MUNI

Cranial nerves. Intracranial hypertension. Consciousness disturbances. Vertebral column examination.

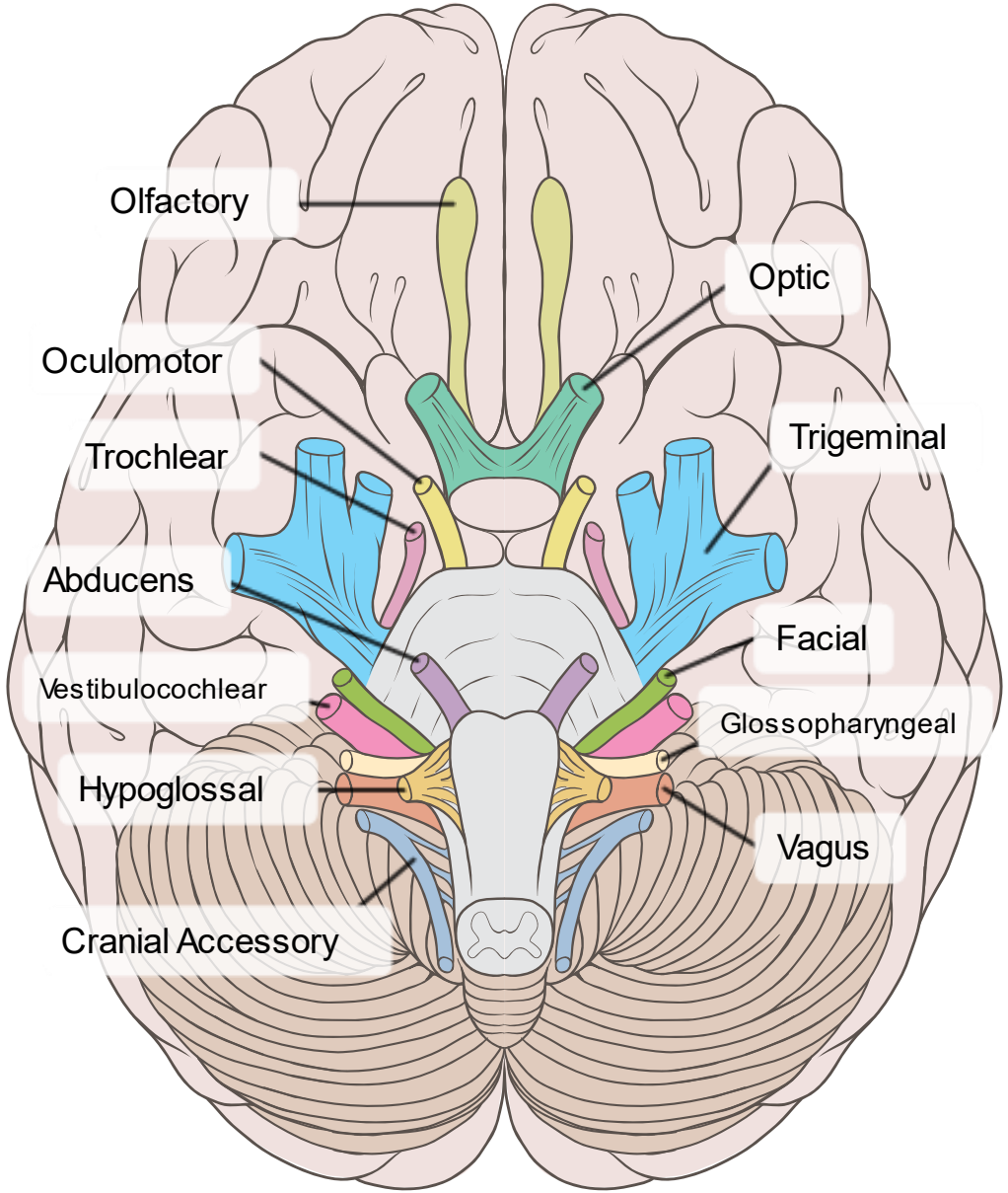
Jan Kolcava

NEUROLOGICKÁ KLINIKA
LF MU a FN BRNO

Cranial nerves

Cranial nerves

- I. Olfactory nerve
- II. Optic nerve
- III. Oculomotor nerve
- IV. Trochlear nerve
- V. Trigeminal nerve
- VI. Abducens nerve
- VII. Facial nerve
- VIII. Vestibulocochlear nerve
- IX. Glossopharyngeal nerve
- X. Vagus nerve
- XI. Accessory nerve
- XII. Hypoglossal nerve



Cranial nerves – part I

- The olfactory nerve (I): The sense of smell.
- The optic nerve (II): This nerve carries visual information from the retina of the eye to the brain.
- The oculomotor nerve (III): The nerve controls most of the eye's movements, the constriction of the pupil, and maintains an open eyelid.
- The trochlear nerve (IV): A motor nerve that innervates the superior oblique muscle of the eye.
- The trigeminal nerve (V): This is responsible for sensation in the face and motor function of the mouth.
- The abducens nerve (VI): A motor nerve that innervates the lateral rectus muscle of the eye.

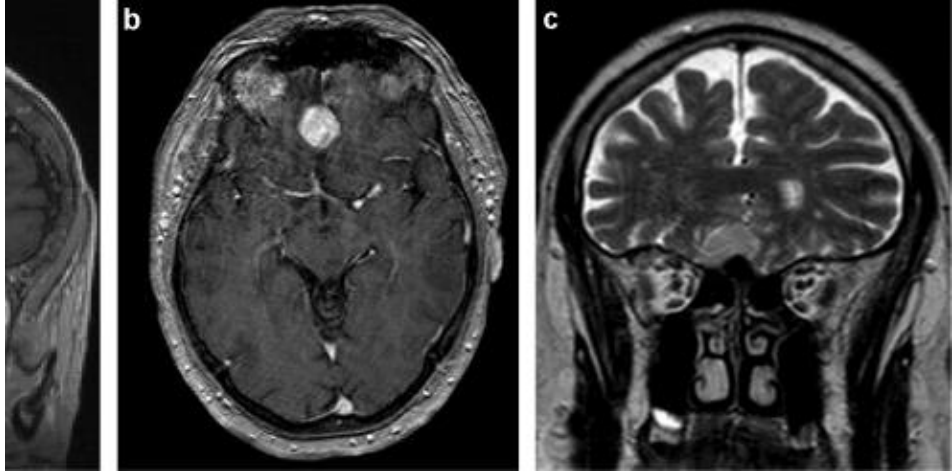
Cranial nerves – part II

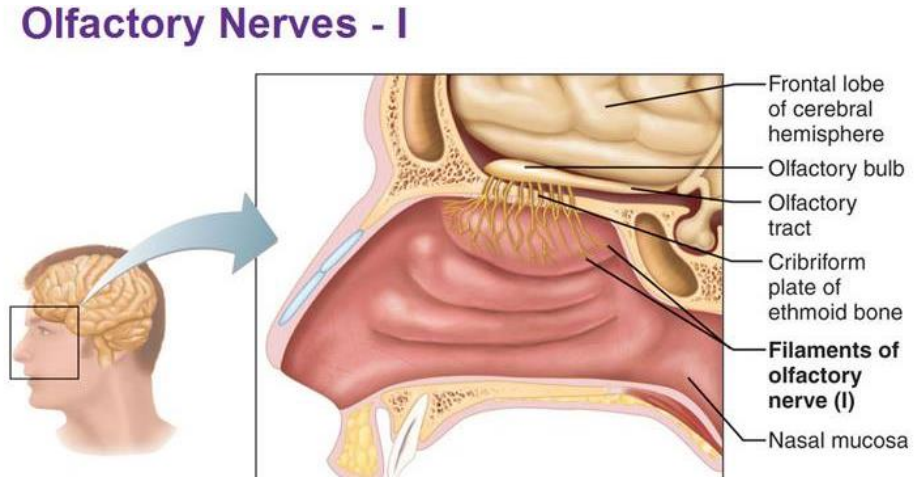
- The facial nerve (VII): Controls the muscles of facial expression, and functions in the conveyance of taste sensations from the anterior two-thirds of the tongue.
- The vestibulocochlear nerve (VIII): Responsible for transmitting sound and balance information from the inner ear to the brain.
- The glossopharyngeal nerve (IX): This nerve receives sensory information from the tonsils, the pharynx, the middle ear, and the rest of the tongue.
- The vagus nerve (X): The nerve is responsible for many tasks, including heart rate, gastrointestinal peristalsis, sweating, and muscle movements in the mouth.
- The spinal accessory (XI): Innervation of trapezius and sternocleidomastoideus muscles.
- The hypoglossal nerve (XII): This nerve controls the tongue movements of speech, food manipulation, and swallowing.

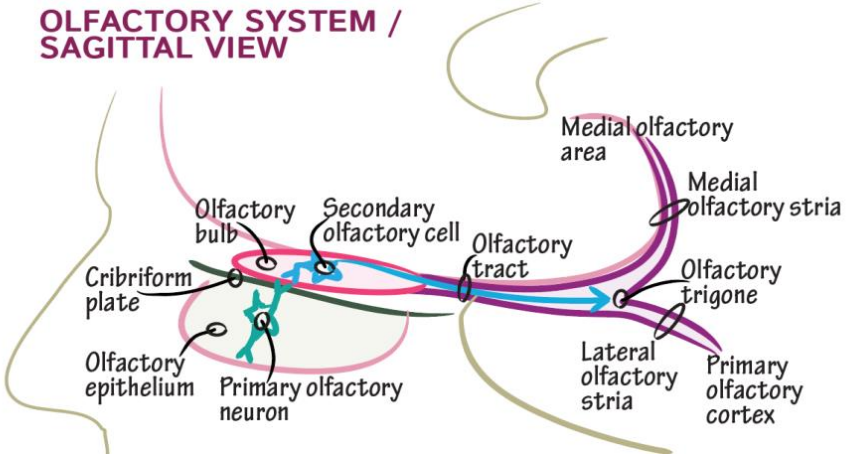
- ** There are many mnemonic devices to remember the cranial nerves („Ooh, Ooh, Ooh To Touch And Feel Very Good Velvet. Such heaven!“…).*

Olfactory nerve (I) - anatomy

- The olfactory nerve relays smell information to the cortex via a three-neuron pathway.
  - First-order sensory neurons are located in the mucosa of the upper nasal septum and superior nasal concha. These bipolar neurons bundles collectively called the olfactory nerves. The thin, unmyelinated olfactory fibers enter the anterior cranial fossa via the cribriform plate of the ethmoid bone.
  - Second-order sensory neurons are located in the olfactory bulb. Their axons course in the olfactory tract to the medial or lateral olfactory striae. These axons synapse in the amygdala, the prepiriform area, or neighboring areas.
  - Third-order neurons relay the information to the cerebral cortex.







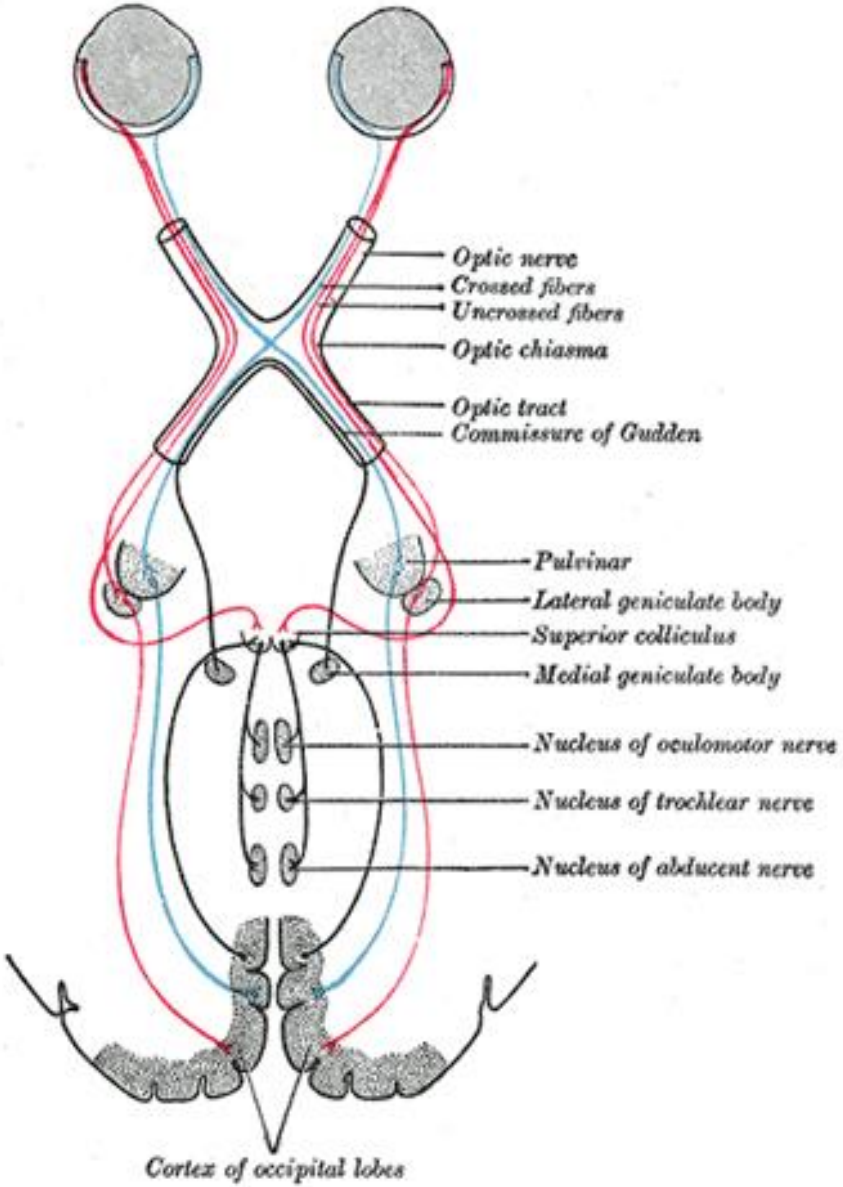
Journal de Radiologie Diagnostique et Interventionnelle, Volume 94, Issue 10, October 2013, Pages 992-999.
Studyblue.com

