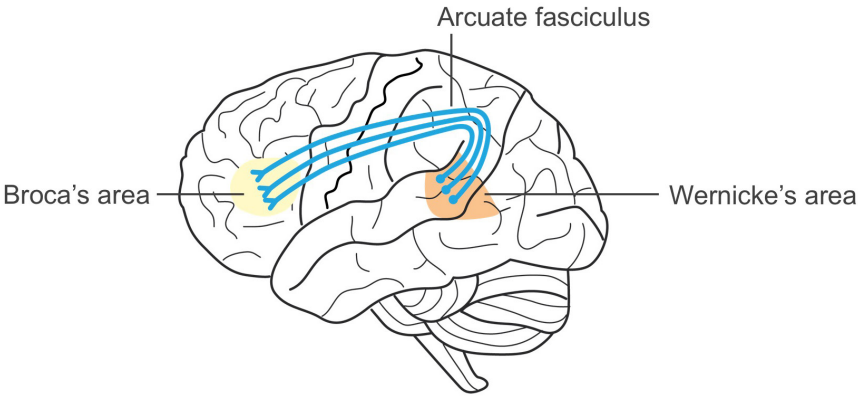
- Involves frontal eye fields
- Activity of right frontal eye field turns eyes toward left
- Stroke (eyes look toward lesion)
- Seizure (eyes look away from lesion)

Stroke

Seizure

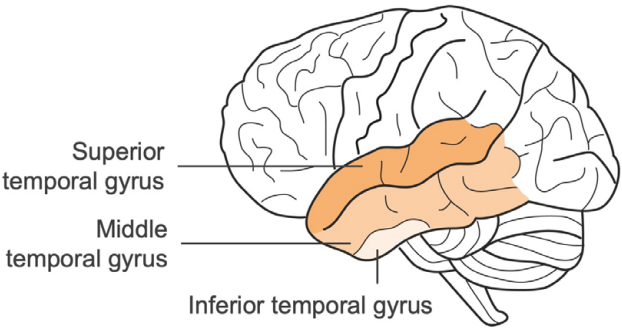
Speech and language

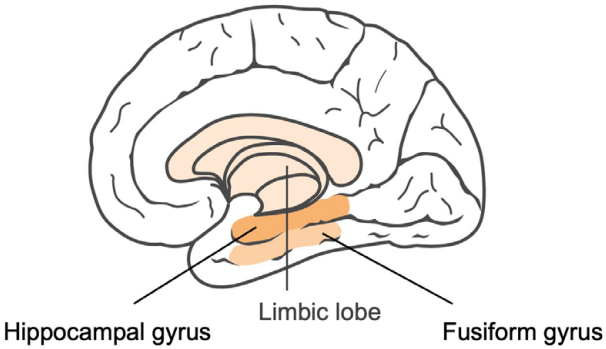
- Broca's area coordinates speech production and couples it with language interpretation through Wernicke's area of temporal lobe
- Broca's aphasia
  - occurs with damage to dominant hemisphere (in all right-handed persons and most left-handed persons the dominant hemisphere is the left)
  - hesitancy in fluidity of speech (often with missing articles, word substitutions, and difficulty repeating)

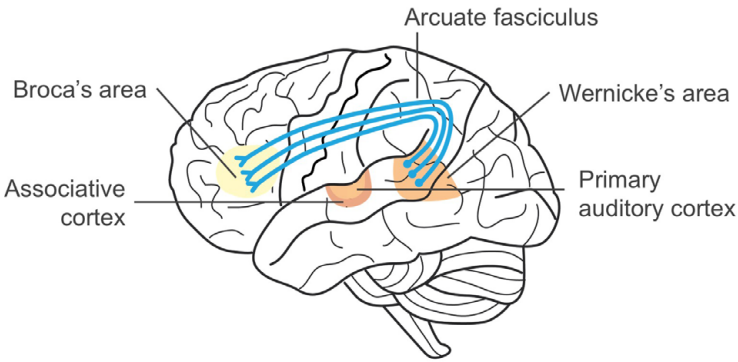


TEMPORAL LOBE

Important regions







Clinicoanatomic correlation

Hearing

- Primary auditory cortex involved in hearing and sound interpretation
- Associative cortex interprets spoken language (also involved in understanding written language)
- Cortical deafness requires bilateral temporal lobe lesions (rare)

Language

- Wernicke's area is important for comprehension of speech
- Wernicke's aphasia
    - occurs with damage to dominant hemisphere
    - fluent speech occurs without meaningful language conveyed
    - often difficulty understanding language
- Associative aphasia
    - occurs with damage to fibers of arcuate fasciculus
    - inability to repeat oral language
    - difficulty writing language

Emotions and memory

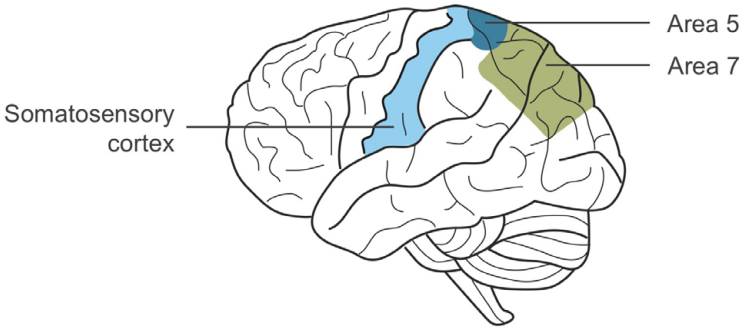
- Mediated by the limbic lobe
- Bilateral lesions of hippocampus and thalamus result in memory loss

Temporal lobe seizure

- Often starts in first or second decade of life
- Involves visual or auditory hallucinations, automatic behaviors, déjà vu or jamais vu, olfactory hallucinations, clouding of consciousness

PARIETAL LOBE

Important regions



Clinicoanatomic correlation

Sensory function

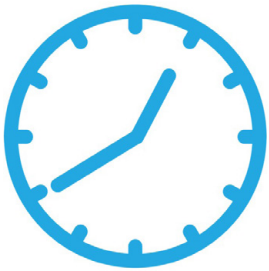
- Mediated by primary somatosensory cortex and associative cortices (Brodmann areas 5 and 7)
- Cutaneous sensibility (pinprick, temperature, light touch, vibration, proprioception)
- Cortical sensibility (two-point discrimination, graphesthesia, stereognosis, double simultaneous stimulation, somatognosia)

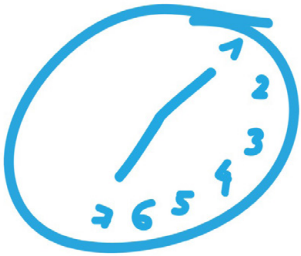
Extinction

- Inability to perceive multiple stimuli—of same type (tactile or visual)—simultaneously

Neglect

- Failure to perceive items on one side of the body





Parietal stroke symptoms

General

- Agraphesthesia
- Loss of two-point discrimination
- Astereognosia
- Extinction

Dominant hemisphere

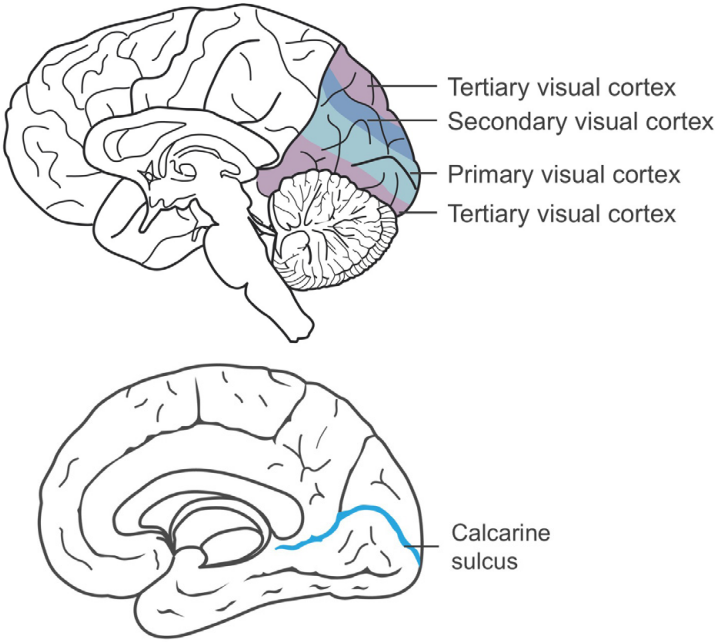
- Right-left confusion
- Finger agnosia
- Acalculia
- Agraphia

Other

- Asomatognosia
- Dressing apraxia
- Constructional apraxia
- Geographic disorientation

OCCIPITAL LOBE

Important regions



Clinicoanatomic correlation

Calcarine cortex

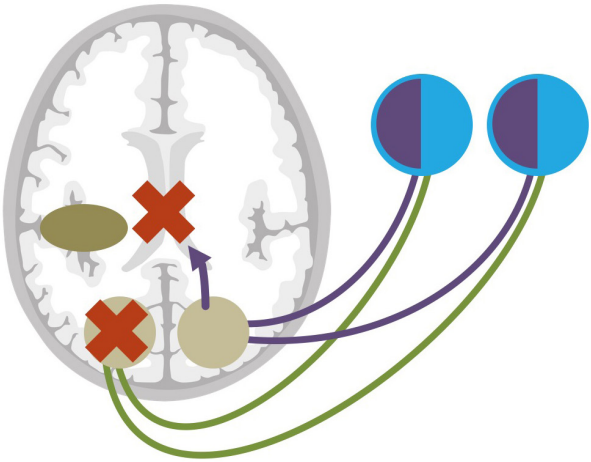
- Located in calcarine fissure
- Important for vision

Cortical blindness

- Requires bilateral occipital lesions
- Anton's syndrome
  - patient doesn't realize they are blind
  - confabulate descriptions

Disconnection syndrome

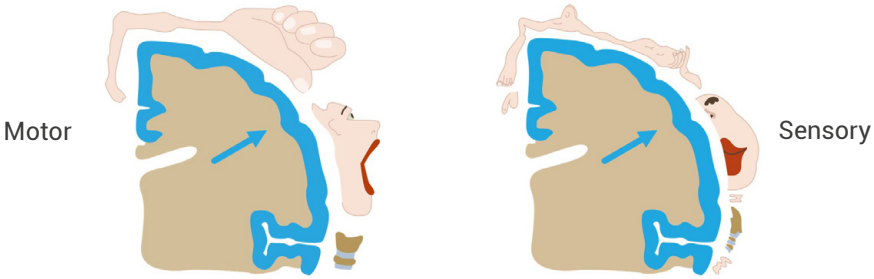
- Occurs with lesion in left visual cortex (if dominant) and corpus callosum
- Loss of input from right visual field
- Corpus callosum lesion prevents transfer of information from right visual field to left parietal language processing regions
- Produces alexia without agraphia
    - patient can see but not read written language
    - ability to write language not affected



INTEGRATED CONCEPTS

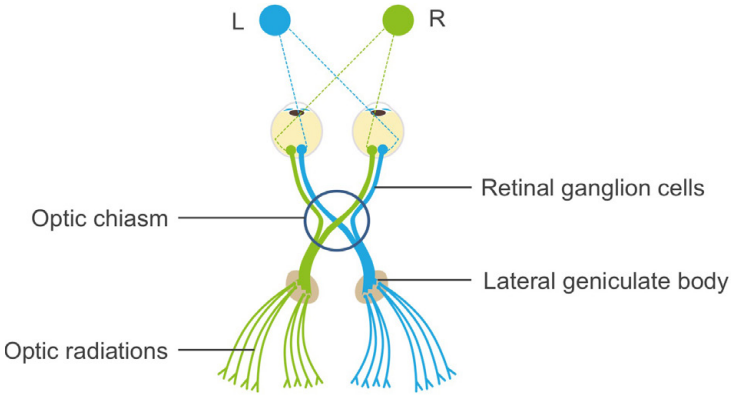
Sensory and motor homunculi

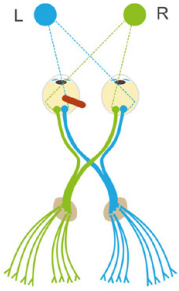
The homunculus is a superimposed diagram of body parts, giving a graphical representation of what motor or sensory function is controlled by the underlying cortical region.



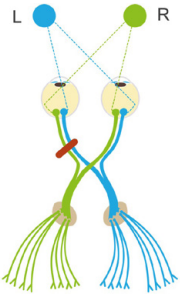
Visual field deficits

Visual field deficits can occur as a result of damage to any part of the visual pathway, from the eyes to the occipital lobe.