Olfactory nerve (I) - examination

- The olfactory nerve mediates the sense of smell (olfaction).
- ***Gross assessment:*** ask the patient about the ability to smell and changes of food flavor
- ***More detailled testing:*** With eyes closed, ask the patient to sniff a mild stimulus such as toothpaste,coffee, cloves or soap and identify the odorant. Each nostril is tested separately.
  - Irritants such as alcohol should not be used because they may be detected as noxious stimuli independent of olfactory receptors.
- Pathology: hyposmia x anosmia x dysosmia;
- smell pseudohallucination – epileptic seizure affecting uncus gyri hippocampi
- **Testing is usually omitted unless there is suspicion for inferior frontal lobe disease, in persons who experience spontaneous loss of smell, in patients with a Parkinson's disease, and patients who have suffered head injury.*

Optic nerve (II)- anatomy

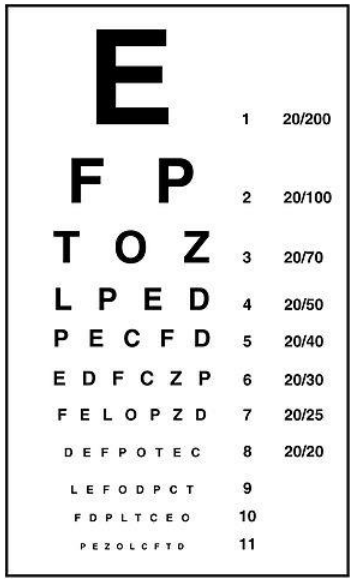
- The optic nerve relays sight information from the retina to the visual cortex via a four-neuron pathway.
  - First-order neurons (rods and cones) in the retina translate incoming photons into impulses, which are relayed to second-order bipolar neurons and third-order ganglion cells.
  - These retinal ganglion cells combine to form the optic nerve.
  - The optic nerve passes from the orbit into the middle cranial fossa via the optic canal.
  - Ninety percent of the third-order neurons in the optic nerve synapse in the lateral geniculate body, which then projects to the cortex.
  - Ten percent of the third-order neurons synapse in the mesencephalon.
- Lesion of the optic nerve may cause partial or complete loss of vision, depending on the point at which the nerve is disrupted.

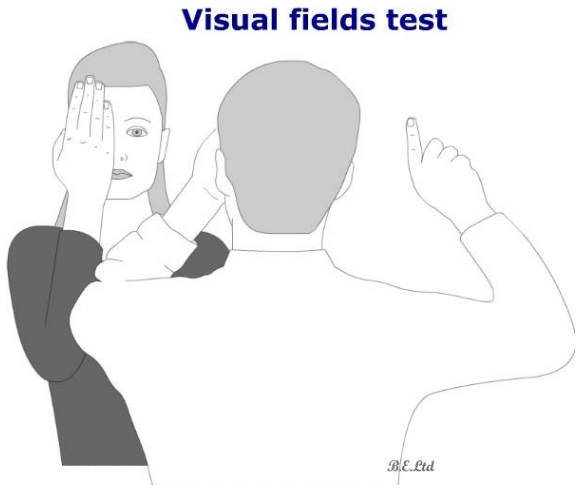


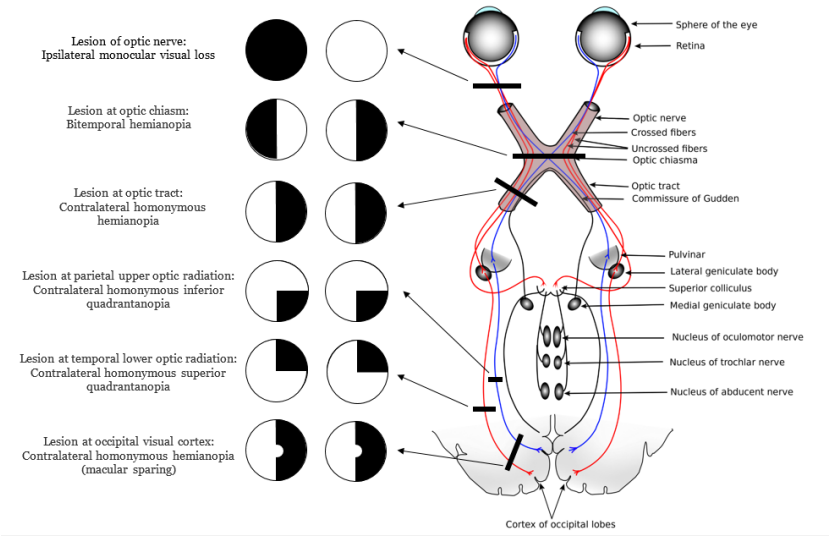
Optic nerve (II) - examination

- Check visual acuity (with eyeglasses or contact lens correction) using a Snellen chart or similar tool.
- Test the visual fields by comparing the patient's visual fields to your own.
- Individual eye fields should be tested.
  - Face the patient at a distance of approximately 1 m and place your hands at the periphery of your visual fields in the plane that is same distance between you and the patient.
  - Instruct the patient to look directly at the center of your face and to indicate when and where he sees one of your fingers moving.
  - Beginning with the two inferior quadrants and then the two superior quadrants, observe whether the patient detects the movements.

Clinicalexams.co.uk; teachmesurgery.com

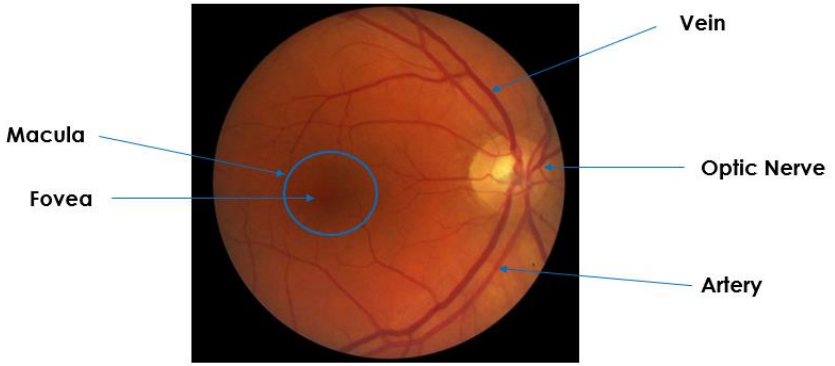






Optic nerve (II) - examination

- Detailled examination:
  - **Optic fundi** examined with an ophthalmoscope - the color, size, and degree of swelling or elevation of the optic disc noted, as well as the color and texture of the retina.
  - **Detalied perimeter (visual fields) examination.**
  - **VEP – visual evoked potentials**
- Pathology:
  - Amaurosis
  - Visual scotoma
  - Hemianopsia (homonymous x heteronymous)
  - Cortical blindness
  - Visual hallucinations

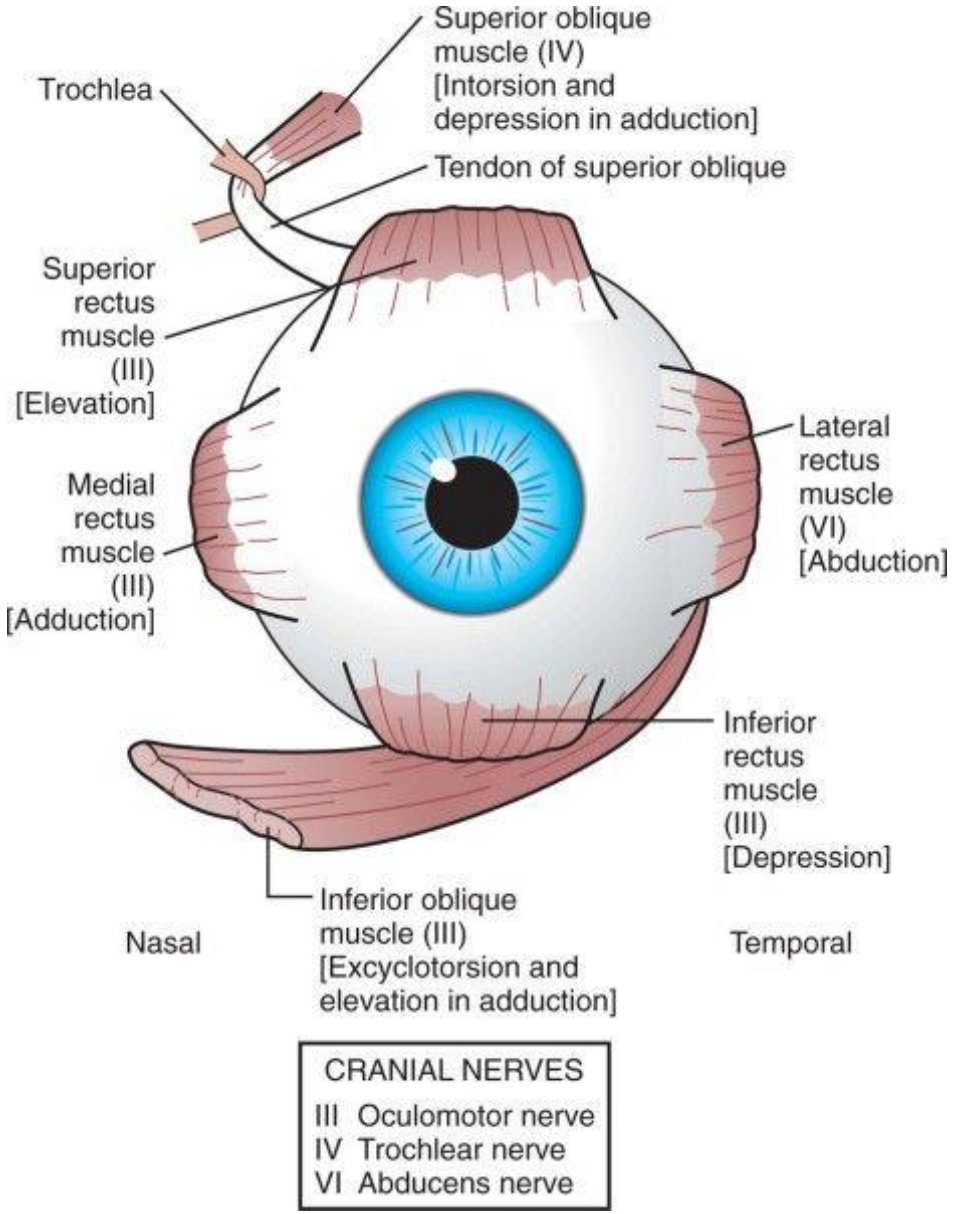


Paris as seen with bitemporal heteronymus hemianopia.

Morancoreutah.edu.com; en.wikipedia.com

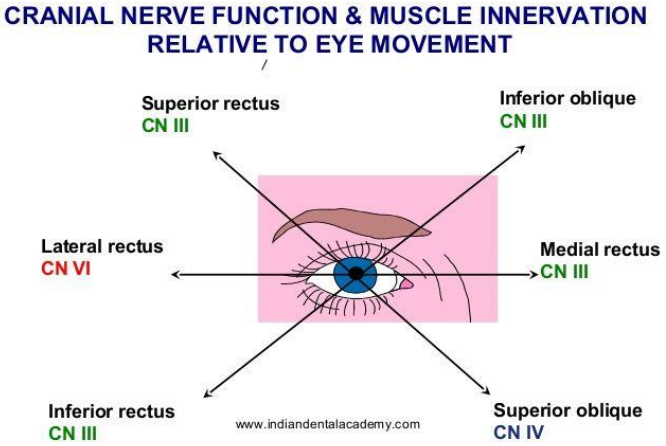
Cranial nerves – oculomotor (III), trochlear (IV), abducens (VI) - anatomy

- The oculomotor nerve (CN III) innervates fiive skeletal muscles in the orbit and parasympathetic preganglionic fibers from the accessory oculomotor (Edinger-Westphal) nucleus.
  - Postganglionic parasympathetic fibers then course via short ciliary nerves to the eyeball (these postganglionic ibers mediate pupillary constriction and accommodation).
- The trochlear nerve (CN IV) innervates the superior oblique muscle.
- The abducens nerve (CN VI) innervates the lateral rectus muscle



Cranial nerves – oculomotor (III), trochlear (IV), abducens (VI) - examination

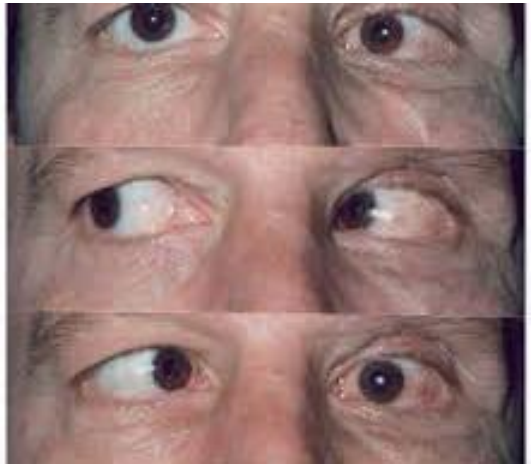
- Describe the size and shape of pupils and reaction to light and accommodation (i.e., as the eyes converge while following your finger as it moves toward the bridge of the nose).
- To check extraocular movements, ask the patient to keep his head still while tracking the movement of the tip of your finger.
  - Move the target slowly in the horizontal and vertical planes; observe any paresis, nystagmus, or abnormalities of smooth pursuit (saccades, oculomotor ataxia, etc.).
- Ask the patient about diplopia in any direction of gaze; true diplopia should almost always resolve with one eye closed.
- Horizontal nystagmus is best assessed at 45° and not at extreme lateral gaze (which is uncomfortable for the patient); the target must often be held at the lateral position for at least a few seconds to detect an abnormality.

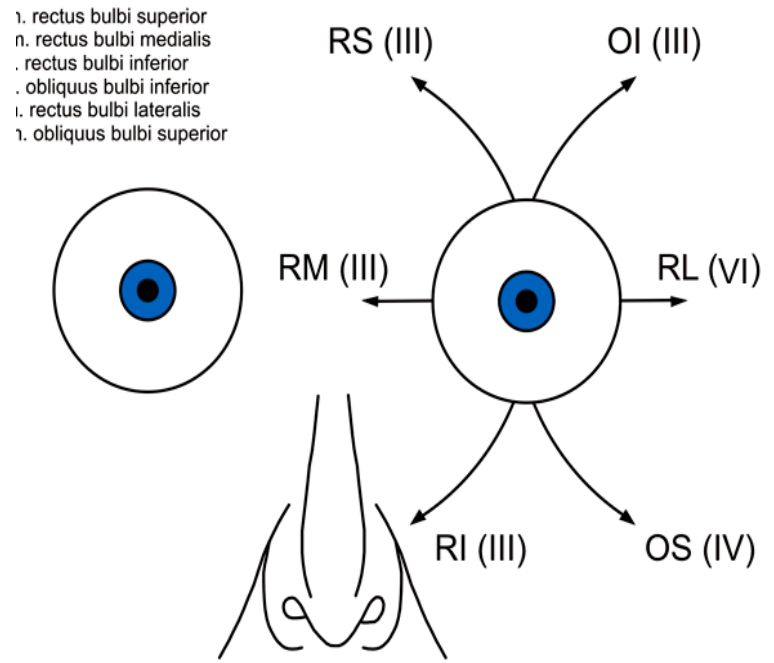


Ophtobook.com; psychology.wikia.org; allmedtest.com

Cranial nerves – oculomotor (III), trochlear (IV), abducens (VI) - examination

- ***Horizontal and vertical eye movements*** (full range / limitations)
- ***Position of the ocular bulbi***:
  - parallel, squint (= strabismus – convergent, divergent)
- ***Presence of nystagmus*** or other ocular oscillations
  - Nystagmus: direction, degree, amplitude, frequency
- ***Pupil size*** (normal – midposition, miotic, mydriatic)
- ***Pupil symmetry*** (isocoria, anisocoria)
- ***Pupil shape*** (round, irregular)
- ***Pupillary response to light***
  - direct (illuminated pupil)
  - indirect (consesual)
- ***Ability of convergence and pupilary reaction to it***
  - (constriction)

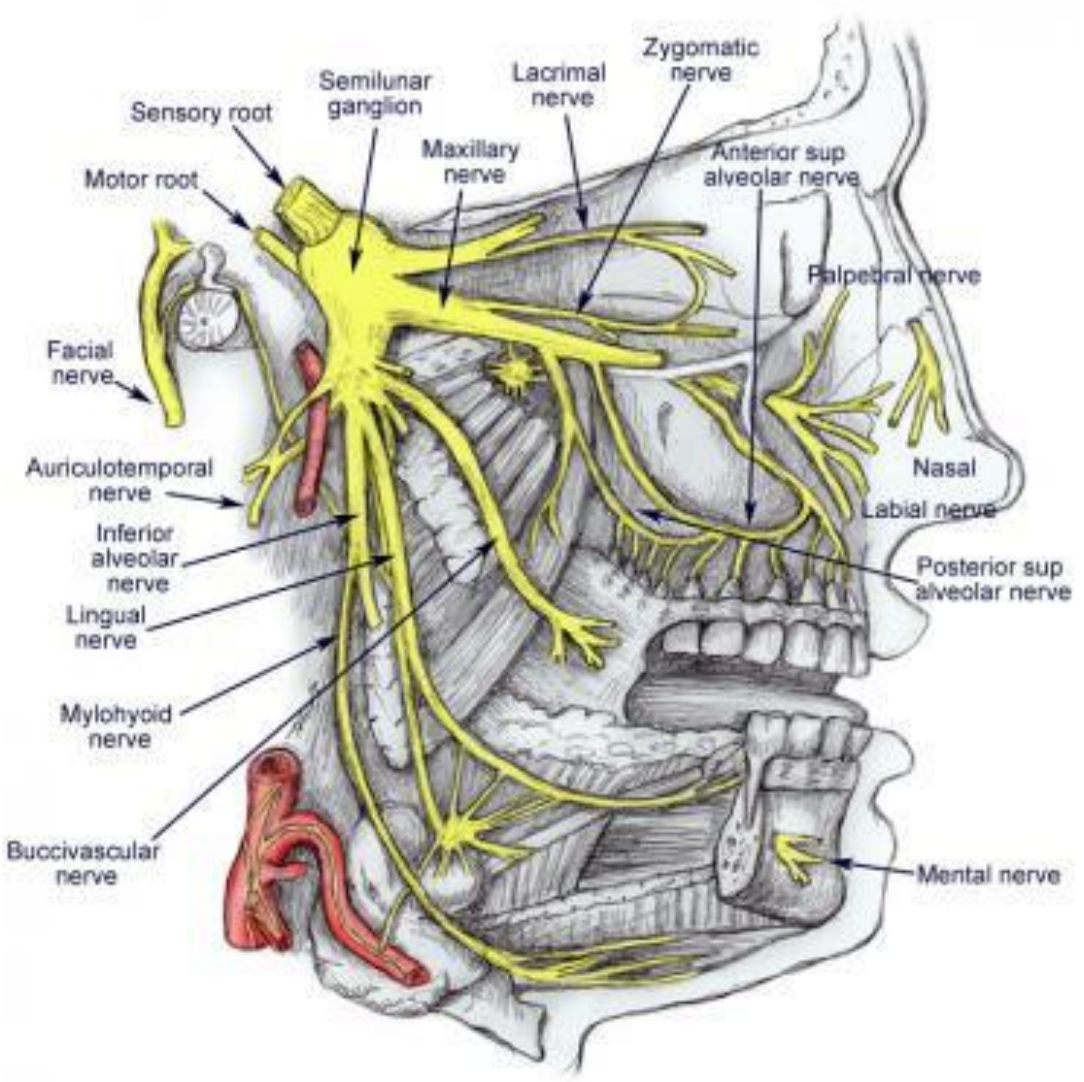




http://ophthalmology.stanford.edu/; scielo.br

Cranial nerves – trigeminal nerve (V)- anatomy

- The major sensory nerve of the head, conveys general somatic aferents centrally to the trigeminal sensory ganglion (also called the semilunar or gasserian ganglion) via its ophthalmic (V1), maxillary (V2), and mandibular (V3) divisions.
- Its mandibular division also innervates skeletal muscles (masticatory muscles).
- Because of the extensive distribution of CN V, most of the parasympathetic fibers from CN III, VII, and IX course with branches of CN V to reach their targets (smooth muscle and glands) these targets include Lacrimal gland, Submandibular and sublingual salivary glands, Nasal glands.
- V3 travels to the anterior 2/3 of the tongue, but this sensory input is not received by trigeminal nuclei but rather by the Solitary Nucleus.

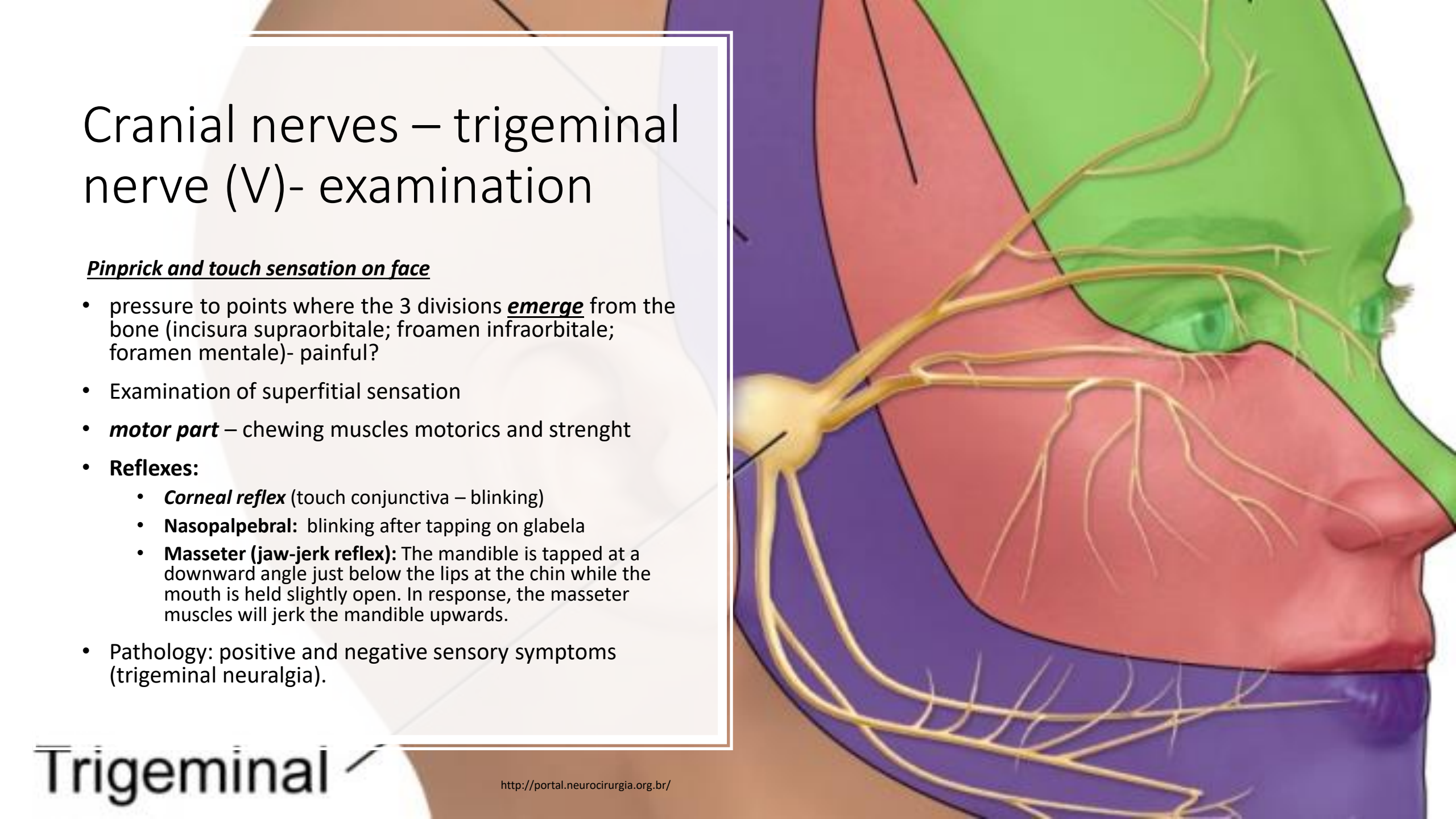


Netter's Clinical Anatomy - 4th Edition. John T. Hansen.