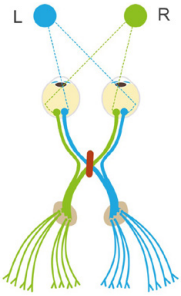




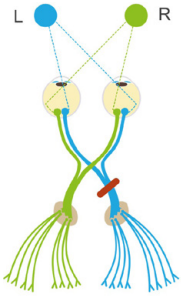
Macula



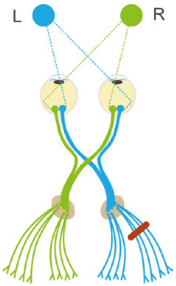
Optic nerve



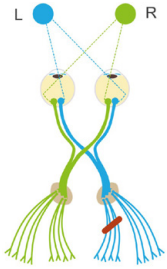
Optic chiasm



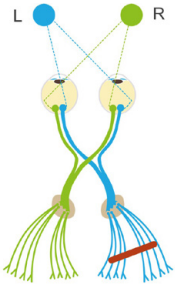
Optic tract



Meyer's loop
(temporal lobe)



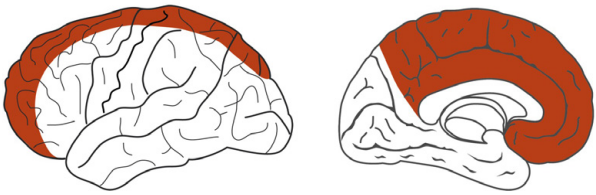
Dorsal optic radiation
(parietal lobe)



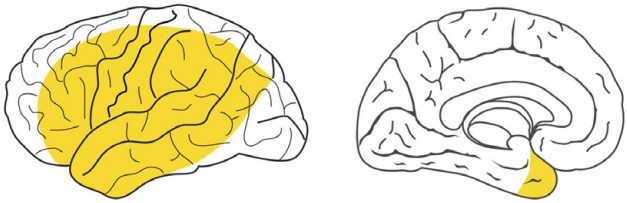
Visual cortex
(parietooccipital region)

BLOOD SUPPLY

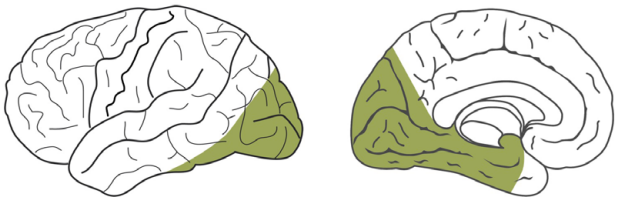
Anterior circulation



Anterior cerebral artery



Middle cerebral artery



Posterior cerebral artery

Anterior cerebral artery stroke

- Sensory-motor deficits
- Contralateral hemiplegia (leg and foot greater than arm and shoulder)
- Head and eyes look toward lesion
- Rigidity

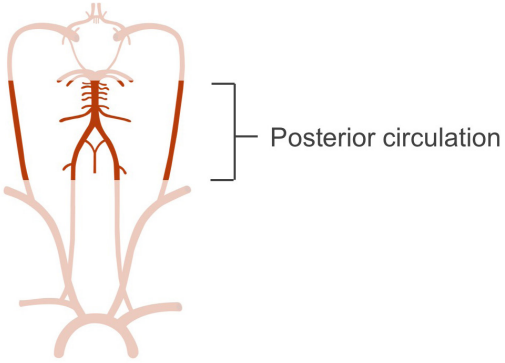
Middle cerebral artery stroke

- Contralateral hemiplegia (arm and shoulder greater than leg)
- Hemianesthesia
- Homonymous hemianopsia
- Eyes look toward lesion
- Aphasia (if dominant hemisphere affected)
- Agnosia and neglect (if non-dominant hemisphere affected)

Posterior cerebral artery stroke

- Affects occipital lobe (including calcarine cortex and optic radiations)
- Homonymous hemianopsia

Posterior circulation



Posterior circulation stroke

- Bilateral / crossed symptoms
- Headache
- Nausea and vomiting
- Vertigo
- Nystagmus
- Weakness
- Extraocular movement abnormalities
- Ataxia

The *D* symptoms of posterior stroke

- Dizziness
- Dysarthria
- Disequilibrium (or dysataxia)
- Deafness
- Diplopia
- Dysphagia
- Dysphonia

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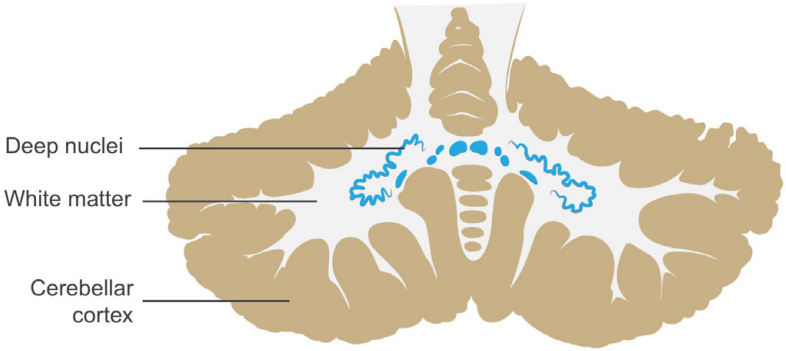
Chapter 3

THE CEREBELLUM AND BASAL GANGLIA

CEREBELLAR ANATOMY

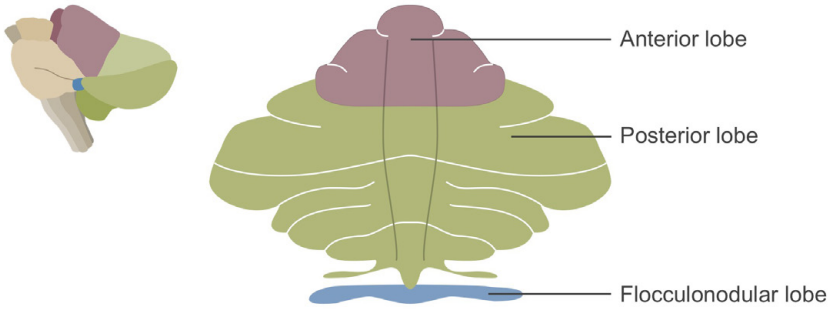
The primary role of the cerebellum is to modulate the descending fibers of the corticospinal tract.

It regulates muscle tone and coordination, and controls posture and gait.



Cerebellar lobes

The cerebellum is divided into three lobes.



Anterior lobe

- Controls posture and muscle tone
- Receives input from the spinal cerebellar tracts
- Damage might produce ataxia of the limbs

Posterior lobe

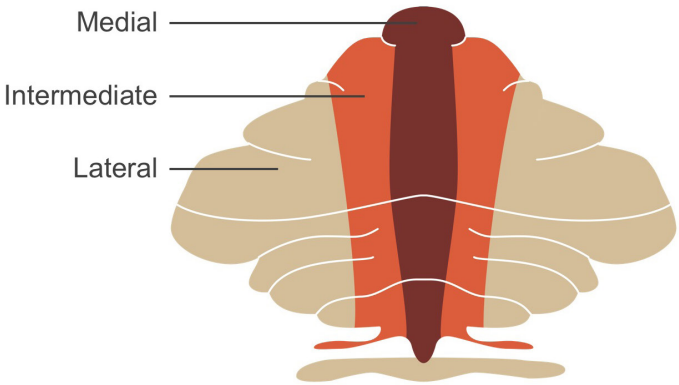
- Controls coordination in skilled activities
- Receives input from the cortex
- Damage produces hypotonia and truncal ataxia

Flocculonodular lobe

- Concerned with equilibrium
- Receives input from the vestibular nuclei
- Damage produces nystagmus, vertigo, and difficulty with equilibrium

Functional regions

The cerebellum can also be divided into three functional zones.



Medial

- Controls proximal and trunk muscles
- Controls the vestibulo-ocular reflexes
- Damage causes gait unsteadiness due to truncal ataxia and nystagmus with vertigo

Intermediate

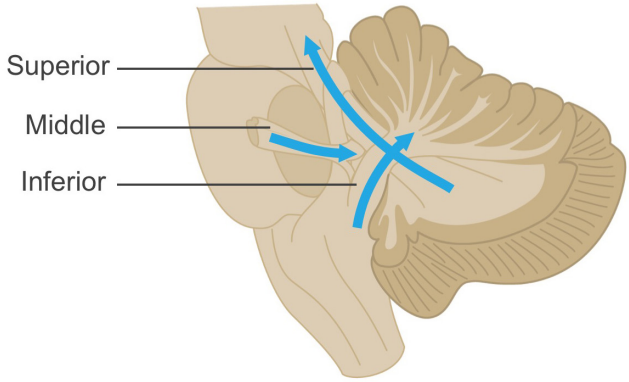
- Controls distal muscles of appendages
- Damage causes appendicular ataxia

Lateral

- Controls motor planning for extremities
- Damage may affect fine motor skills

Cerebellar connections

Three pairs of peduncles—inferior, middle, and superior—connect the cerebellum to other structures of the nervous system.



Cerebellar input

- Mostly through inferior and middle cerebellar peduncles
- Sensory input from various regions in brainstem, midbrain, and spinal cord
- Modulate cerebellar output activity

Cerebellar output

- All output arises from deep cerebellar nuclei
- All cerebellar output is inhibitory
- Output projects through the superior cerebellar peduncle (eventually connecting cerebellum to cortex)
- Several structures work together to modulate cerebellar motor control (based on various sensory inputs)

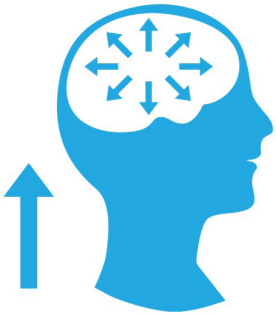
Cerebellar dysfunction

Signs of cerebellar dysfunction

- Ataxia (ipsilateral)
- Decreased muscle tone
- Decreased muscle tendon reflexes (ipsilateral)
- Horizontal nystagmus
- Cerebellar dysarthria
- Intention tremor

COMMON CEREBELLAR AFFLICTIONS

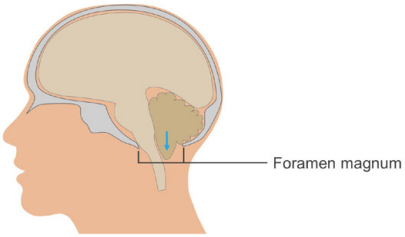
Tonsillar herniation

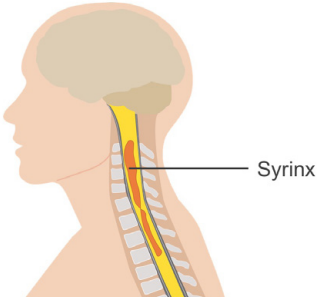


When the intracranial pressure increases diffusely, or in the posterior fossa acutely, herniation of the cerebellar tonsils into the foramen magnum can occur. This compresses the brainstem, particularly the medulla, and produces coma, respiratory arrest, and finally death.

Chiari's malformation

Chiari type I malformation is associated with displacement of the cerebellar tonsils downward, below the level of the foramen magnum. In more advanced cases, an enlargement of the central canal of the spinal cord, called a syrinx, develops.





Common symptoms

- Posterior headache pain
- Elevated intracranial pressure
- Signs of spinal cord dysfunction (especially when syrinx present)
- Dizziness
- Hearing loss
- Swallowing dysfunction
- Speech changes (e.g., dysarthria)
- Ataxia
- Vertigo
- Downbeating nystagmus (in the primary forward gaze position)

Toxins

Ethanol

- Acute effects cause postural instability
- Long-term neurotoxic effects damage vermis (leading to truncal ataxia and gait instability)
- Thiamine deficiency may also contribute to cerebellar damage

Neoplasms

Common cerebellar tumors

- Medulloblastoma (in children)
- Astrocytomas
- Metastatic tumors
- Meningiomas

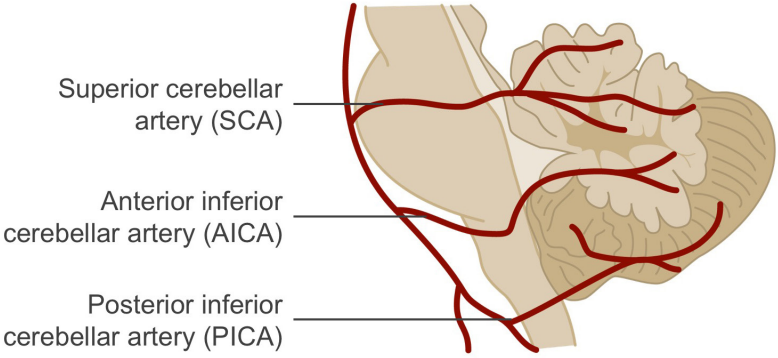
Signs associated with cerebellar tumors

- Headache
- Nausea and vomiting
- Blurred or double vision
- Problems with balance and coordination
- Gait disturbance
- Urinary incontinence
- Changes in personality
- Cognitive changes or memory loss

Signs associated with cerebellar tumors in children

- Increased head circumference
- Developmental delays
- Lethargy
- Drowsiness
- Irritability
- Sunsetting of eyes

Cerebellar stroke



Signs of AICA / SCA strokes

- Appendicular ataxia
- Nystagmus
- Nausea and vomiting

Signs of PICA stroke (Wallenberg's stroke syndrome)

- Nausea and vomiting
- Nystagmus
- Vertigo
- Pinprick sensory loss of face (ipsilateral) and body (contralateral)

Other pathologies

Hypertensive hemorrhage

- Can produce a rapid increase in intracranial pressure (leading to tonsillar herniation syndrome and death)

Degenerative disease

- Friedreich's ataxia
- Olivopontocerebellar atrophy (OPCA) / multiple system atrophy (MSA)

THE EXTRAPYRAMIDAL SYSTEM

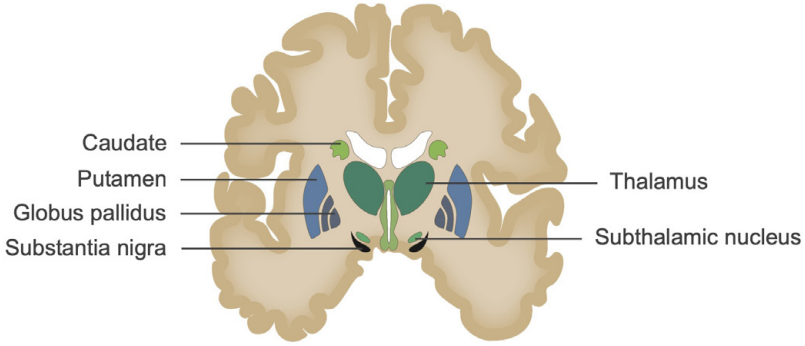
The concept of pyramidal versus extrapyramidal motor systems is an artificial construct. All direct movement outflow comes from the pyramidal system (i.e., the corticospinal tract).

The **pyramidal system** activates muscle movement by sending signals through the corticospinal tract.

The **extrapyramidal system** is more of a conductor for how movement is orchestrated, because it modulates all corticospinal tract functions. The connections are indirect, but they have a significant impact on motor activity.

The extrapyramidal system is made up of the connections of the **cerebellum** and the **basal ganglia**.

Structures of the basal ganglia

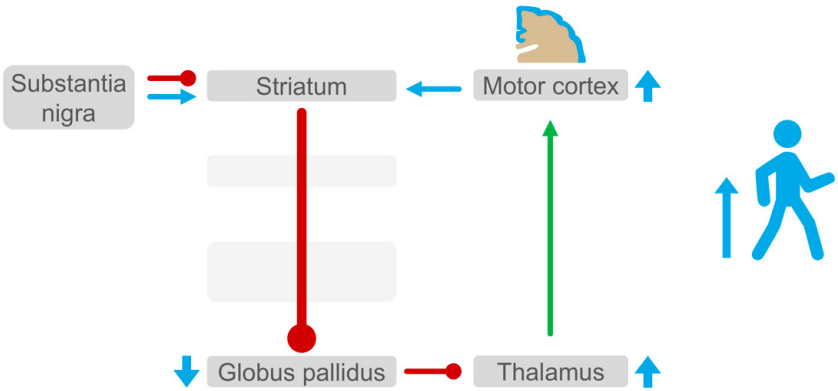


Note that the caudate nucleus and putamen together make up the corpus striatum. Although the thalamus is in the vicinity and intricately connected, it is not considered part of the basal ganglia.

Motor pathways

There are two pathways in the basal ganglia made up of interconnections, which form a series of feedback loops, all of which modulate motor activity.