Cranial nerves – trigeminal nerve (V)- examination

Pinprick and touch sensation on face

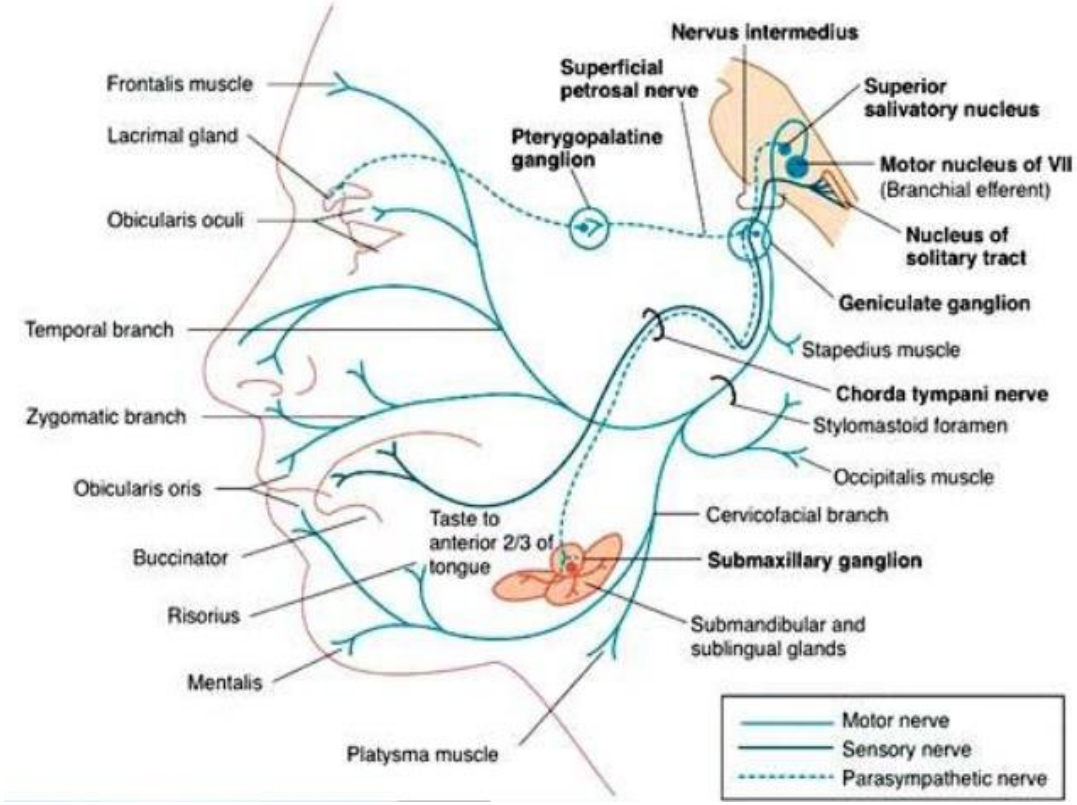
- pressure to points where the 3 divisions ***emerge*** from the bone (incisura supraorbitale; froamen infraorbitale; foramen mentale)- painful?
- Examination of superfitial sensation
- ***motor part*** – chewing muscles motorics and strenght
- **Reflexes:**
  - ***Corneal reflex*** (touch conjunctiva – blinking)
  - **Nasopalpebral:** blinking after tapping on glabela
  - **Masseter (jaw-jerk reflex):** The mandible is tapped at a downward angle just below the lips at the chin while the mouth is held slightly open. In response, the masseter muscles will jerk the mandible upwards.
- Pathology: positive and negative sensory symptoms (trigeminal neuralgia).

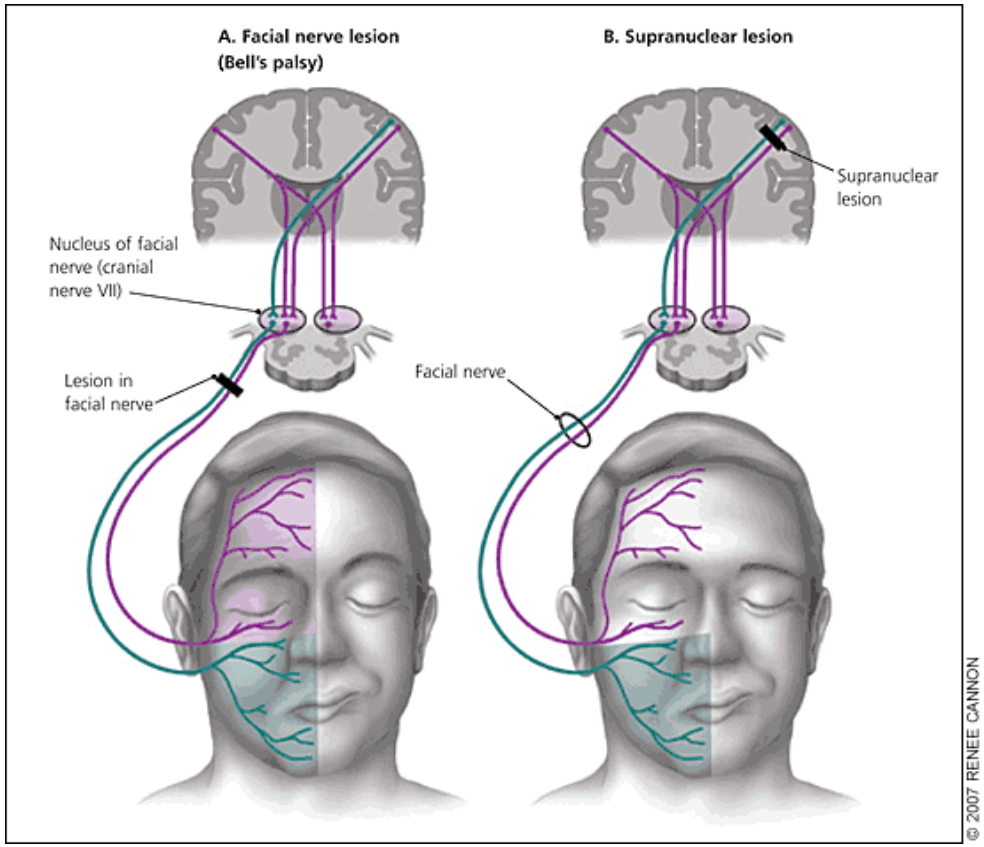


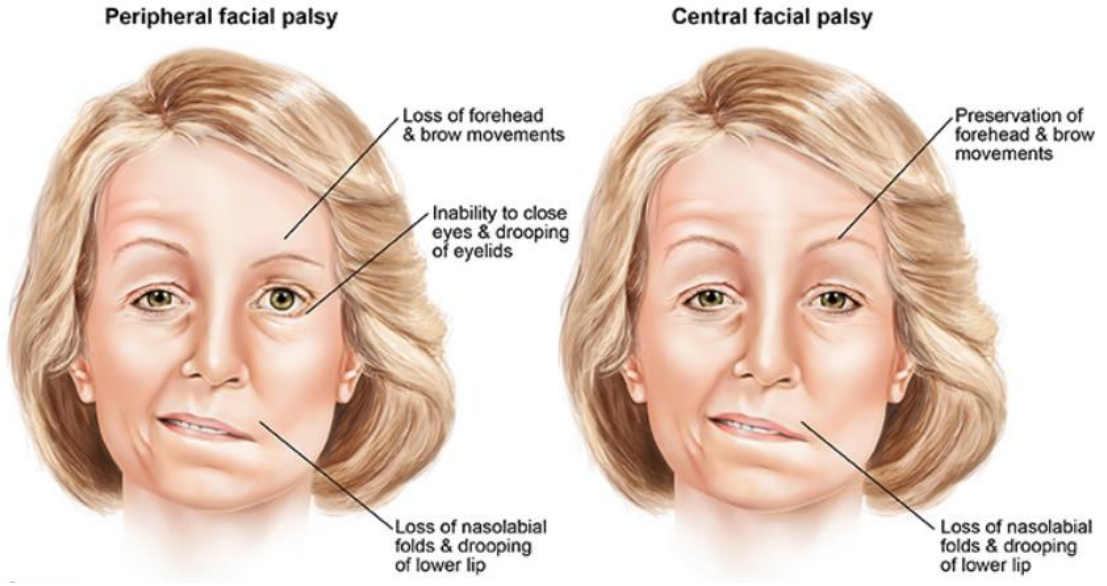
http://portal.neurocirurgia.org.br/

facial nerve (VII) - anatomy

- The major motor nerve of the head - somatic eferents to skeletal muscles.
- Additionally, sends preganglionic parasympathetic fibers from the superior salivatory nucleus to the pterygopalatine ganglia.
- The facial nerve also conveys special visceral aferents from taste receptors on the anterior two thirds of the tongue along the chorda tympani to the geniculate sensory ganglion.





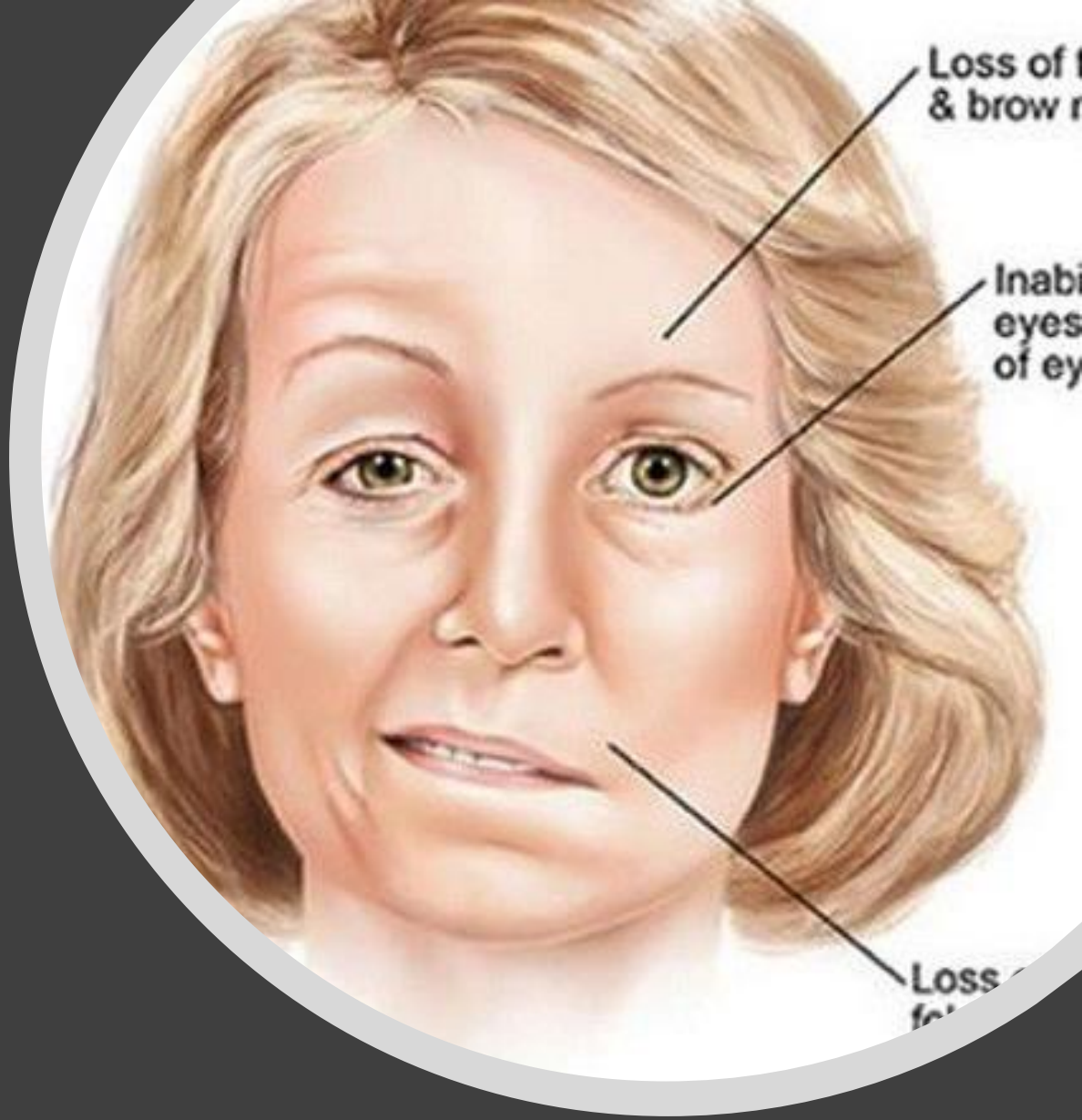


Facial nerve – central vs. periferal palsy

Netter's Clinical Anatomy - 4th Edition. John T. Hansen.; lookfordiagnosis.com

facial nerve (VII) - examination

- Look for facial asymmetry at rest and with spontaneous movements.
- Test eyebrow elevation, forehead wrinkling, eye closure, smiling, and cheek puff.
- **Look in particular for differences in the lower versus upper facial muscles**
  - weakness of the lower two-thirds of the face with preservation of the upper third suggests an upper motor neuron lesion, whereas weakness of an entire side suggests a lower motor neuron lesion
- Ask about taste - on the anterior two thirds of the tongue.
- Hyperacusis – due to a stapiedius nerve laesion.