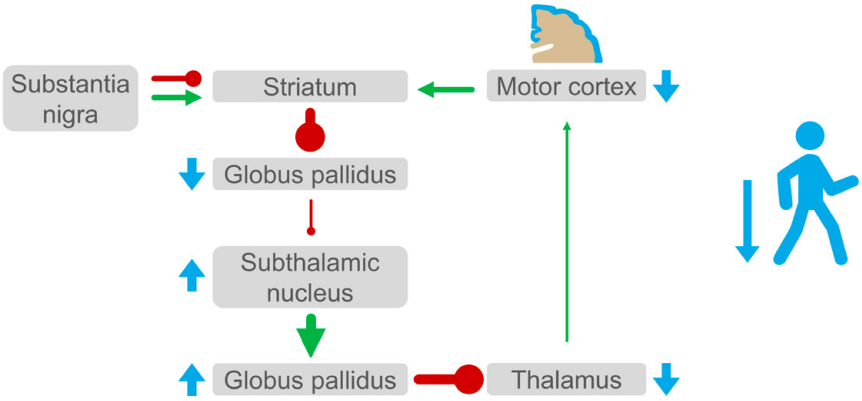
Direct pathway



Output from the globus pallidus internus inhibits the activity of the thalamus and motor cortex, producing an ever-present tendency towards decreased motor activity.

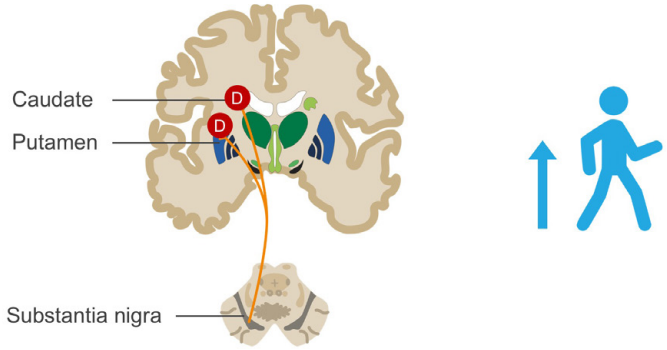
In the direct pathway, the striatum lessens the activity of the globus pallidus interna, removing this brake from the system, and resulting in increased motor activity, through an effective release of the thalamic drive.

Indirect pathway



In the indirect pathway, the striatum inhibits the globus pallidus externa, which in turn reduces inhibition of the subthalamic nucleus. Increased activity of the subthalamic nucleus further strengthens the inhibitory activity of the globus pallidus interna, resulting in reduced thalamic and cortical activity, which leads to an overall decrease in motor activity.

The dopaminergic system



Neurons from the substantia nigra project to the striatum, where they release the neurotransmitter **dopamine**. The striatum and the substantia nigra have connections in both directions. It is through these that the release of dopamine in the striatum releases the inhibitions on movement.

Another neurotransmitter, **acetylcholine**, is also produced by neurons in the substantia nigra, and released in the striatum. Acetylcholine has the opposite effect as dopamine, and tends to inhibit movement. Thus, acetylcholine and dopamine are antagonistic and are normally balanced.

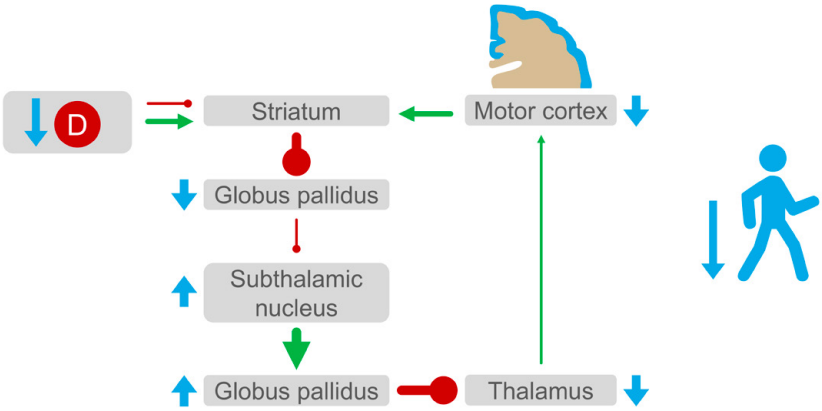
EXTRAPYRAMIDAL DISORDERS

Hypokinetic movement disorders

Hypokinetic movement disorders are characterized by too little movement. Classic examples are Parkinson's disease and parkinsonism.

Parkinson's disease

Parkinson's disease results from loss of dopamine producing neurons in the substantia nigra in the midbrain.



The indirect pathway generally results in decreased motor activity. With the loss of dopamine from the substantia nigra, there is an excess of activity of the indirect pathway within the basal ganglia, resulting in excess globus pallidus interna activity suppressing the thalamocortical tracts, which normally promote activation of the motor neurons and thus the corticospinal tract. Therefore, inhibition of movement results.

Classic symptoms of Parkinson's disease

- Rigidity (with cogwheeling)
- Resting tremor (pill-rolling characteristic)
- Postural tremor
- Bradykinesia (e.g., reduced blink rate)
- Postural instability
- Festination (Parkinsonian gait)

Examining for Parkinson's disease

- Observe movements (particularly gait and face)
- Observe for 4–8 Hz pill-rolling tremor at rest
- Elicit cogwheeling rigidity by moving a limb at two joints
- Test for retropulsive tendencies with pull test

Hyperkinetic movement disorders

Hyperkinetic movement disorders are characterized by excessive movement. Classic examples include Huntington's disease and Tourette's syndrome.

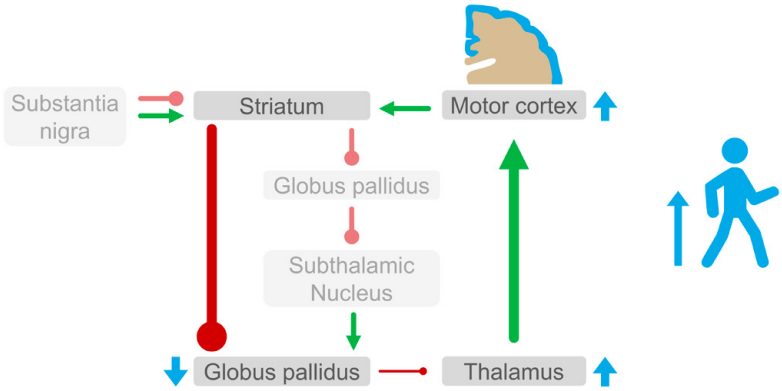
Common symptoms of hyperkinetic disorders

- Chorea
- Ballism
- Tics
- Dystonias

Huntington's disease

Huntington's disease is a genetic disease with an insidious onset, characterized by involuntary choreaformic movements and cognitive changes with psychiatric manifestations.

The pathology involves the loss of neurons in the caudate and putamen.



The disease results from a loss of striatal neurons, which leads to favoring of the direct pathway, thus facilitating motor activity of the cortex. Loss of inhibition in the globus pallidus results in increased thalamocortical motor enhancement, resulting in chorea.

Further reading

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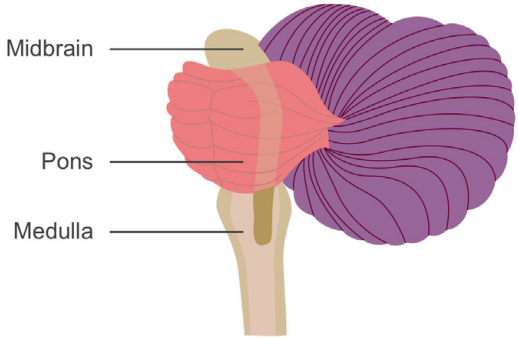
Chapter 4

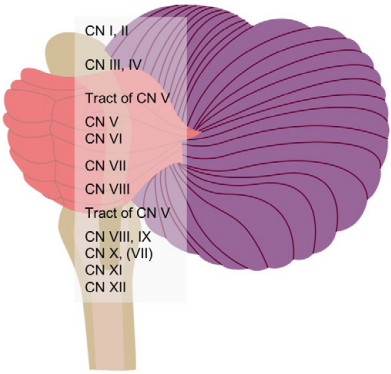
THE BRAINSTEM

ANATOMY OF THE BRAINSTEM

The brainstem is organized into three regions—the medulla, pons, and midbrain.

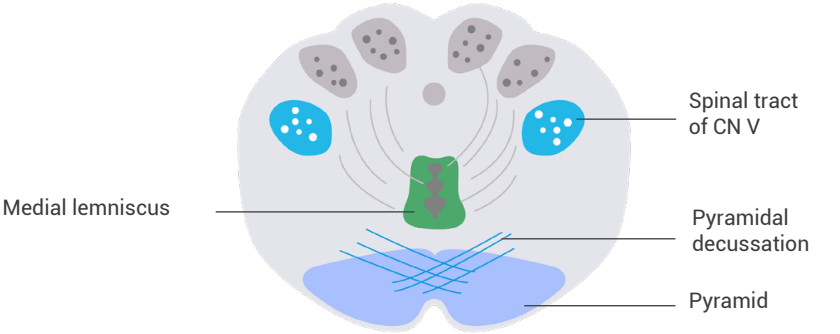


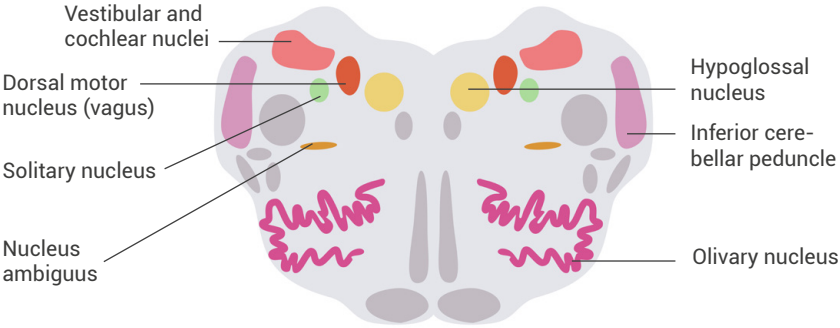
The brainstem also contains the cranial nerve nuclei. The locations within each region of these nuclei is shown in the diagram below.



THE MEDULLA

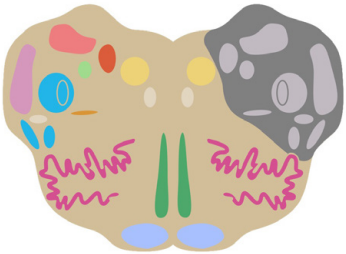
Anatomy





Lateral medullary syndrome (Wallenberg's syndrome)

This syndrome, which affects the lateral medulla, commonly results from stroke affecting the posterior inferior cerebellar artery (PICA) or its branches.



Lesion involves

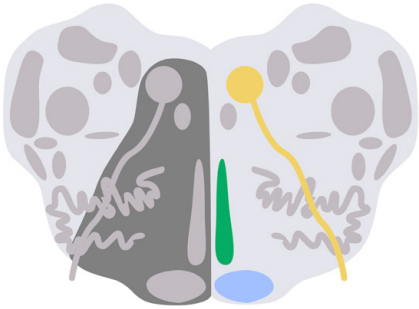
- Inferior cerebellar peduncle
- Vestibular nuclei
- Fibers or nuclei of cranial nerves IX and X
- Spinal nucleus and tract of cranial nerve V
- Spinothalamic tract
- Sympathetic pathways

Symptoms

- Contralateral loss of pain and temperature sensation in body
- Ipsilateral loss of pain and temperature sensation in face
- Dysphagia
- Dysphonia
- Decreased gag reflex
- Vertigo
- Nystagmus
- Ipsilateral ataxia
- Diplopia
- Ipsilateral Horner's syndrome

Medial medullary syndrome

Occlusion of the anterior spinal artery or vertebral artery can lead to medial medullary syndrome.



Lesion involves

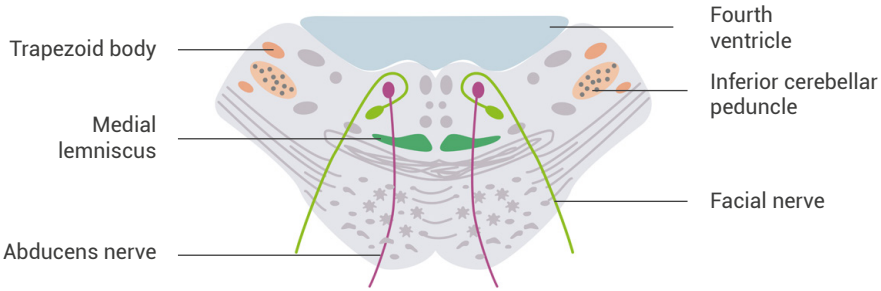
- Medial lemniscus
- Hypoglossal nerve (cranial nerve XII)
- The pyramid (corticospinal tract)

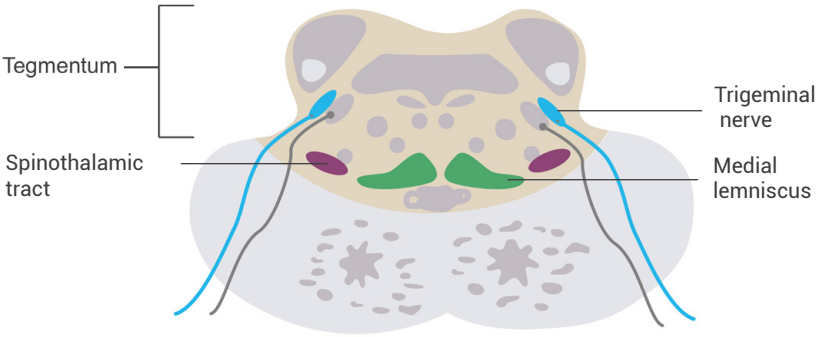
Symptoms

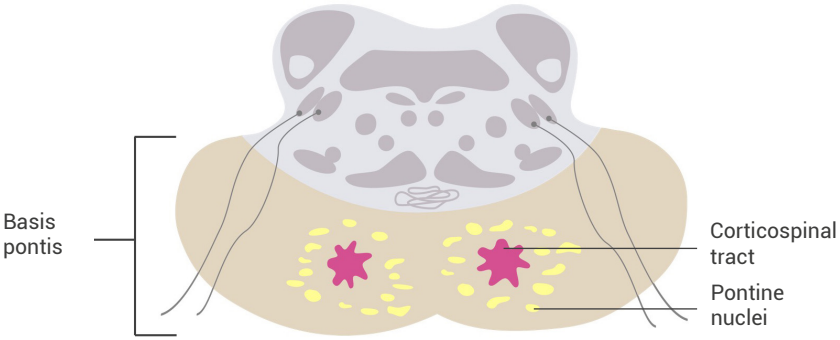
- Contralateral body paralysis
- Ipsilateral tongue weakness (tongue deviation away from weakness)
- Contralateral hemianesthesia of body (sparing the face)

THE PONS

Anatomy







Pontine syndromes

Pontine syndromes are usually the result of small vessel lacunar strokes, where the lesions are generally less defined and can occur in differing regions of the long axis of the pons from cranial to caudal ends. They involve perforating branches of the basilar artery into the pons.

Lesions can involve

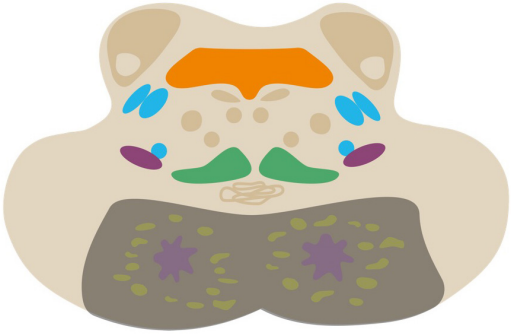
- Corticospinal tract
- One or more cranial nerves (either V, VI or VII)

Symptoms

- Contralateral hemiplegia
- Contralateral hemianesthesia (large lesions)

Locked-in syndrome

Basilar artery occlusion can lead to a large lesion in the basis pontis, resulting in locked-in syndrome.



Lesion involves

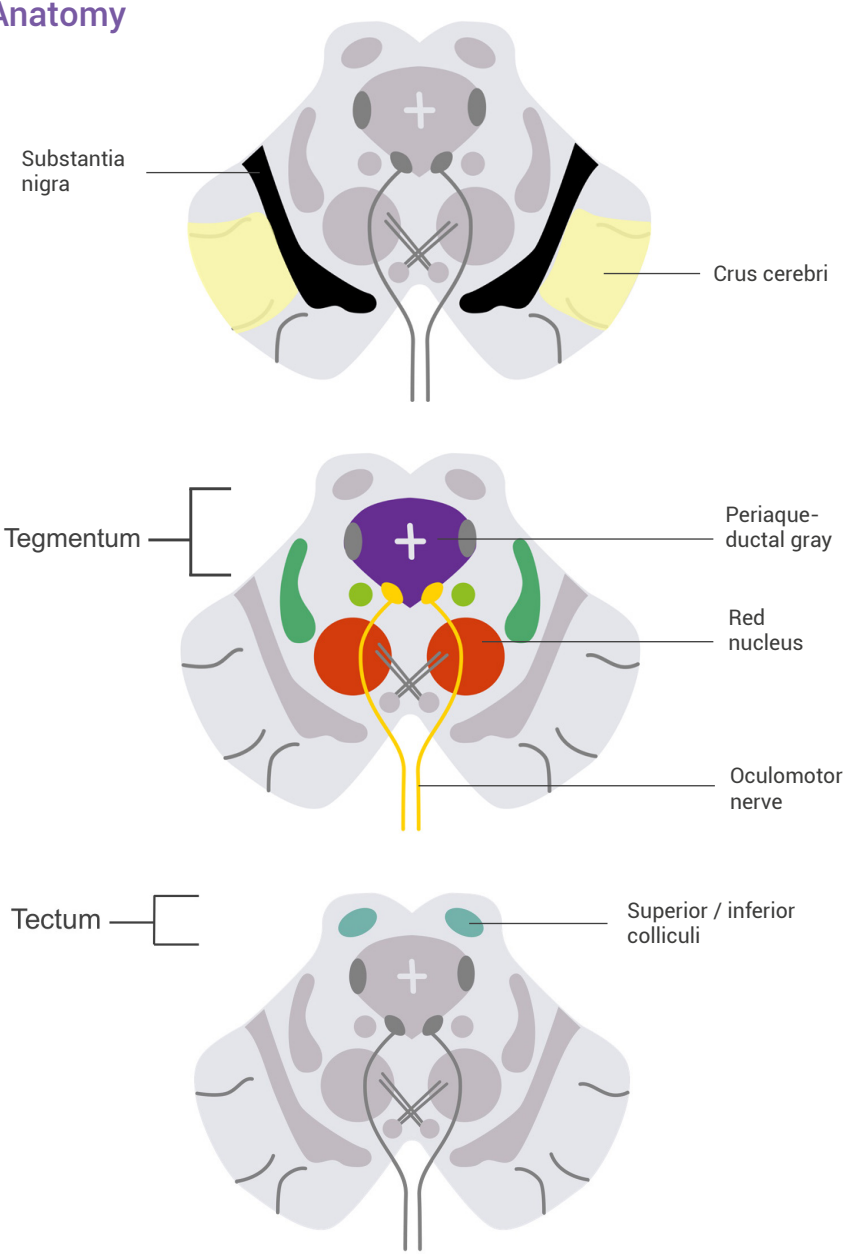
- Corticospinal tracts
- Corticobulbar tracts

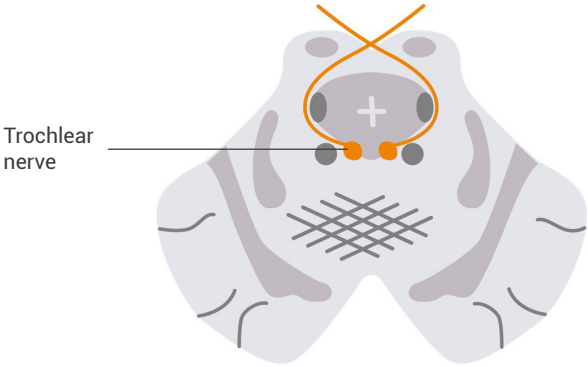
Symptoms

- Impaired speech
- Loss of facial movement
- Quadriplegia
- Patient is awake and aware (only able to move eyes)

THE MIDBRAIN

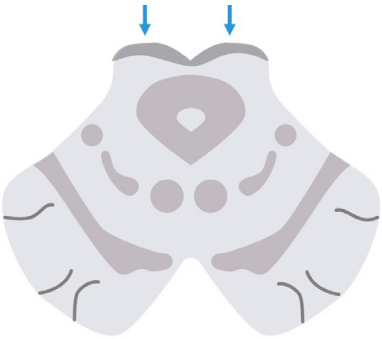
Anatomy





Parinaud's syndrome

Dorsal midbrain syndrome, also known as Parinaud's syndrome, occurs with compression of the tectum, usually as the result of a mass, such as a pineal tumor.



Lesion affects

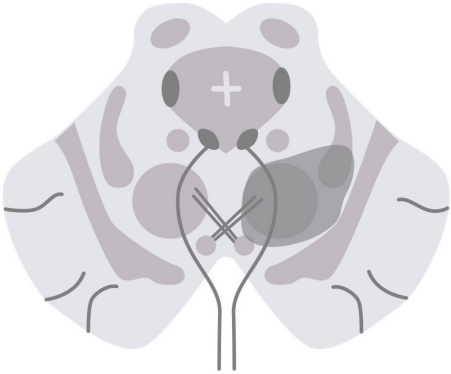
- Inferior / superior colliculi

Symptoms

- Paralysis of vertical gaze
- Convergence-retraction nystagmus
- Downbeat nystagmus
- Mid-pupillary dilation (with loss of reactivity)

Benedikt's syndrome

Benedikt's syndrome results from a lesion involving the midbrain tegmentum. This most commonly results from vascular, inflammatory or mass lesions.



Lesion affects

- Oculomotor nucleus (CN III)
- Red nucleus

Symptoms

- Ipsilateral oculomotor palsy
- Contralateral ataxia
- Intention tremor

Weber's syndrome

Weber's syndrome can result from some masses and strokes.

Lesion affects

- Cerebral peduncle
- Oculomotor nerve (CN III)

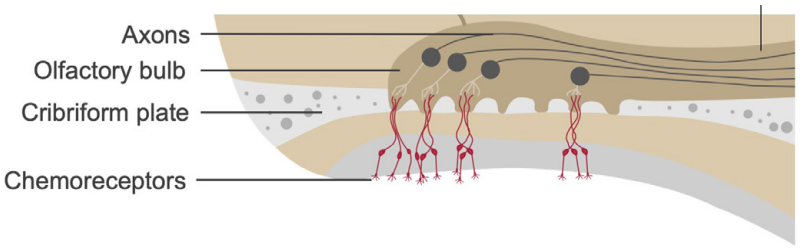
Symptoms

- Contralateral hemiplegia
- Ipsilateral oculomotor palsy

OLFACTORY NERVE (CN I)

Function

- Smell



Testing CN I

- Use common scents (e.g., spices, mint flavored toothpaste or chewing gum)
- Avoid using volatile chemicals

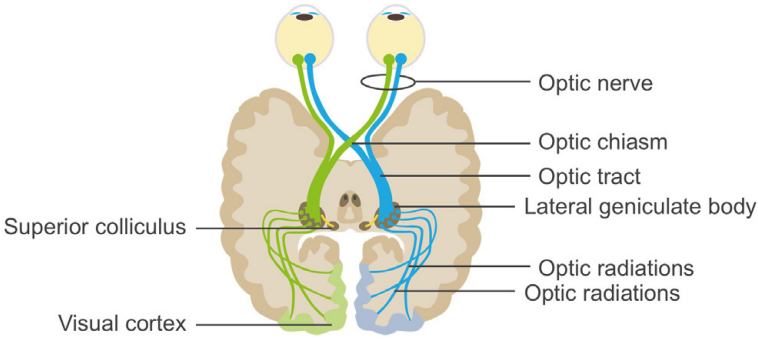
Common pathology

- Head injury / blunt force trauma
- Subfrontal or olfactory groove meningioma
- Parkinson's disease hyposmia

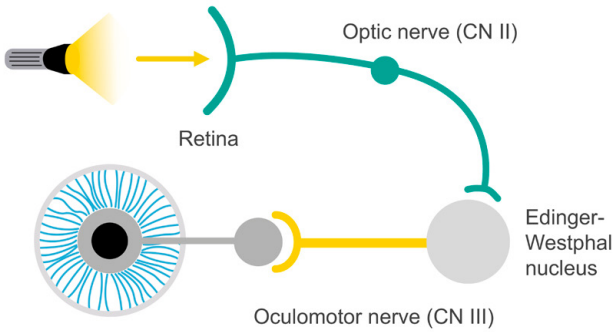
OPTIC NERVE (CN II)

Functions

- Vision
- Efferent pathway for pupillary light reflex



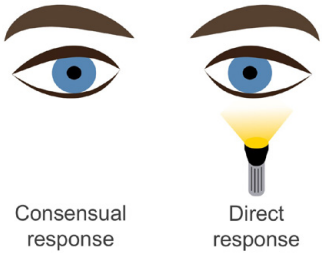
Visual pathways



Pupillary light reflex

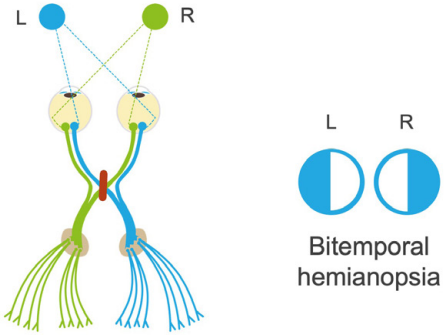
Testing CN II

- Pupillary light reflex
  - examine pupillary size and reactivity
  - look for direct and consensual responses
- Fundoscopic examination
- Visual acuity



Common pathology

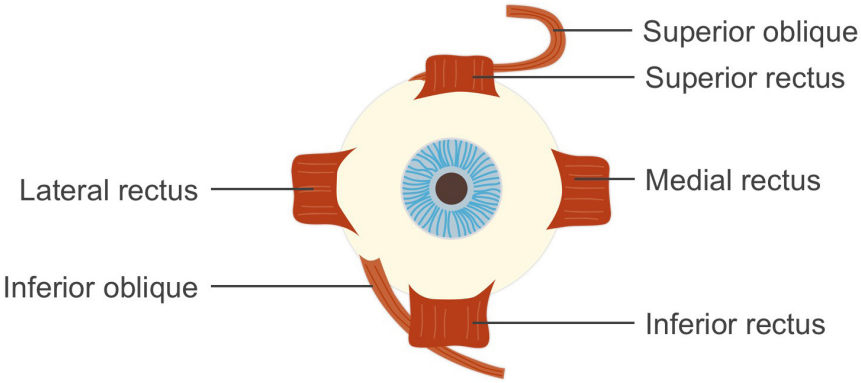
- Optic neuritis
  - inflammation of optic nerve (damages myelin)
  - symptoms
    - blurred vision
    - eye pain
    - afferent pupillary defect
- Chiasmal lesion
  - produces bitemporal hemianopsia (e.g., pituitary tumor)



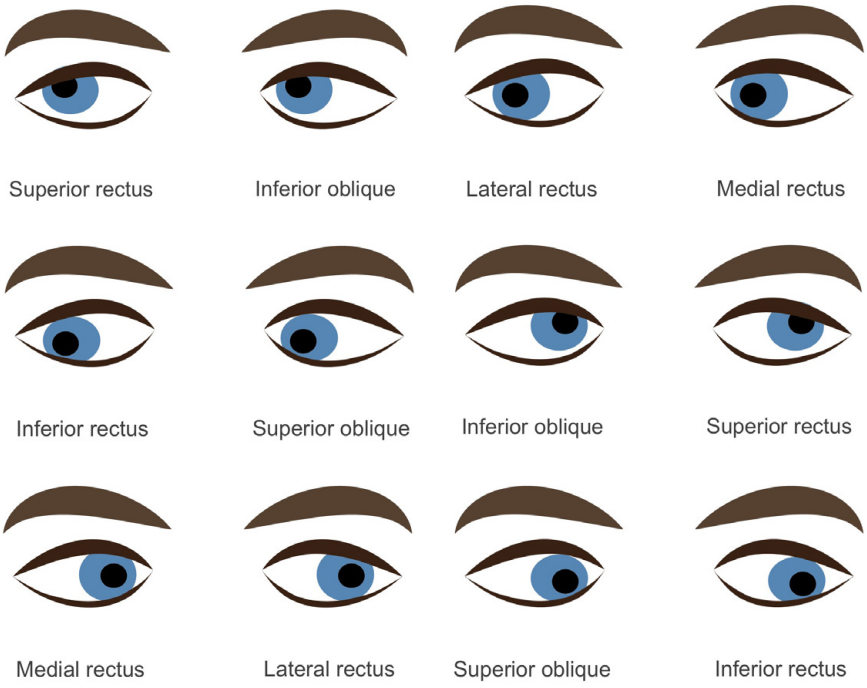
OCULOMOTOR, TROCHLEAR, AND ABDUCENS NERVES (CN III, IV, AND VI)