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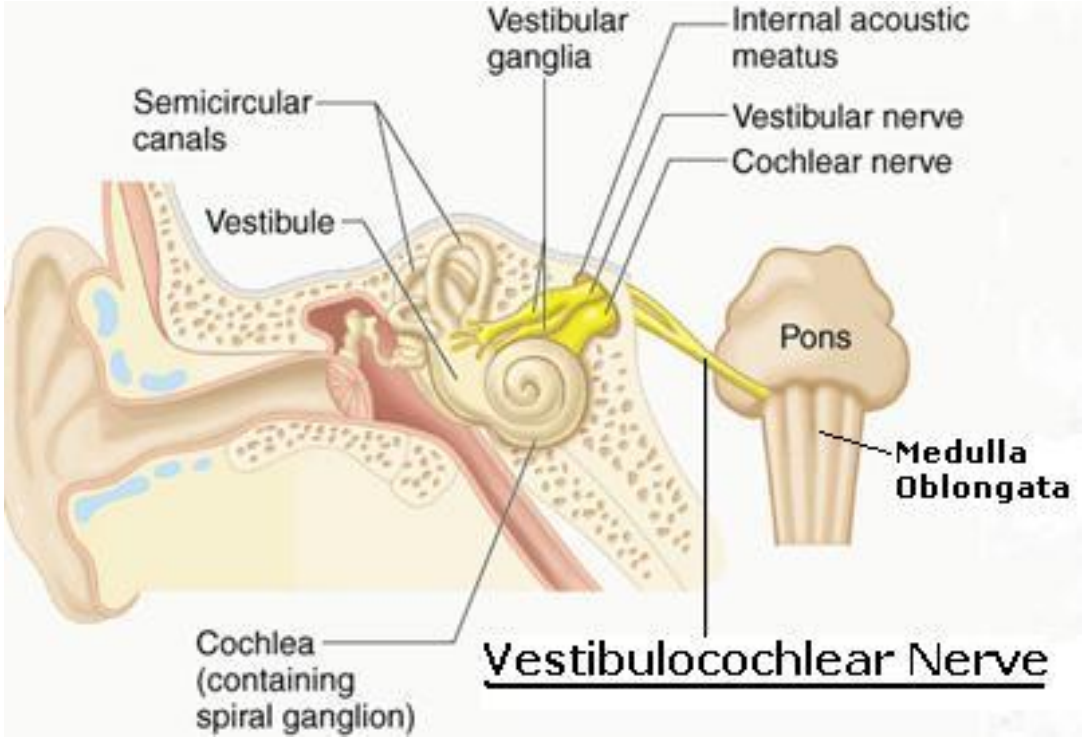
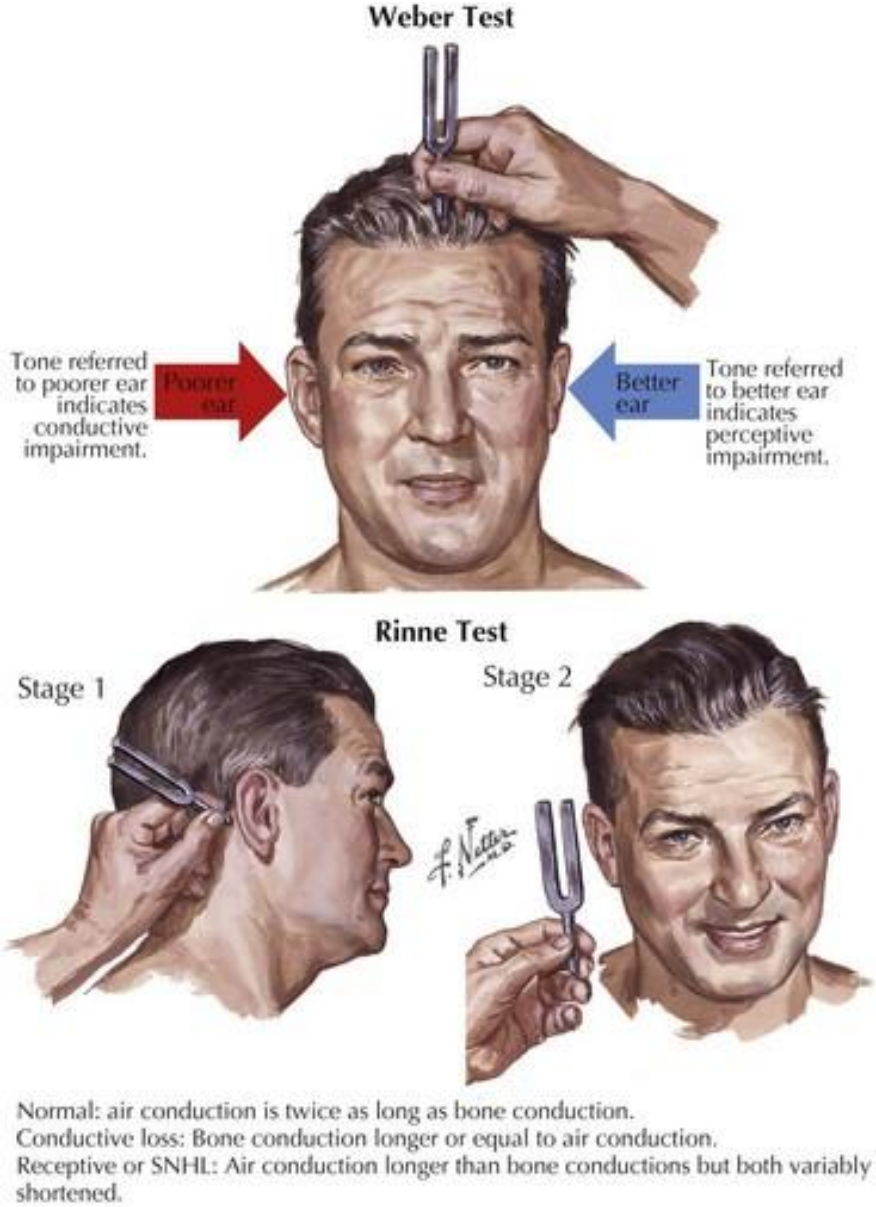
EXAMINATION OF CRANIAL NERVES –VIII – vestibulocochlear nerve - anatomy

- Two branches: vestibular (balance) and cochlear (acustic) nerve
- **cochlear nerve**
  - 1. neuron – bipolar cells in ganglion spirale Corti travels via meatus acusticus internus and pontocerebellar angle to brainstam
  - 2. neuron – auditory nucleii in brainstem (pons) travels as lemniscus lateralis to colliculi inferiores and corpus geniculatum mediale (3. neuron) than to cortical area 41 and 42
- **vestibular nerve**
  - 1. neuron - specific sensory cells in semicircular canals and maculae of the saccule and utricle travels via meatus acusticus internus and pontocerebellar angle to brainstam (pons)
  - 2. neuron – connected to cerebellum and ocular movement system; travels to vestibulospinal tract and cortex



VIII – vestibulocochlear nerve – examination – cochlear nerve

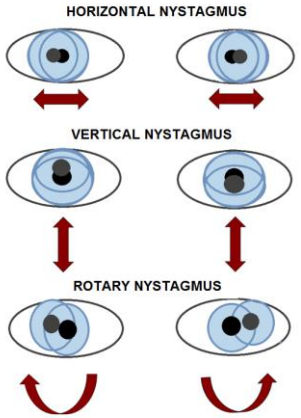
- Check the patient's ability to hear a finger rub or whispered voice with each ear.
- Further testing for air versus mastoid bone conduction (Rinne) and lateralization of a 512-Hz tuning fork placed at the center of the forehead (Weber) should be done if an abnormality is detected by history or examination.
- Any suspected problem should be followed up with formal audiometry.
- Ask for tinnitus („perception of sound in proximity to the head in the absence of an external source").
- Pathology: hypacusis/anacusis; tinnitus

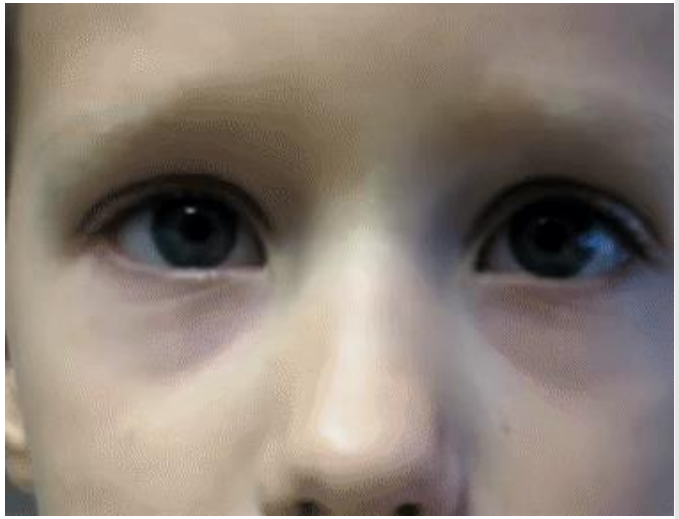


Netter's Clinical Anatomy - 4th Edition. John T. Hansen.

VIII – vestibulocochlear nerve – examination – vestibular nerve

- Problems with balance? Vertigo?
- Hautant test: The patient is seated. The examiner stands close to the patient. The patient flexes both arms to 90 with forearms supinated. The patient is instructed to close their eyes – examiner observes drifting of one of the arms.
- Romberg test:
  - I = the subject to stand up
  - II = the subject stands with feet together, eyes open
  - III = the subject stands with feet together and closes the eyes
  - IV + V = III + head to the right/left sice
- Gait: normal + eyes closed





VIII – vestibulocochlear nerve – examination – vestibular nerve

- Vertigo + vomiting
- Nystagmus
  - Involuntary eye movement, acquired in infancy or later in life, that may result in reduced or limited vision
  - Horizontal x vertical x rotatoric
  - Direction - named according to quick component of nystagmus (but the pathogenic is the slow component of nystagmus)
  - Intensitiy
    - I. Degree - moderate lateral gaze
    - II. Degree - spontaneous on forward gaze
    - III. Degree – forvard and both sides
  - Speed (slow x fast)
  - Amplitude (fine x coarse)