Functions

- Extraocular eye movements
  - innervate superior, medial, and inferior rectus (CN III)
  - innervate superior oblique (CN IV)
  - innervate lateral rectus (CN VI)
  - innervate levator palpebrae (CN III)
- Efferent pathway for pupillary light reflex (CN III)



Muscular contributions to eye movements



Testing the eyes

- Cardinal planes of view
  - move through each plane (stopping for 1–2 seconds before bringing gaze back to center)
- Observe for
  - conjugate movement
  - nystagmus
  - pupillary constriction during accommodation
- Red glass test
- Pupillary light reflex

Common pathology of CN III

- Diabetic oculomotor palsy
    - small vessel infarcts damage internal parasympathetic fibers
    - ptosis and abducted eye (with pupil sparing)
- Posterior communicating artery aneurysm
    - compression of superficial nerve fibers
    - symptoms
        - pupillary dilation
        - diplopia
- Uncal herniation
    - increased intracranial pressure causes uncus to bulge into and between falx cerebri (compressing the nerve)
    - symptoms
        - pupillary dilation
        - oculomotor palsy (ptosis and abducted eye)
        - coma
- Stroke
- Myopathies

Common pathology of CN IV

- Closed head injuries
- Aneurysm
- Infection
- Neoplasm

General symptoms of CN IV damage

- inability to gaze down and in toward nose (eye deviated up and out)
- vertical diplopia

Common pathology of CN VI

- Orbital trauma
- Cavernous sinus thrombosis
- Cerebellopontine angle tumors

Common eye movement abnormalities

Dysconjugate gaze

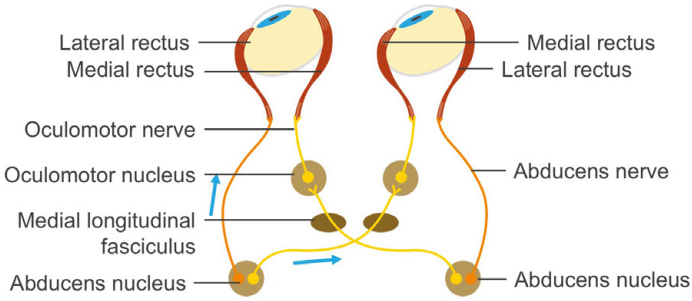
- Eyes don't move synchronously
- Produces double vision

Vertical gaze

- Superior and inferior rectus muscles
- Controlled by oculomotor nerve (CN III)
- Coordinated movement involves superior colliculus
- Altered in Parinaud's syndrome (pressure on superior colliculus)

Horizontal gaze

- Involves coordination between CN III and CN VI (innervating medial and lateral rectus muscles respectively)
- Involves coordination through the medial longitudinal fasciculus (MLF)
- Affected in internuclear ophthalmoplegia
- Isolated lesion of medial longitudinal fasciculus (MLF)
    - normal abduction but inability to adduct
    - nystagmus in adducting eye
    - convergence is normal



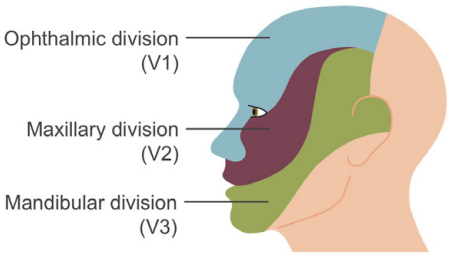
Vestibular influences on gaze

- CN VIII sends data from vestibular nuclei into MLF
  - helps keep eyes stable with head turning
- Cortical stroke
  - patient looks toward lesion
- Brainstem stroke
  - patient looks away from lesion
- Testing vestibular reflex
  - oculocephalic maneuver
  - ice water cold caloric testing

TRIGEMINAL NERVE (CN V)

Functions

- Sensation in the face
  - light touch
  - pain
  - temperature
  - vibration
- Motor
  - muscles of mastication
  - tensor tympani muscle
- Efferent pathway for corneal reflex



Testing CN V

- Sensory examination
  - assess light touch, pinprick, and temperature appreciation in all divisions
  - corneal reflex
- Motor examination
- Look for wasting and atrophy
- Jaw jerk reflex

Common pathology of CN V

- Facial paresthesia
- Cerebellopontine angle tumors
- Cavernous sinus thrombosis
- Zoster ophthalmicus
- Trigeminal neuralgia

FACIAL NERVE (CN VII)

Functions

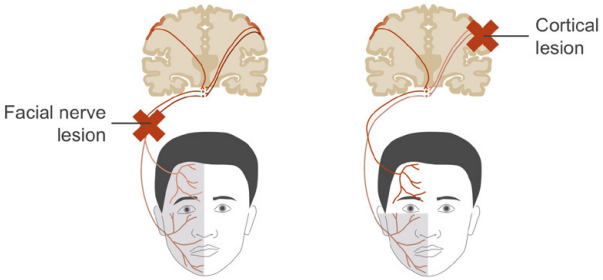
- Muscles of facial expression
- Stapedius
- Taste for anterior two-thirds of tongue
- Sensation on external auditory canal
- Tearing, salivation, mucus production

Testing CN VII

- Observe general motility and facial expression
- Observe nasolabial folds and palpebral fissures
- Look for symmetrical movement and strength when patient smiles, shows teeth, whistles, and closes eyes tight
- Observe for Bell's phenomenon

Common pathology of CN VII

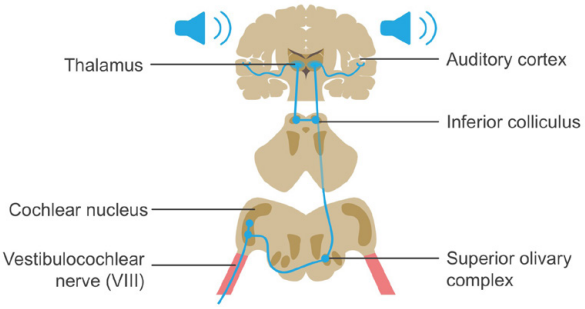
- Bell's palsy
- Peripheral nerve lesion
- Weakness of upper and lower face
- Ramsay Hunt syndrome
- Pontine stroke syndrome



VESTIBULOCOCHLEAR NERVE (CN VIII)

Functions

- Hearing
- Balance



Testing CN VIII

- Auditory testing
  - whisper, ticking, finger scratch
  - Weber / Rinne tests
    - distinguish conduction deficits from sensorineural loss
- Vestibular testing
  - nystagmus
  - gait
  - bedside tests (e.g., Dix-Hallpike maneuver, head impulse test)

Common pathology of CN VIII

- Cerebellopontine angle tumors
- Deafness
  - conduction deficits
  - sensorineural loss
- Vertigo
- Central (brainstem / cerebellum)
- Peripheral (labyrinthine)

GLOSSOPHARYNGEAL AND VAGUS NERVES (CN IX AND X)

Functions of CN IX

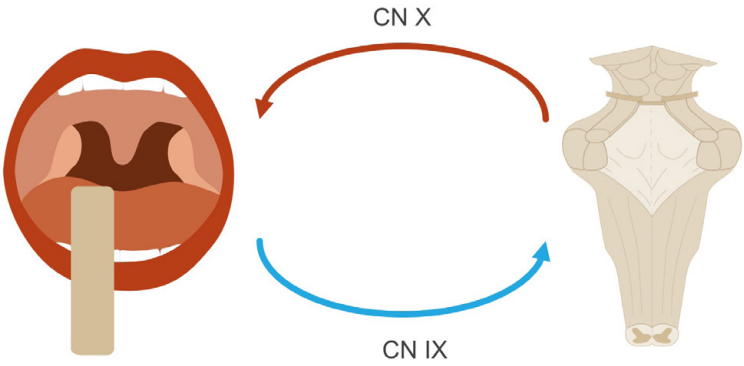
- Taste and sensation to posterior third of tongue
- Sensation to pharynx and external ear
- Swallowing
- Salivation (parotid gland)
- Afferents from carotid sinus baroreceptors and carotid body chemoreceptors
- Afferent loop of gag reflex

Functions of CN X

- Speech production
- Swallowing
- Taste
- Elevation of palate
- Parasympathetic regulation of heart and abdominal viscera
- Somatic sensory innervation of neck, thorax, and viscera
- Efferent loop of gag reflex

Testing CN IX and X

- Test gag reflex
  - uvula deviation away from side of lesion
  - palatal paresis
- Assess for hoarseness or weak cough
- Carotid sinus reflex



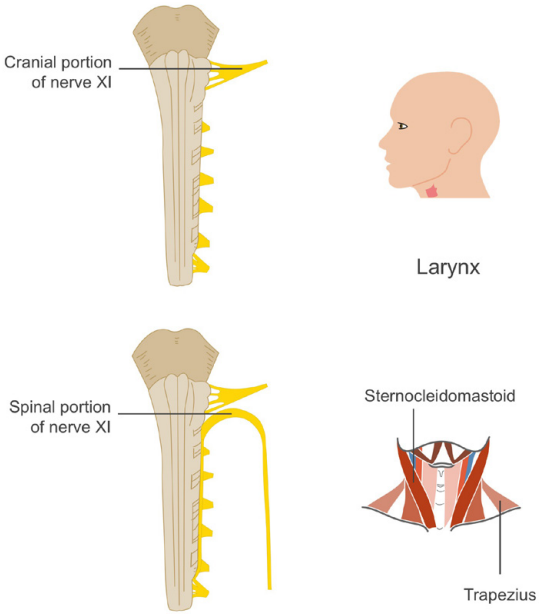
Common pathology of CN IX and X

- Glossopharyngeal neuralgia
- Cerebellarpontine angle tumors
- Lesions in the jugular foramen

SPINAL ACCESSORY NERVE (CN XI)

Function

- Motor innervation
  - larynx (recurrent laryngeal nerve)
  - sternocleidomastoid
  - trapezius



Testing CN XI

- Weakness of sternocleidomastoid and / or trapezius

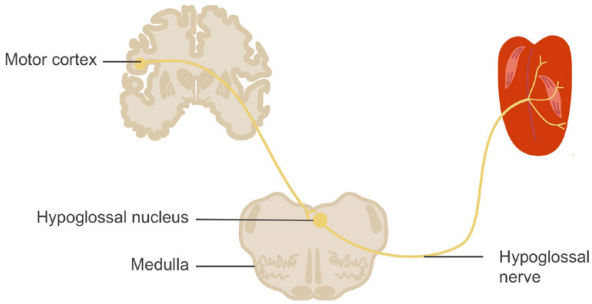
Common pathology of CN XI

- Compression around jugular foramen (e.g., tumor)
- Neck surgery (damage to recurrent laryngeal nerve causing hoarseness)

HYPOGLOSSAL NERVE (CN XII)

Function

- Tongue movement



Testing CN XII

- Have patient protrude tongue
  - observe for atrophy and deviation (toward side of weakness)
  - hold for 15–30 seconds to test for impersistence
- Observe tongue in mouth (with mouth open)
  - look for fasciculations
  - have patient push against your hand—on their cheek—to test strength

Common pathology of CN XII

- Stroke
- Tumor
- Motor neuron disease
- Meningeal disease
- Huntington's disease
- Cerebellar disease
- Essential tremor

Further reading

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Chapter 5

THE SPINAL CORD

SPINAL CORD ANATOMY

The spinal column or vertebral column encases and protects the spinal cord.

Vertebrae

- 7 cervical
- 12 thoracic
- 5 lumbar
- 5 sacral

Spinal nerves

- 8 cervical
- 12 thoracic
- 5 lumbar
- 5 sacral

Note that the cord only extends down to the L2 vertebral body. Nerve roots exit many levels below this and the roots travel down to exit at the appropriate level.

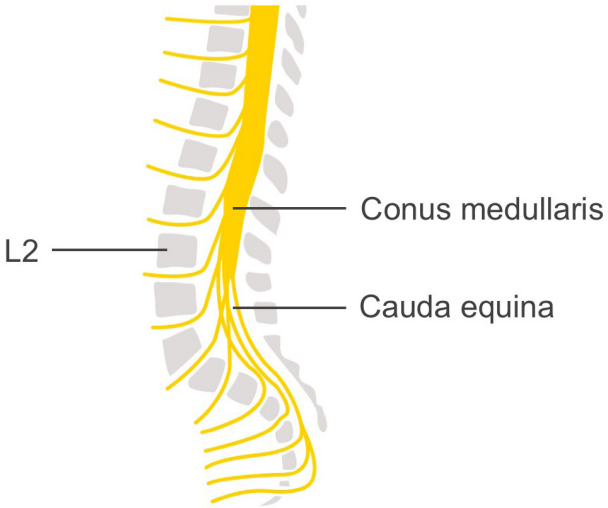
This extension of spinal roots collectively is known as the **cauda equina** or the horse's tail.

Thus, because of the discrepancies in the length of the cord and the spinal column, you should be aware that the segments of the cord do not always match the vertebral body (see chart below).

Corresponding relationship between spinal segments and vertebrae

Spinal segments	Vertebrae bodies
C1–C4	C1–C4
C5–C8, T1–T4	C4–T3
T5–T8	T3–T6
T9–T12	T6–T9
L1–L5	T10–T12
S1–S5, Co1	L1

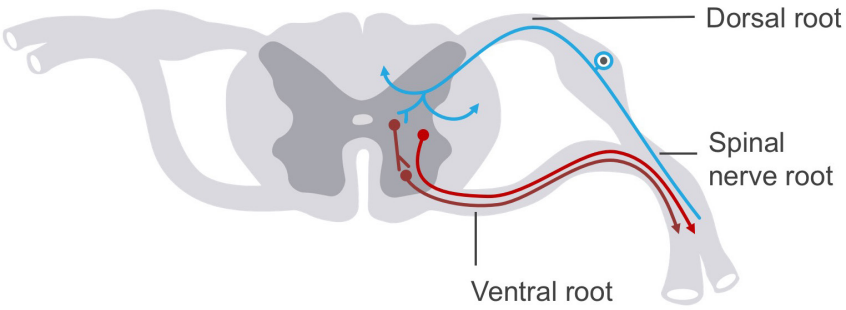
The termination of the spinal cord is known as the **conus medullaris**. This region is located behind L2. The conus contains all the segments of the sacral spinal cord.



Afferent (or sensory) input to the nervous system arrives in the spinal cord via the dorsal root.

Efferents (or motor output) exit via the ventral root.

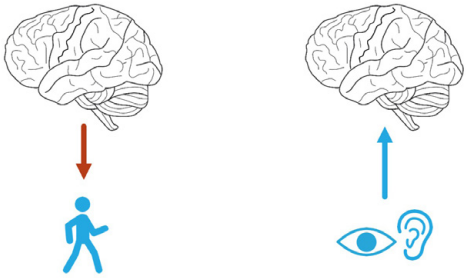
Gray matter is present centrally, and contains neuron cell bodies. The **anterior horn** contains the cell bodies of motor neurons (anterior horn cells). The **lateral horn** contains cell bodies of autonomic neurons, which leave the cord through the ventral root. The **posterior horn** contains cell bodies of sensory interneurons. The cell bodies of sensory neurons bringing information in through the dorsal root, are found outside of the spinal cord, in the **dorsal root ganglion.**



The axons in the peripheral **white matter** are divided into tracts, which represent a set of axons all carrying a similar type of information.

WHITE MATTER TRACTS

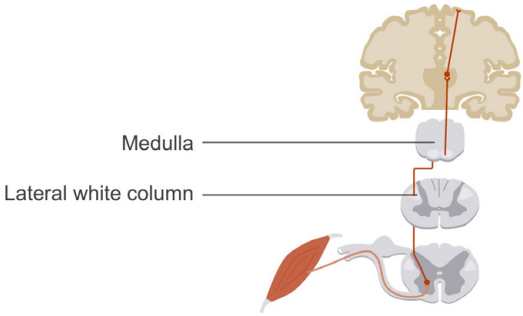
White matter tracts can be categorized as either **descending** (carrying motor information from the brain to the periphery), or **ascending** (carrying sensory information from the periphery to the brain).



Descending tracts

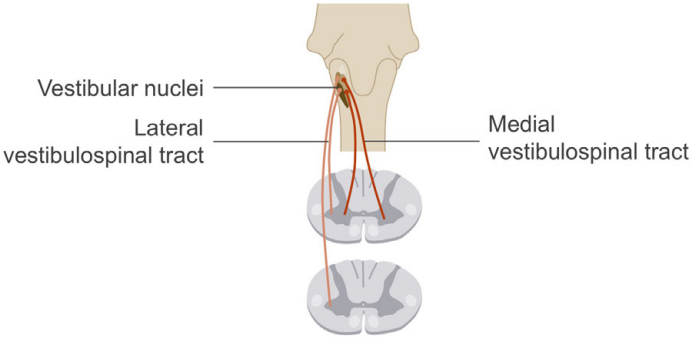
Corticospinal tract

- Carries motor information from motor cortex to spinal cord
- Crosses in the medulla (pyramidal decussation)
- Axons makeup the upper motor neurons
- Synapse with anterior horn cells (lower motor neurons)
- Left brain controls right body and right brain controls left body



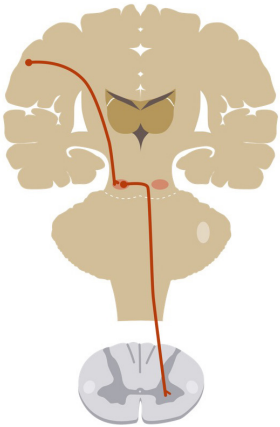
Vestibulospinal tracts

- Carry information from vestibular nuclei in brainstem to spinal cord
- Important for rapid correction of neck movement and posture (in response to changing body position)



Rubrospinal tract

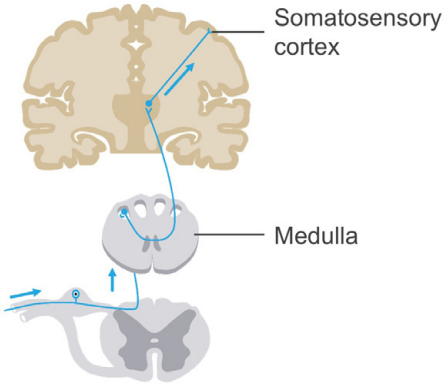
- Carries information from red nucleus to anterior horn cells
- Modulates voluntary motor movements
- Fibers cross before descending in lateral columns

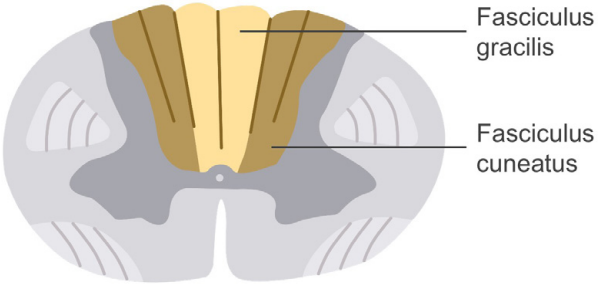


Ascending tracts

Posterior columns

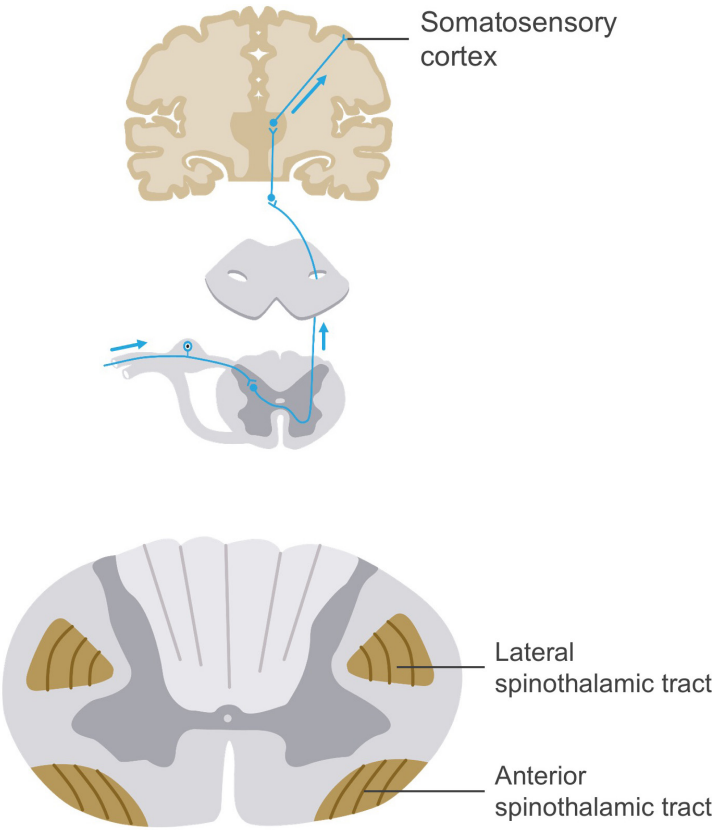
- Carry sensory information from periphery to cortex
  - proprioception
  - vibration
  - fine touch
  - two-point discrimination
- Fibers cross at spinal cord then ascend
- Somatotopic organization
  - fasciculus gracilis and cuneatus





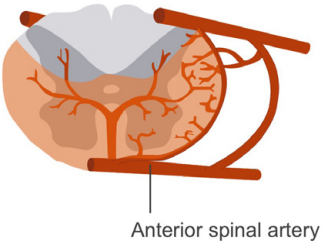
Spinothalamic tract

- Carries sensory information from periphery to cortex
  - pain and temperature (lateral spinothalamic tract)
  - light touch (anterior spinothalamic tract)
- Somatotopic organization

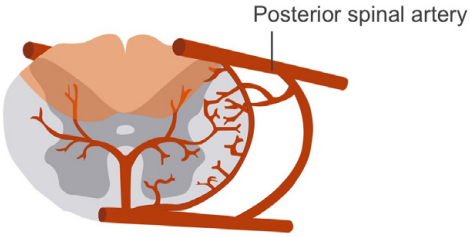


SPINAL CORD BLOOD SUPPLY

The **anterior spinal artery** supplies blood to the anterior two-thirds of the spinal cord.



The **posterior spinal arteries** are paired arteries along the dorsal surface of the spinal cord. They provide blood supply to the posterior third of the spinal cord.



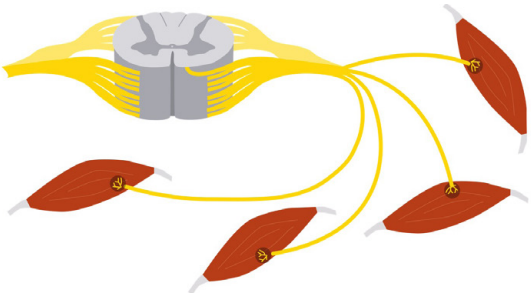
Radicular arteries are reinforcements from the thoracic aorta and the abdominal aorta, which supply the ventral and dorsal roots as well as the cord.

The largest radicular artery is the **spinal artery of Adamkiewicz**, which is derived from the aorta and is important in the thoracic and lumbar regions. It can be damaged during surgery on the aorta.

Most segmental arteries supply the cervical and lumbar enlargements; therefore, the thoracic cord is most vulnerable to hypotension and poor blood supply.

MYOTOMES

A myotome refers to all the muscles or groups of muscles innervated by the motor horn cells within a segment of the cord.



Upper limb myotomes

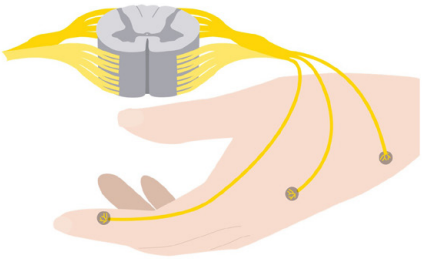
- C5—shoulder control
- C6—elbow range of motion
- C7—muscles of wrist
- C8—finger flexion / extension
- T1—intrinsic hand muscles

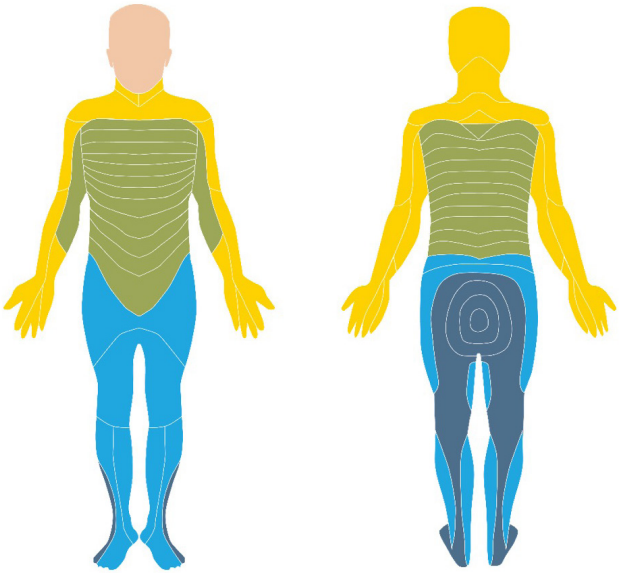
Lower limb myotomes

- L1 and L2—hip flexion
- L3 and L4—knee extension
- L5, S1, and S2—knee flexion
- L4—ankle dorsiflexion
- L5—extension of large toe
- S1 and S2—foot plantar flexion
- S2 and S3—adduction of toes

DERMATOMES

A dermatome is a region of skin or a structure, which has somatic afferent sensory innervation provided by a single spinal cord segment and a single nerve root.

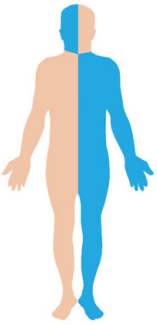




LOCALIZING SPINAL CORD LESIONS

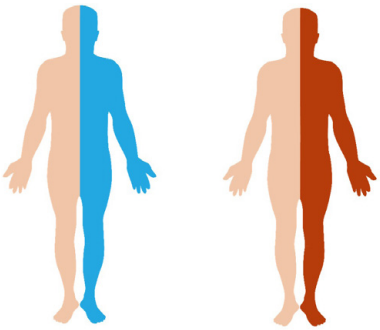
What signs and symptoms suggest a spinal cord deficit?

1. Motor or sensory losses on one or both sides of the body, that exclude the head and cranial nerve regions, are more likely to originate from the spinal cord.
2. Spinal cord lesions often have a demonstrable and discrete level of motor and / or sensory loss. This is often a significant clue to a cord problem.
3. Cranial nerve findings referable to one side of the head, presenting with long tract signs on opposite sides of the body (known as crossed signs), suggest lesion localization is most likely in the brainstem.



Crossed signs

4. When motor and sensory findings localize on the same side of the head and body, localization is most often in the cerebrum.



Rules to guide spinal cord localization

The specific sensory and / or motor abnormalities produced by a spinal cord lesion will depend on the function and level of the tracts involved.

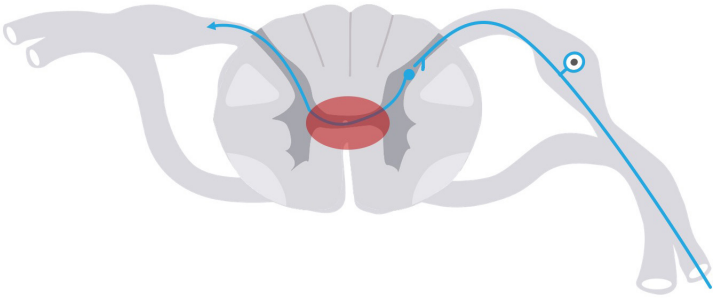
During and after your examination you should seek answers to the following questions.

- Which tracts are involved?
- Which side is involved?
- What level is involved?
- Are upper motor neurons or lower motor neurons involved?

Use this analysis to localize the lesion!