http://resumepedia.us/; http://commons.wikimedia.org/

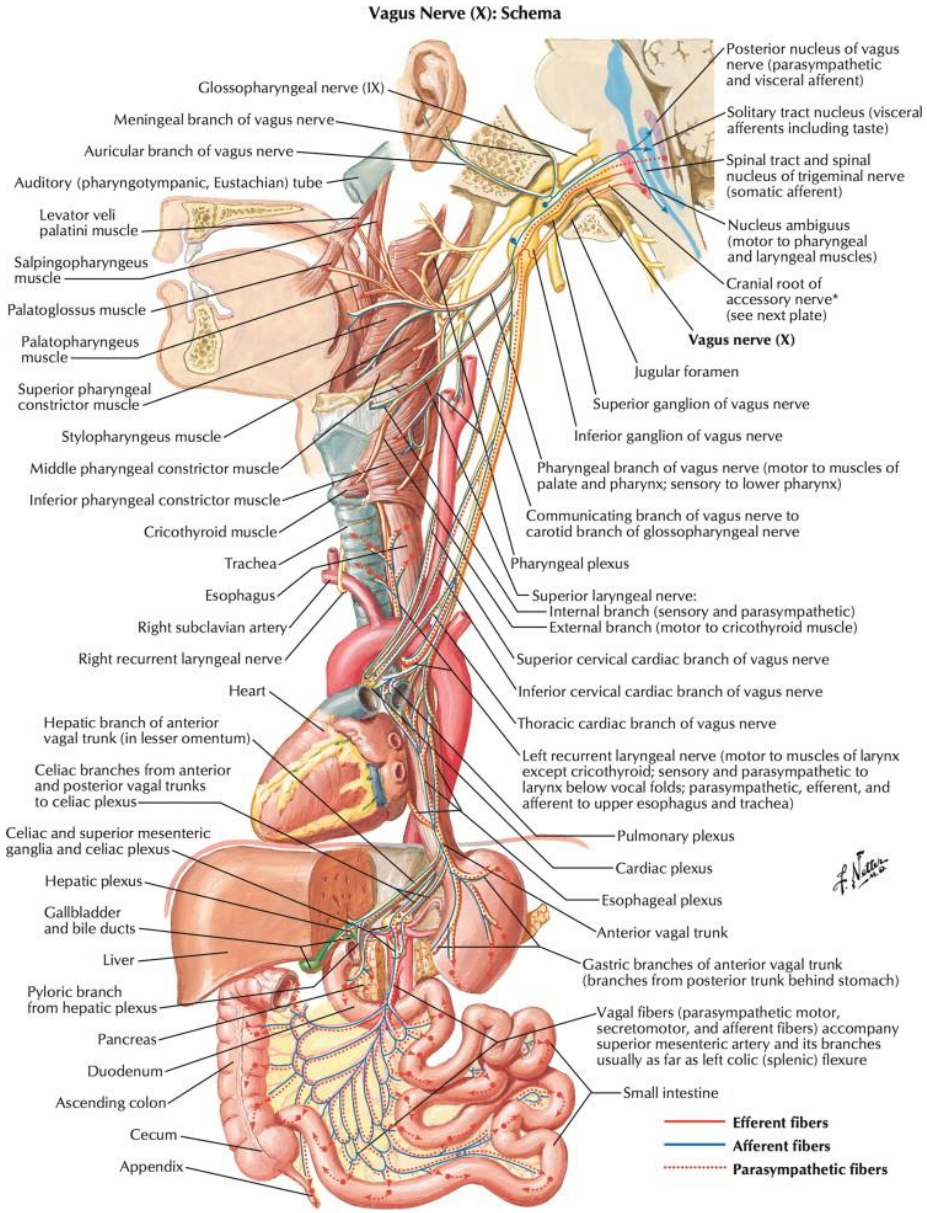
VIII – vestibulocochlear nerve – examination – vestibular nerve syndromes

- Peripheral vestibular dysfunction
  - Result from any lesion affecting the vestibular nerve, the receptors, or the structures that house the receptors. Clinical signs may consist of vestibular ataxia, ipsilateral Hautant/Romberg, and nystagmus in the direction away from the lesion.
  - Usually last no longer than one week.
- Central vestibular dysfunction
  - Central vestibular dysfunction may look peripheral, but peripheral vestibular dysfunction will never look central.
  - May include vertical nystagmus or nystagmus that changes direction, a Hautant/Romberg test can be towards or away from the lesion.

	Central	Peripheral
Imbalance	Severe	Mild to moderate
Neurologic symptoms	Frequent	Rare
Nystagmus	Changes direction in different gaze positions; no change with visual fixation	Unidirectional in all gaze positions; decreases with visual fixation
Hearing loss	Rare	Frequent
Nausea	Variable, may be absent	Severe
Recovery (central compensation)		Rapid

EXAMINATION OF CRANIAL NERVES – IX-X

- **The glossopharyngeal nerve (IX)** innervates the muscles from the third embryonic branchial arch and sends preganglionic parasympathetics from the inferior salivatory nucleus via the lesser petrosal nerve to the otic ganglion.
- **The vagus nerve (X)** innervates the pharyngeal and laryngeal muscles of the fourth embryonic branchial arch and sends preganglionic parasympathetic fibers from its dorsal nucleus to smooth muscle and glands of the neck, thorax (including cardiac muscle of the heart), and proximal two thirds of the abdominal GI tract.
    - Vagal aferents arise from visceral structures of the same thoracic and GI regions and from aortic baroreceptors and chemoreceptors that course to the brainstem. Special sensory fibers from taste buds on the epiglottis and general somatic aferents arising from skin around the ear, larynx, external acoustic meatus, and posterior dura mater also travel in the vagus nerve.



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EXAMINATION OF CRANIAL NERVES – IX-X

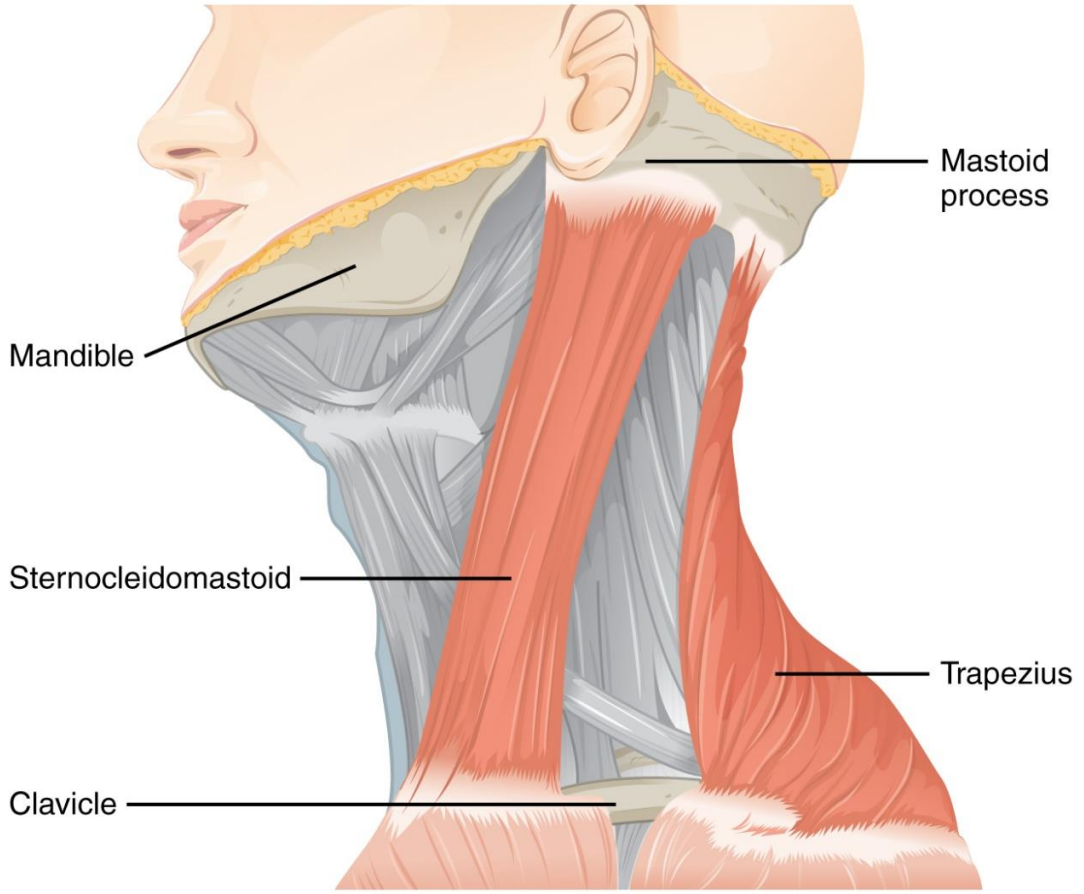
- Observe the position and symmetry of the palate and uvula at rest and with phonation (“aah”).
- The pharyngeal (“gag”) reflex is evaluated by stimulating the posterior pharyngeal wall on each side with a sterile, blunt object (e.g. by cotton wool bud), but the reflex is sometimes absent in normal individuals.

- IX, X: ***Palate (uvula) in rest in the midline position,***
  - ***lifts symetrically*** during phonation,
  - ***no dysarthria, no dysphonia*** (hoarse voice)

- *Palate reflex*: particular (left or right) palatine arch lifts slighly in a response to slight touch of particular arch

EXAMINATION OF CRANIAL NERVES – XI – accesory nerve

- The spinal accessory nerve innervates the sternocleidomastoid and trapezius muscles.
- The sternocleidomastoid is tested by asking the patient to rotate the head against resistance provided by the examiner's hand, which is placed on the patient's jaw. Sternocleidomastoid weakness results in decreased ability to rotate the head away from the weak side.
- The trapezius is tested by having the patient shrug the shoulders against resistance and noting any asymmetry.



http://en.wikipedia.org/

EXAMINATION OF CRANIAL NERVES – XII - hypoglossal

- The hypoglossal nerve innervates the tongue muscles.
- It can be tested by having the patient push the tongue against the inside of the cheek while the examiner presses on the outside of the cheek. With unilateral tongue weakness, the ability to press against the opposite cheek is reduced.
- There may be also deviation of the protruded tongue toward the weak side, although facial weakness may result in false-positive tests. Tongue weakness also produces dysarthria with prominent slurring of labial sounds.
- Finally, denervation of the tongue may be associated with wasting (atrophy) and twitching (fasciculation).

http://wikivisually.com/

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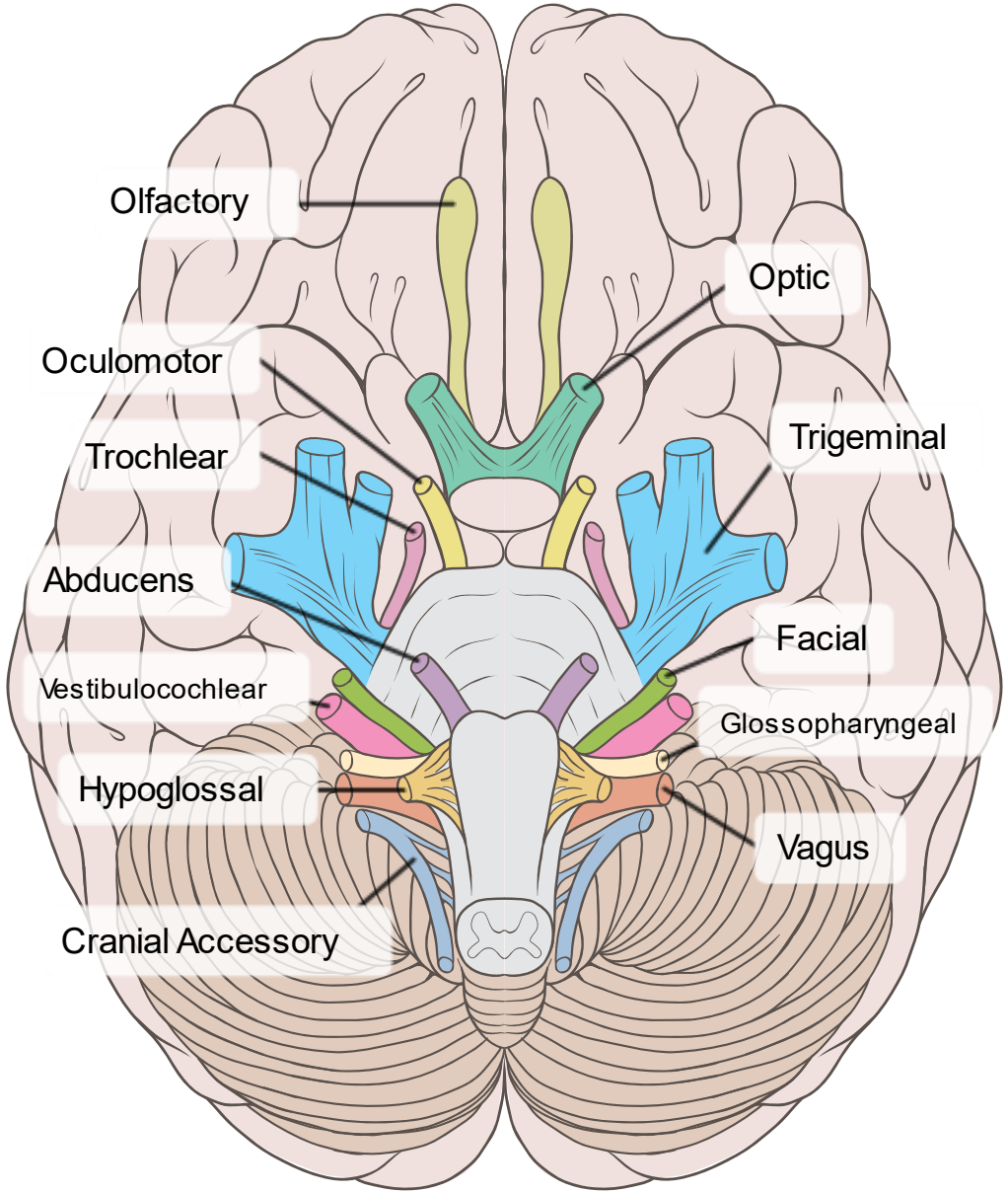