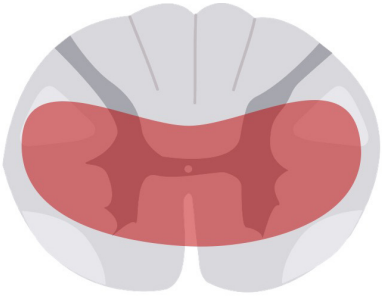
SPINAL CORD SYNDROMES

Small central lesion

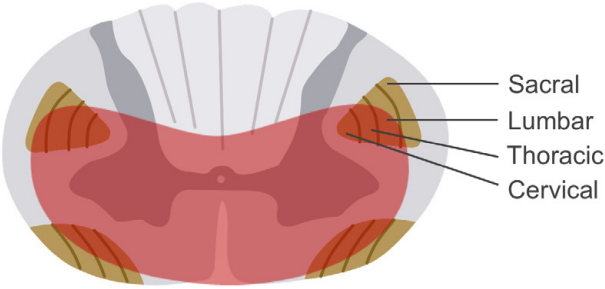


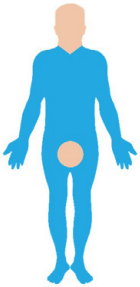
- Affects crossing spinothalamic tract fibers
- Bilateral loss of pain and temperature sensation
- Proprioception and vibration sense spared
- Possible causes
  - expanded central canal
  - syrinx

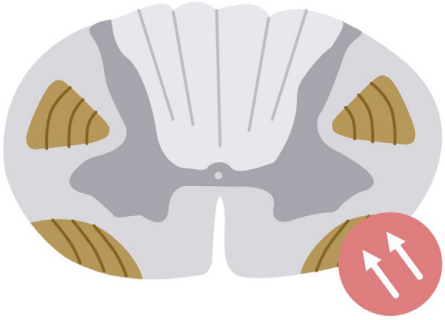
Large central cord lesion

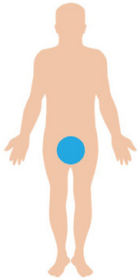


- Bilateral loss of pain and temperature sensation
- Lower motor neuron signs at level of lesion
- Upper motor neuron signs below level of lesion
- May be sacral sparing of sensory loss
    - external compression produces sacral anesthesia
- Possible causes
    - multiple sclerosis
    - astrocytoma
    - tumors

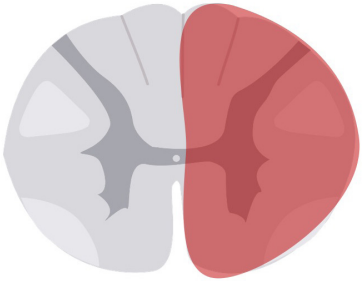






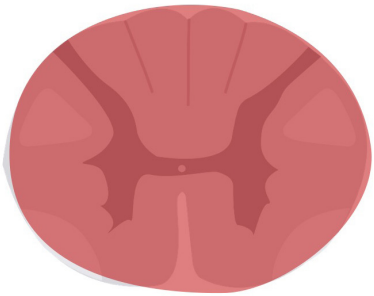


Hemisection (Brown-Séquard's syndrome)



- Ipsilateral loss of proprioception, vibration, and two-point discrimination below lesion
- Ipsilateral loss of pain and temperature sensation at level of lesion
- Contralateral loss of pain and temperature sensation 1–2 levels below lesion
- Lower motor neuron signs at level of lesion
- Upper motor neuron signs below level of lesion
- Possible causes
    - penetrating injuries
    - tumors
    - hemorrhage

Complete transection

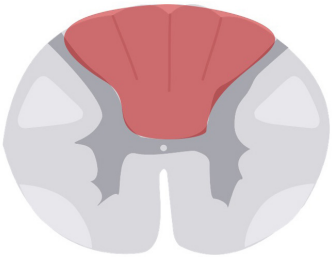


- Bilateral loss of sensation and motor activity below lesion
- Lower motor neuron signs at level of lesion
- Upper motor neuron signs below level of lesion
- Spinal shock and total flaccidity early after injury
- Loss of bladder and bowel function
- Respiratory dysfunction if lesion above C5
- Possible causes
    - trauma
    - penetrating injury
    - transverse myelitis
    - tumor
    - hemorrhage
    - cord compression

Combined degeneration of the cord

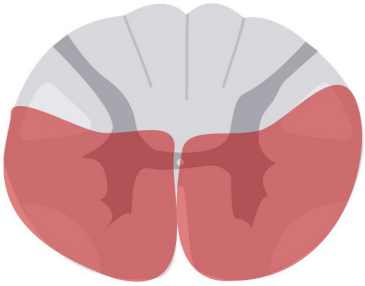
- Bilateral loss of proprioception and vibration sense
- Bilateral loss of motor function
- Pain and temperature sensation not affected
- Possible causes
    - nitrous oxide abuse
    - B12 deficiency
    - copper deficiency

Tabes dorsalis



- Most often bilateral damage (affecting legs)
- Loss of proprioception, deep pain, and touch
- Sensory ataxia
- Loss of reflexes
- Hypotonia
- Lightning-like pain in legs
- Possible causes
    - tertiary syphilis

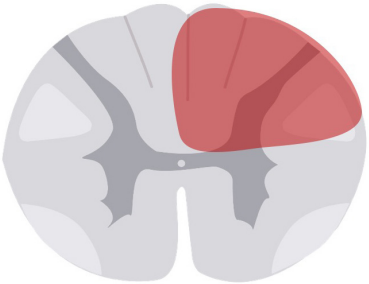
Anterior spinal artery syndrome



- Bilateral loss of pain and temperature sensation 1–2 levels below lesion
- Proprioception and vibration sense remain intact
- Bilateral loss of motor activity below lesion

- Lower motor neuron signs at level of lesion
- Upper motor neuron signs below level of lesion
- Possible causes
    - aortic aneurysm
    - hypotension
    - surgery
    - atherosclerosis
    - embolic stroke
    - hemorrhage
    - damage to spinal artery of Adamkiewicz

Posterior spinal artery syndrome



- Ipsilateral total loss of sensation at level of lesion
- Loss of reflexes for segment at level of lesion
- Ipsilateral loss of proprioception and vibration sense below lesion

Further reading

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PMID: 24169873

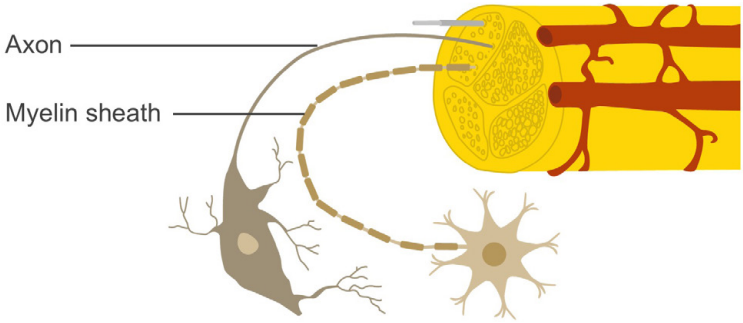
Chapter 6

THE PERIPHERAL NERVOUS SYSTEM

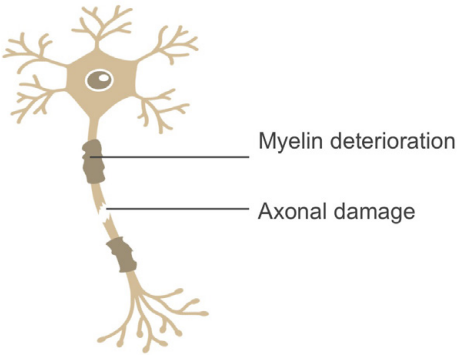
PERIPHERAL NERVOUS SYSTEM ANATOMY

The connection between the peripheral nervous system and the central nervous system (CNS) occurs at the nerve root level.
The nerve roots organize into a series of relays, known as **plexuses**, where mixing and switching of motor and sensory nerves and their data occur for distribution into and out of the CNS.

Peripheral nerves are made up of a **mix of sensory and motor fibers**, which are derived from several spinal roots via the plexuses.
Nerves are constructed of numerous axons bundled together. In larger nerves, the axons are surrounded by myelin sheaths.

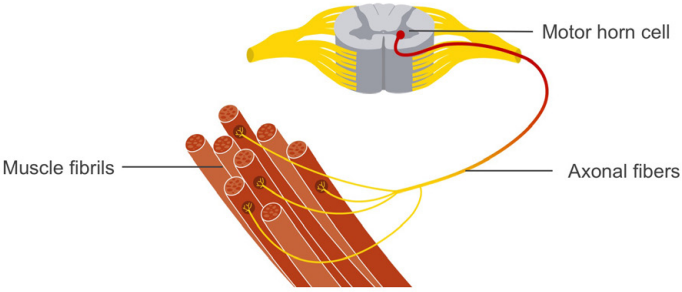


Pathology of nerves can involve the destruction of axons, or the deterioration of the myelin coatings.



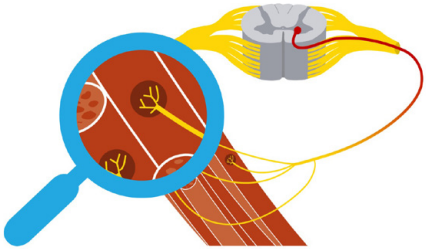
Motor unit

A motor unit is the connections one motor horn cell has with numerous muscle fibrils.

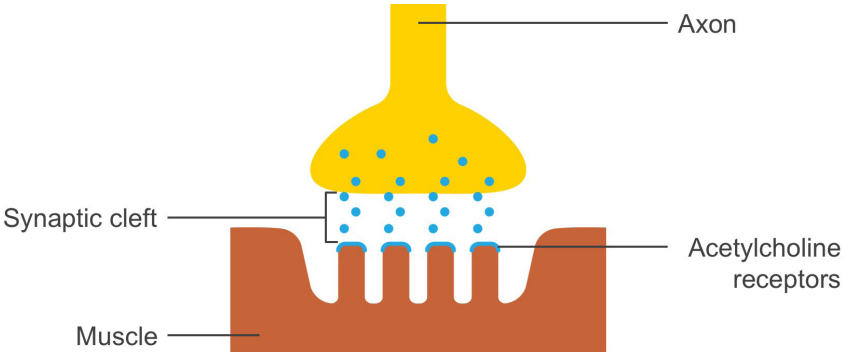


The neuromuscular junction

The connection a motor horn cell axon has with each muscle fibril is called the neuromuscular junction.



This is a chemical synapse between the axon terminus and the muscle fiber. Acetylcholine is the neurotransmitter released from the axon terminal. Acetylcholine traverses the synaptic space, binding to specific receptors, which activates the muscle, resulting in contraction.



LOCALIZING A PERIPHERAL NERVE INJURY

What signs and symptoms can help you localize a peripheral nerve injury?

1. Lower motor neuron signs
2. Pattern of dermatomal nerve root loss
3. Pattern of nerve or plexus distribution
4. Glove and stocking distal sensory loss

Signs of motor nerve damage

- Atrophy
- Cramping
- Weakness and fatigue
- Change in reflexes

Signs of sensory nerve damage

- Anesthesia
- Hypoesthesia
- Hyperesthesia

Signs of axonal damage

- From lack of oxygen / blood supply
- Impairment most distally first
- Glove and stocking sensory loss and atrophy (e.g., diabetic neuropathy)

Signs of demyelination

- Loss of muscle stretch reflexes
- Weakness
- Sensory alterations

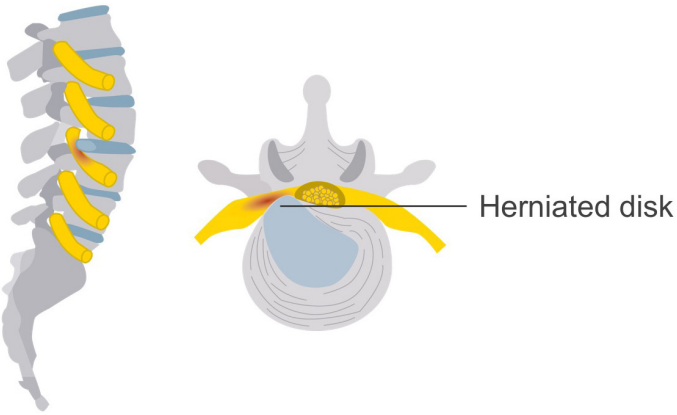
PATHOLOGY AFFECTING PERIPHERAL NERVES

Motor neuron disease

- Results from degeneration of anterior horn cells
- Varying degrees of upper motor neuron loss
- Amyotrophic lateral sclerosis (ALS)
  - upper and lower motor neuron involvement
  - Babinski response
  - hyperreflexia
  - atrophy
  - fasciculations
- No sensory loss

Nerve root lesions

Herniations



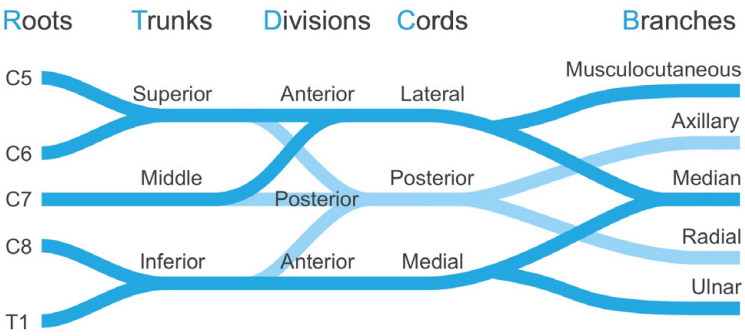
- Herniation of nucleus pulposus in intervertebral disc
- Irritation of nerve root causes radiculopathy
- Sensory and / or motor symptoms
- Cauda equina symptoms may be bilateral
- C5–C7 and L4–S1 most vulnerable

Dorsal root lesion

- Shingles (herpes zoster)
- Tabes dorsalis (syphilis)

THE BRACHIAL PLEXUS

The brachial plexus involves motor and sensory nerves that innervate the upper limbs.



Upper plexus injuries

- Generally from excessive separation of neck and shoulder (e.g., difficult birth)
- Erb's palsy
  - arm rotated medially

    - forearm pronated
    - fingers and wrist flexed (unable to straighten)
    - scapula often winged

Lower plexus injuries

- Generally nontraumatic compression (e.g., pancoast tumor of upper lung)
- Klumpke's palsy
    - avulsion of C8 and T1 roots
    - claw hand
    - forearm supinated
    - wrist and fingers flexed
- Horner's syndrome
    - damage to T1 root and nearby paraspinal ganglionic chain
    - miosis
    - anhidrosis
    - ptosis

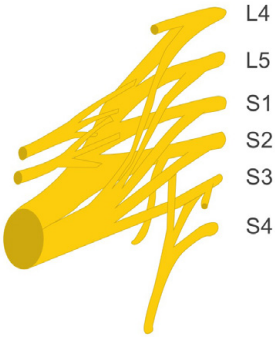
Other causes of brachial plexus injury

- Diabetic amyotrophy
- Brachial plexitis
- Neoplasia
- Post-irradiation injury
- Obstetrical palsies
- Postoperative plexopathy
- Thoracic outlet syndrome

THE LUMBOSACRAL PLEXUS

In reality, the lumbosacral plexus is actually two plexuses—the lumbar and sacral.

Lumbar plexus



Sacral plexus

The major nerves which arise from the lumbosacral plexus include both motor and sensory nerves, which innervate the lower extremities.