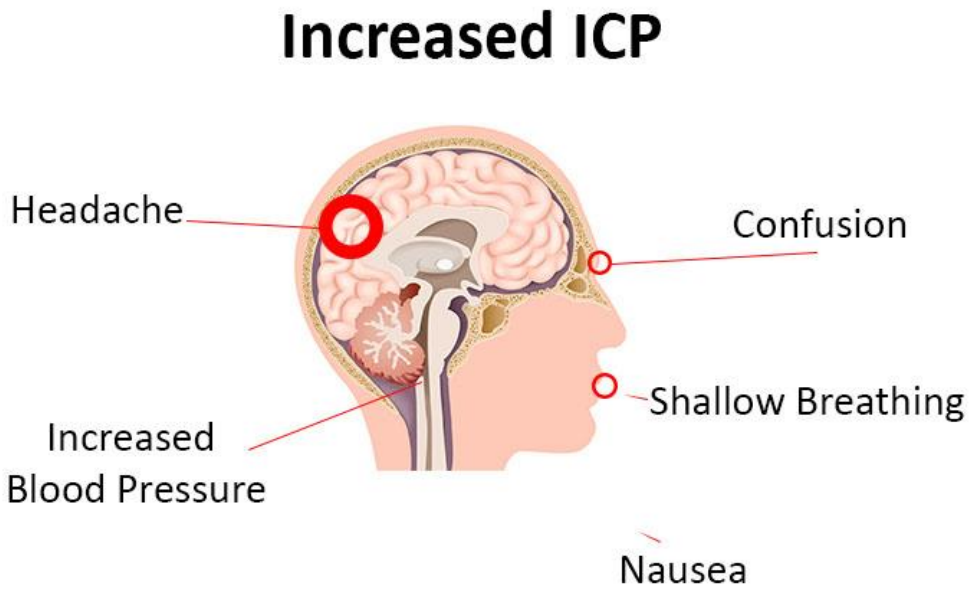
Intracranial hypertension

Intracranial pressure

- Intracranial pressure is the pressure exerted by fluids such as cerebrospinal fluid inside the skull and on the brain tissue. Normal pressure is 7–15 mmHg for a supine adult.
- The body has various mechanisms by which it keeps the ICP stable, with CSF pressures varying by about 1 mmHg in normal adults through shifts in production and absorption of CSF. Changes in ICP are attributed to volume changes in one or more of the constituents contained in the cranium.

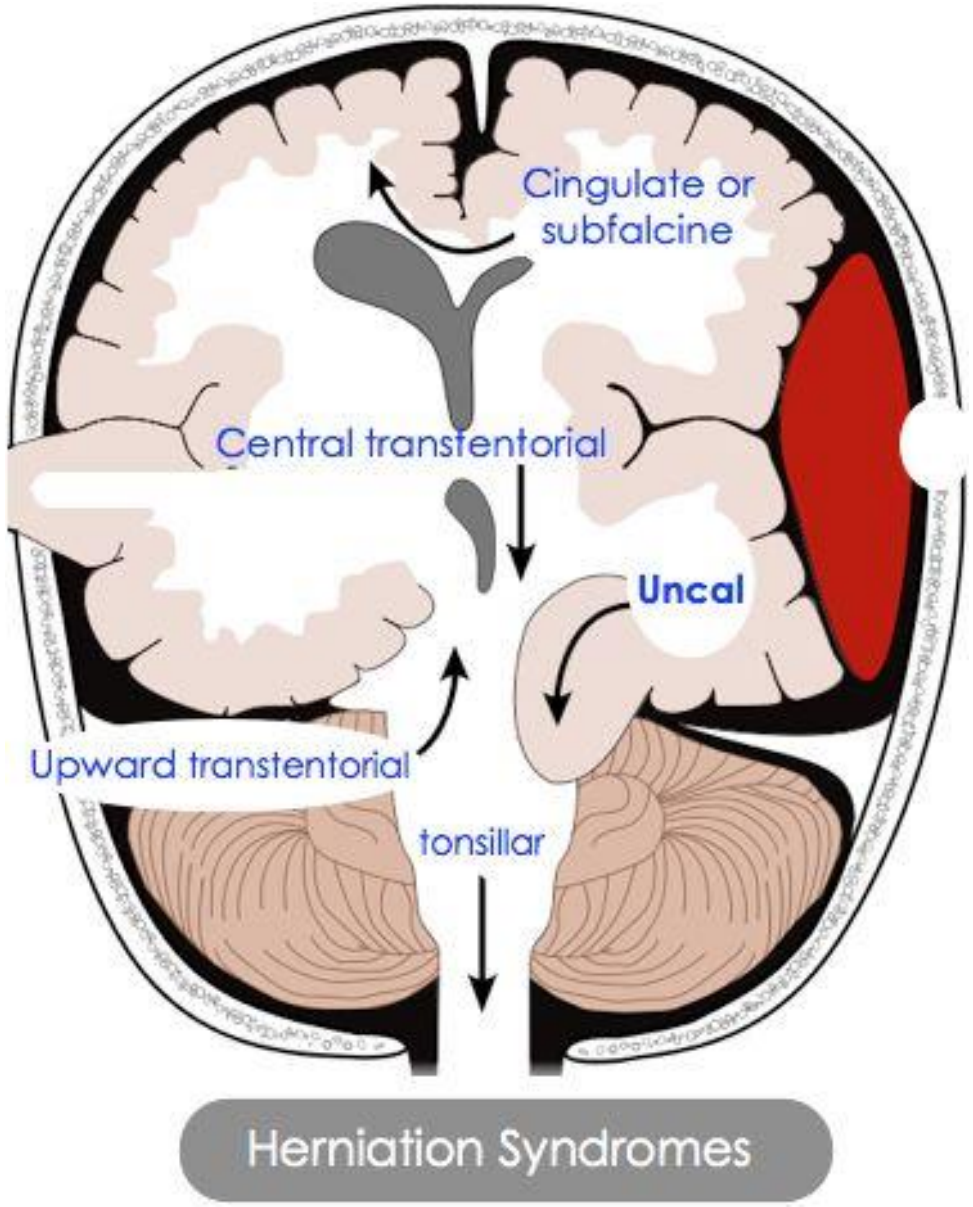
Intracranial hypertension

- Intracranial hypertension is elevation of the pressure in the cranium. ICP is normally 7–15 mm Hg; at 20–25 mm Hg, the upper limit of normal, treatment to reduce ICP may be needed.
  - Headache, worse in laying-down position.
  - Vomiting – sudden without nausea
  - Vertigo
  - Papiledema
  - Bradykardia
  - VI. Nerve palsy
  - Epileptic seizures
  - Consciousness disturbances



Brain herniation

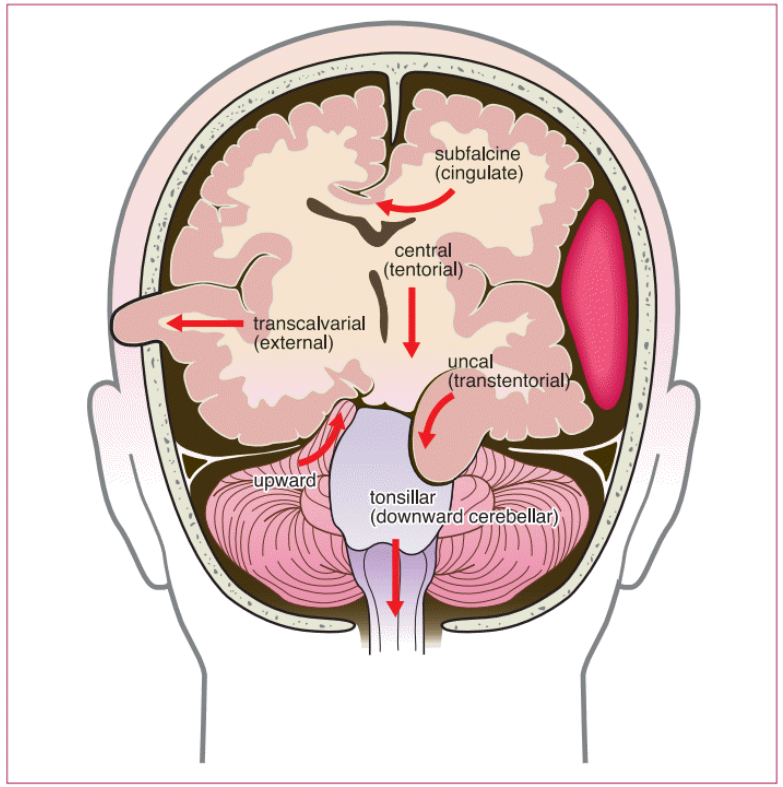
- Brain herniation is a potentially deadly side effect of very high pressure within the skull that occurs when a part of the brain is squeezed across structures within the skull.
- The brain can shift across such structures as the falx cerebri, the tentorium cerebelli, and even through the foramen magnum.
- Herniation can be caused by a number of factors that cause a mass effect and increase intracranial pressure:
  - traumatic brain injury
  - intracranial hemorrhage
  - brain tumor
  - etc.

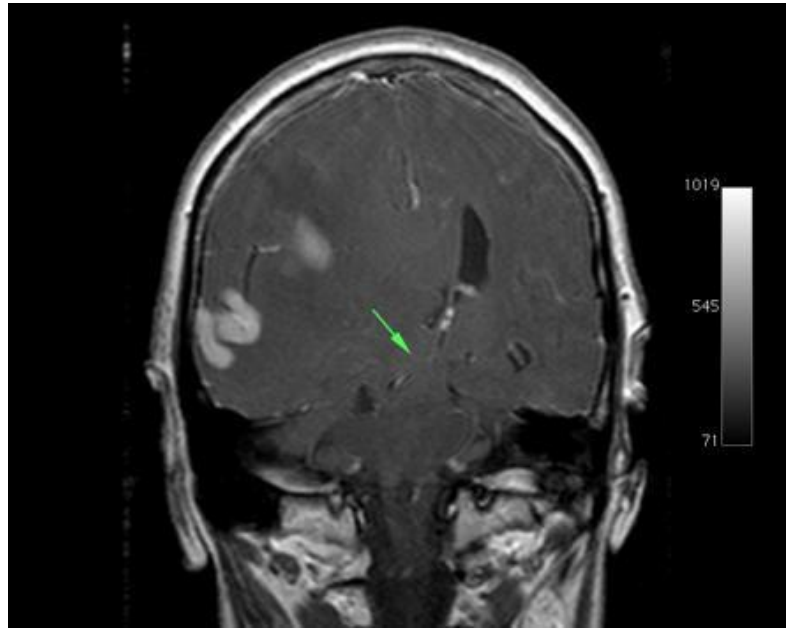


http://emergencymedicinecases.com/

Brain herniation

- Brain herniation is classified based on the structure through which tissue is herniated:
  - **Transtentorial (uncal) herniation:** The medial temporal lobe is squeezed by a unilateral mass across and under the tentorium that supports the temporal lobe. N III. Palsy and homolateral mydriasis.
  - **Subfalcine herniation:** The cingulate gyrus is pushed under the falx cerebri by an expanding mass high in a cerebral hemisphere resulting to compression of a. cerebri anterior.
  - **Central herniation:** Both temporal lobes herniate through the tentorial notch because of bilateral mass effects or diffuse brain edema.
  - **Upward transtentorial herniation:** This type can occur when an infratentorial mass compresses the brain stem, kinking it and causingbrain stem ischemia.
  - **Tonsillar herniation:** Usually, the cause is an expanding infratentorial mass (eg, cerebellar hemorrhage), forcing the cerebellar tonsils, through the foramen magnum and death.