Clinically important nerves

- Femoral
- Sciatic
  - tibial
  - peroneal

Common causes of lumbosacral plexus injury

- Diabetes
- Neoplasia
- Retroperitoneal hemorrhage
- Post-irradiation plexopathy

PERIPHERAL EXAMINATION

Examination of peripheral nervous system issues requires pattern recognition. Is there involvement suggesting a nerve root, the plexus or a peripheral nerve?
One needs to examine and assess the appearance of the muscles, muscle power, peripheral sensation, and muscle reflexes to get a good localization of peripheral nervous system impairments.

Upper extremity examination

Motor examination

The strongest muscles in the upper arms are the flexors. Therefore, the most likely muscles to show subtle weakness are the extensors.

Assess pronator drift to look for lateralized muscle weakness.

Observe for atrophy and fasciculations.

All of the muscles in the upper extremity are innervated by more than one nerve root. Recognition of patterns of weakness becomes most important in peripheral neurological assessment.
This can be accomplished best by reviewing general motor control and assessing reflexes controlled by each nerve root.

C5

- Controls shoulder abduction and elbow flexion
- Test biceps reflex

C6

- Controls elbow flexion (especially in a semi-pronated state)
- Test brachioradialis reflex

C7

- Controls finger extension and elbow extension
- Test triceps reflex

C8

- Controls finger flexors
- Test finger flexor reflex

T1

- Controls movement of the intrinsic hand muscles
- No applicable reflex
- Test by having patient spread fingers while resisting external efforts to push them together

Nerves of importance for motor function of the arm

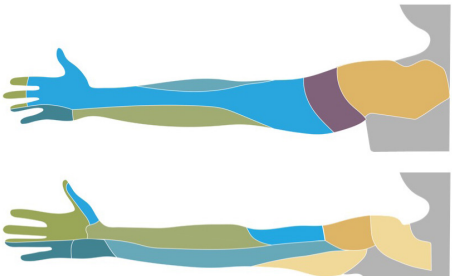
- Axillary
  - innervates deltoid muscle (movements of the shoulder)
- Musculocutaneous
  - innervates muscles important for flexion of arm at the elbow
- Radial
  - innervates all extensor muscles of upper extremity

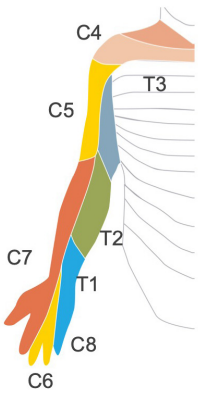
- Ulnar
  - innervates all but four intrinsic hand muscles
- Median
  - innervates most flexor and pronator muscles of forearm and several intrinsic hand muscles

Sensory examination

Nerves involved with sensation in the arm

- Musculocutaneous
- Median
- Ulnar
- Radial





Lower extremity examination

Motor examination

The strongest muscles in the lower extremities are the extensor muscles. Therefore, weakness is more easily shown in the flexor groups.

Observe for atrophy, fasciculations, and trophic changes such as changes in hair patterns, thinning of skin, and changes in arches.

Measure the circumference of the legs. They should not differ by more than 2 cm.

In the leg, as in the arm, all muscles are innervated by more than one nerve root so that recognition of patterns of weakness become most important in peripheral neurological assessment.
This can be accomplished best by reviewing general motor control and reflexes by nerve root.

L1 and L2

- Control hip flexion
- No corresponding reflex to test for these
- Test patient laying down—have them flex hip and hold against resistance

L3 and L4

- Control knee extension
- Test knee jerk reflex

L5

- Controls dorsiflexion, inversion and eversion of foot, and extension of big toe
- No definitive reflex to test
- Hamstrings reflex or posterior tibial reflex can test this root level (only useful if asymmetric—as supportive evidence of other findings)

S1

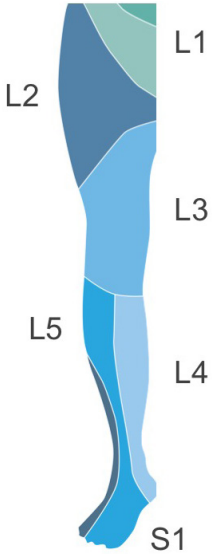
- Controls hip extension, knee flexion, and plantar flexion
- Test ankle reflex

Nerves of importance for motor function of the leg

- Femoral
  - supplies muscles associated with knee extension
- Sciatic
  - supplies knee flexion
  - separates into tibial and common peroneal branches
- Tibial
  - supplies foot plantar flexion and inversion as well as small muscles of foot
- Common peroneal
  - supplies dorsiflexion of foot and eversion of ankle

Sensory examination

The dermatomes that are most frequently affected by radiculopathy, for example when disc disease is present, are L4, L5, and S1.



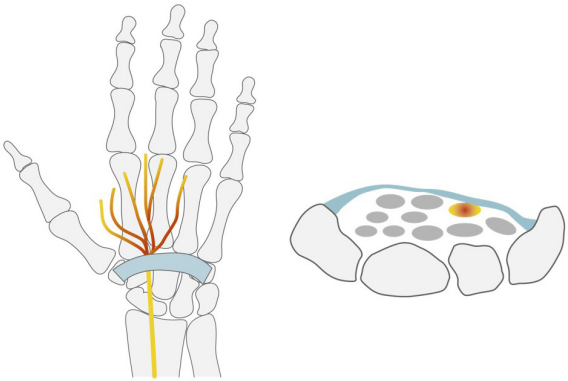
Nerves involved with sensation in the leg

- Anterior femoral cutaneous
- Medial femoral cutaneous
- Saphenous
- Sciatic (sural, peroneal)

COMMON PERIPHERAL NEUROPATHIES

Carpal tunnel syndrome

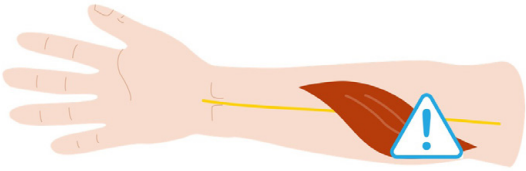
- Compression of median nerve in carpal tunnel at wrist
- Pain, numbness, and paresthesia in wrist and palm
  - worse at night
  - exaccerbated by excessive movement of fingers, hand or wrist
  - thenar eminence sensation often spared
- Positive Tinel's sign
- Phalen's maneuver elicits paresthesia



Pronator teres syndrome

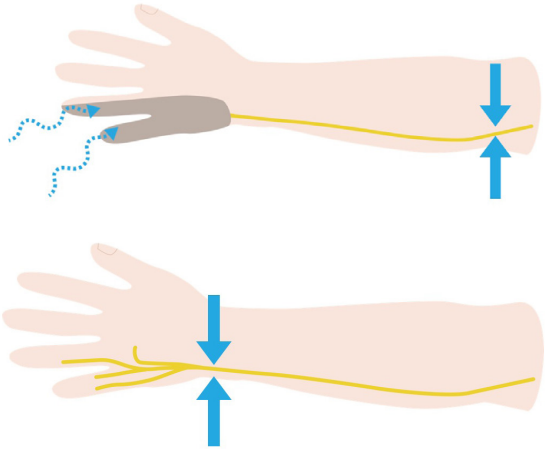
- Compression of median nerve passing through pronator teres muscle
- Pain in forearm and hand
  - less pain at night
- Weakness of wrist pronators and distal finger flexors
- Difficulty pinching

- Pronation against resistance can elicit symptoms
- Positive Tinel's sign
- Negative Phalen's test



Ulnar neuropathy

- Compression at medial epicondyle or in Guyon's canal (at wrist)
- Tingling in the medial hand at fifth and half of fourth digit
- Difficulty with fine motor control of hand (due to weakness of intrinsic muscles)
- Tinel's sign may be present
- Froment's sign may be present

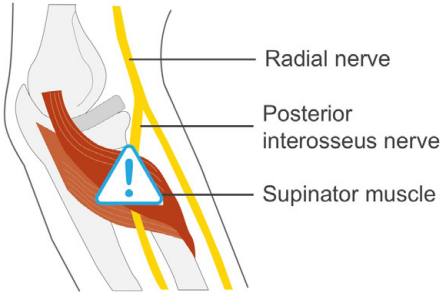


Saturday night palsy

- Compression of radial nerve in spiral groove
- Wrist drop
- Weak finger extension at metacarpophalangeal joints
- Triceps may be spared or only partially involved

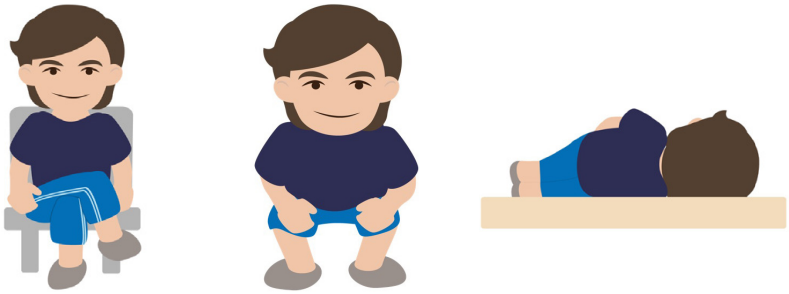
Posterior interosseous syndrome

- Compression of posterior interosseous nerve (branch of radial) between heads of supinator muscle
- Weakness of finger extensors
- Radial deviation of wrist



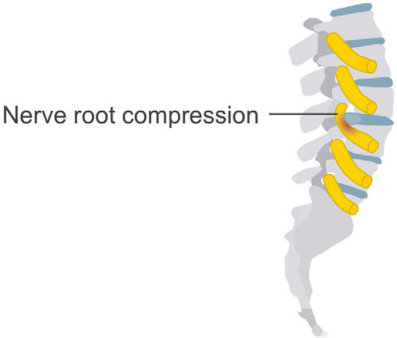
Peroneal neuropathy

- Compression of common peroneal nerve as it winds around fibular head
- Weakness of ankle dorsiflexion of foot and toes, and ankle eversion
- Foot drop occurs when severe
- Often caused by excessive crossing of legs, squatting or laying on side



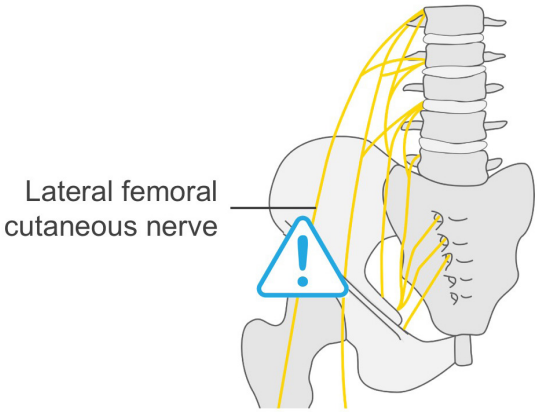
L5 radiculopathy

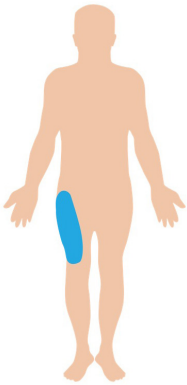
- Injury of the spinal nerve roots (sensory and / or motor)
- Usually caused by compression of root at or near foraminal exit
- Lancinating pain in leg
- Weakness of inversion and big toe flexion
- Medial hamstring reflex lost



Meralgia paresthetica

- Compression of the lateral femoral cutaneous nerve beneath inguinal ligament
- Pain and paresthesia at anterolateral thigh (above knee)

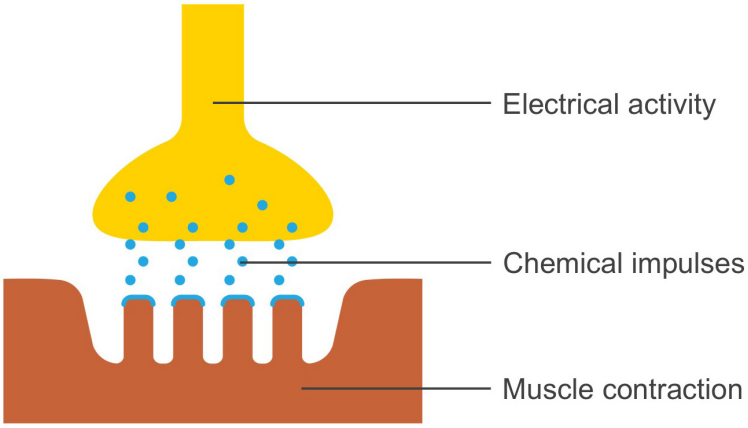




DISEASES AFFECTING THE NEUROMUSCULAR JUNCTION

The neuromuscular junction is a specialized synapse allowing the conversion of nerve electrical action potentials to chemical impulses, which transmit the message for a muscle to contract.

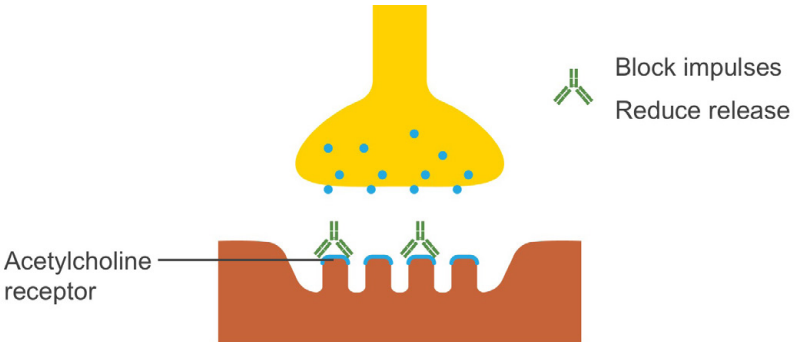
There are multiple clinical syndromes affecting the neuromuscular junction, with the most common being myasthenia gravis.



Myasthenia gravis

This autoimmune disorder generally occurs in younger women or in older men.

Antibodies against the acetylcholine receptor are produced, which block transmission of impulses as well as reduce the amount of acetylcholine that gets released at the synapse.



Clinical characteristics

- Fluctuating weakness and fatigue
- Facial weakness
- Limb muscle weakness (especially with repetitive movements)
- Ptosis, diplopia, blurred vision
- Slurred speech
- Dysphagia
- Neck weakness
- Shortness of breath

Testing for myasthenia gravis

- Test for limb muscle weakness with repetitive movements
- Test for respiratory fatigue—have patient count at steady cadence on one breath
- Examine extended upgaze (watch for ptosis)
- Ice pack test
  - improvement of ptosis suggests neuromuscular junction problem (not stroke or nerve injury)

Further reading

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