http://radiopaedia.org/

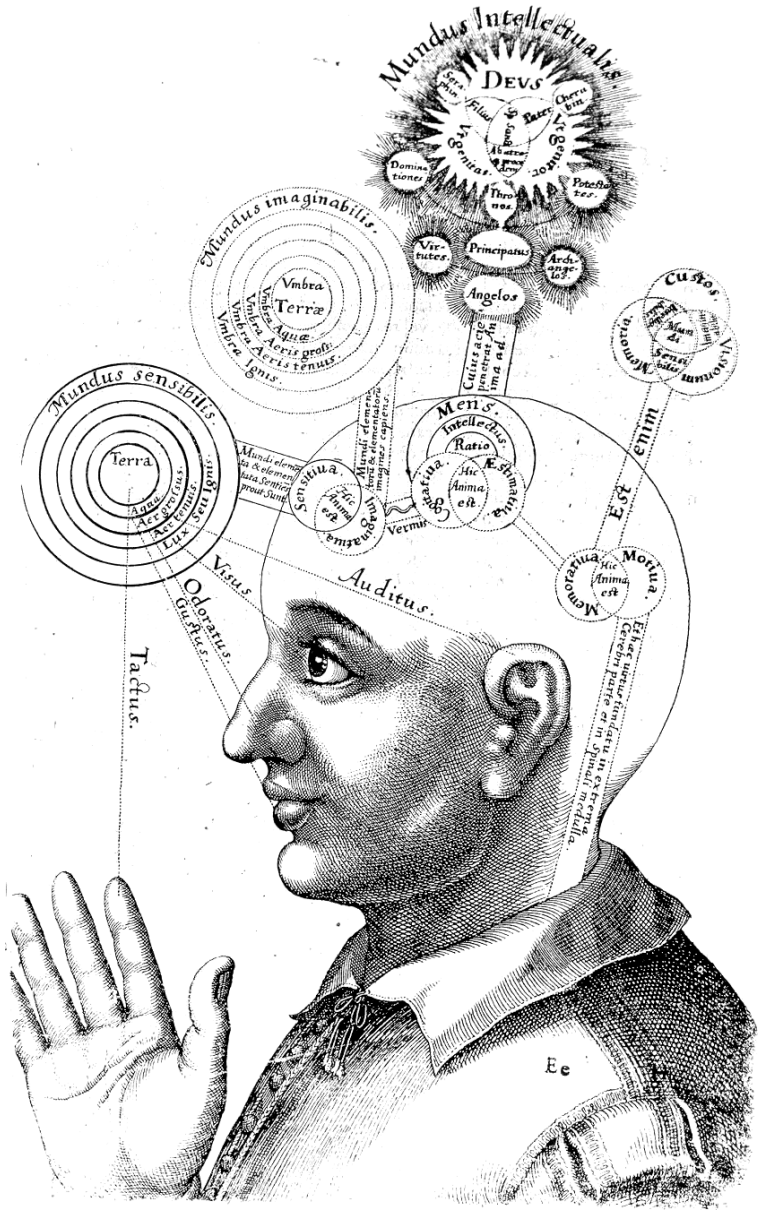
Intracranial hypotension

- Intracranial hypotension is a condition in which there is negative pressure within the brain cavity.
- There are several possible causes:
  - Cerebrospinal fluid (CSF) leak from the spinal canal: A leak following a lumbar puncture.
  - A defect in the dura mater.
  - A congenital weakness.
  - Following spinal surgery.
  - Following spinal trauma.
  - Ventriculoperitoneal shunt with a low pressure valve.
  - In some cases, spinal CSF leaks can lead to a descent of the cerebellar tonsils into the spinal canal, similar to a Chiari malformation.
- The classic **symptom** is severe headache when upright, which is relieved when lying flat. Other symptoms can include nausea, vomiting, double vision and difficulty with concentration.

Consciousness disturbances

Consciousness disturbances

- Consciousness is awareness of the internal and external world, and the level of consciousness is described in terms of the patient's apparent state of wakefulness and response to stimuli.
- **A patient with a normal level** of consciousness is **awake** (or can be easily awakened), **alert** (responds appropriately to visual or verbal cues), and **oriented** (knows who and where he or she is and the approximate date and time).
- Abnormal consciousness represents a continuum ranging from mild sleepiness to unarousable unresponsiveness (coma).
- Depressed consciousness should be characterized more precisely in terms of the stimulus–response patterns observed.
- Progressively more severe impairment of consciousness requires stimuli of increasing intensity to elicit increasingly primitive (nonpurposeful or reflexive) responses
- **Assessed while recording the history.**



http://wikivisually.com/

Consciousness disturbances

TWO TYPES OF ALTERATION OF THE CONSCIOUSNESS (a state of awareness of self and surroundings):

- The first type AFFECTS AROUSAL (sometimes called „quantiatative“)
- described on four levels (not very accurate –continuum of subtly changing status)
  - *ALERT* – perfectly normal state of arousal
  - *LETHARGY* (drowsinness) – between alertness and stupor („sleepy“, fine stimuli enough)
  - *STUPOR* – a state of baseline unresponsiveness that requires repeated application of vigorous stimuli to achieve arousal
  - *COMA* - coma is a state of complete unresponsiveness to arousal, in which the patient lies with the eyes closed

THE GLASGOW COMA SCALE

- Widely used scale to assess the initial severity of traumatic brain injury.
- Separate assessment of three aspects of a patient's behavior:
  - the stimulus required to induce eye opening
  - the best motor response
  - the best verbal response
- Each symptom is scored, sum score is obtained.
- Simple and reproducible.
- An ideal method of assessment for non-neurologists involved in the care of comatose patients.
- Limited use in patients who are intubated or who have suffered facial trauma or have aphasia.

Domain	Response	Score
Eye opening	Spontaneous	4
	To speech	3
	To pain	2
	None	1
Best verbal response	Oriented	5
	Confused	4
	Inappropriate	3
	Incomprehensible	2
	None	1
Best motor response	Obeying	6
	Localizing	5
	Withdrawal	4
	Flexing	5
	Extending	3
	None	1
Total score	Deep coma or death	3
	Fully alert and oriented	15

Consciousness disturbances

- The second type INVOLVES COGNITIVE AND AFFECTIVE MENTAL FUNCTION
- sometimes referred to as the "content" of mental function
- dementia, delusions, confusion

http://dementiaguide.ca/

symptoma.com

Delirium

- **Delirium**, also known as "**acute confusional state"**, is an organically caused decline from a previous baseline mental functioning that develops over a short period of time, typically hours to days.
- Delirium is a syndrome encompassing disturbances in attention, consciousness, and cognition. It may also involve other neurological deficits, such as psychomotor disturbances (e.g. hyperactive, hypoactive, or mixed), impaired sleep-wake cycle, emotional disturbances, and perceptual disturbances (e.g. hallucinations and delusions), although these features are not required for diagnosis.
- Delirium is caused by an acute organic process, which is a physically identifiable structural, functional, or chemical problem in the brain that may arise from a disease process outside the brain that nonetheless affects the brain.
- Delirium x delirium tremes!

EXAMINATION OF THE COMATOSE PATIENT

The examiner should first assess the degree of coma (The Glasgow Coma Scale).

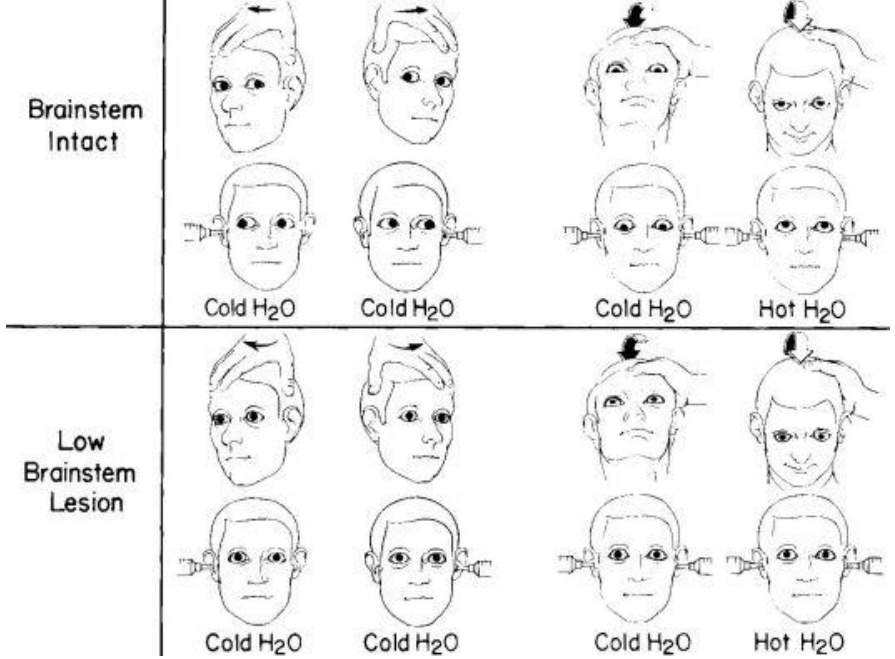
If the patient is determined to be in coma based on the GCS <8, the examiner should then determine whether brain stem functions are intact.

Breathing - From cranial to caudal:

- Cheyne-Stokes respiration – a pattern of alternating hyperventilation and hypoventilation seen with bilateral cerebral dysfunction or basal ganglia lesions.
- Central neurogenic hyperventilation, produces rapid respiratory rate of up to 40-70/minute and is seen with midbrain dysfunction.
- Apneustic breathing refers to slow breathing with a prolonged pause after inspiration. This occurs with pontine dysfunction.
- Ataxic (Biot's) respirations refer to irregular respiratory frequency and amplitude and indicates medullary dysfunction with impending respiratory arrest.
- The respiratory patterns may occur in a step-wise pattern in transtentorial herniation.

EXAMINATION OF THE COMATOSE PATIENT

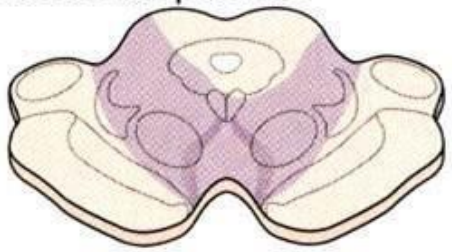
- **Pupillary Reactions:** Examination should be aimed at determining the size of each pupil and its direct and consensual reaction to the light. The size and equality of the pupils may give localizing clues.
- **Eye Movements:**
  - Sustained lateral conjugate deviation of the eyes suggests disease of the ipsilateral cerebral hemisphere or contralateral pons.
  - Downward deviation of the eyes with convergence is seen with thalamic or dorsal midbrain lesions.
  - Ocular bobbing (rapid downward movements, slow return to primary position) classically occurs with pontine lesions.
- **Corneal Reflex:** The corneal reflex tests the reflex arc of V1 to VII.
  - The afferent limb tested via corneal stimulation via the opthalamic division of the trigeminal nerve (V1).
  - The efferent limb in the contraction of the obicularis oculi (VII).



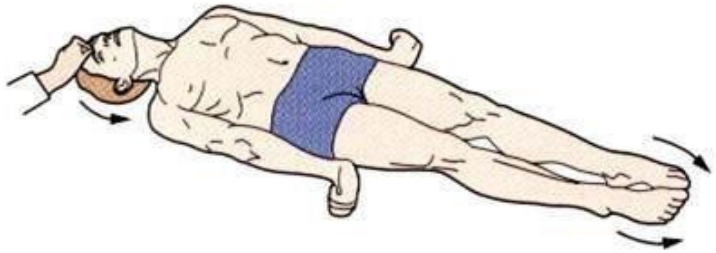
http://kaltura.com/

EXAMINATION OF THE COMATOSE PATIENT

Upper midbrain damage
Decorticate posture



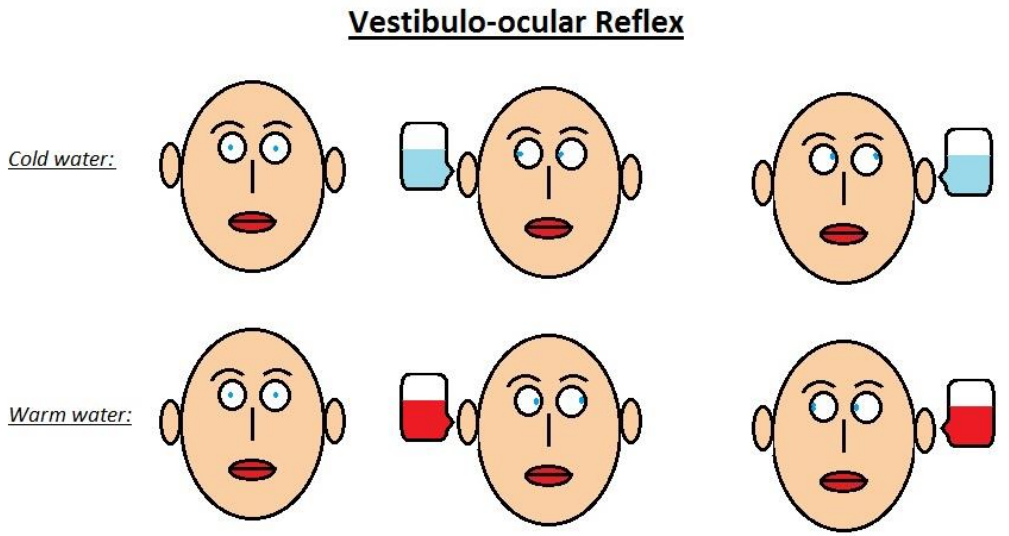
Upper pontine damage
Decerebrate posture



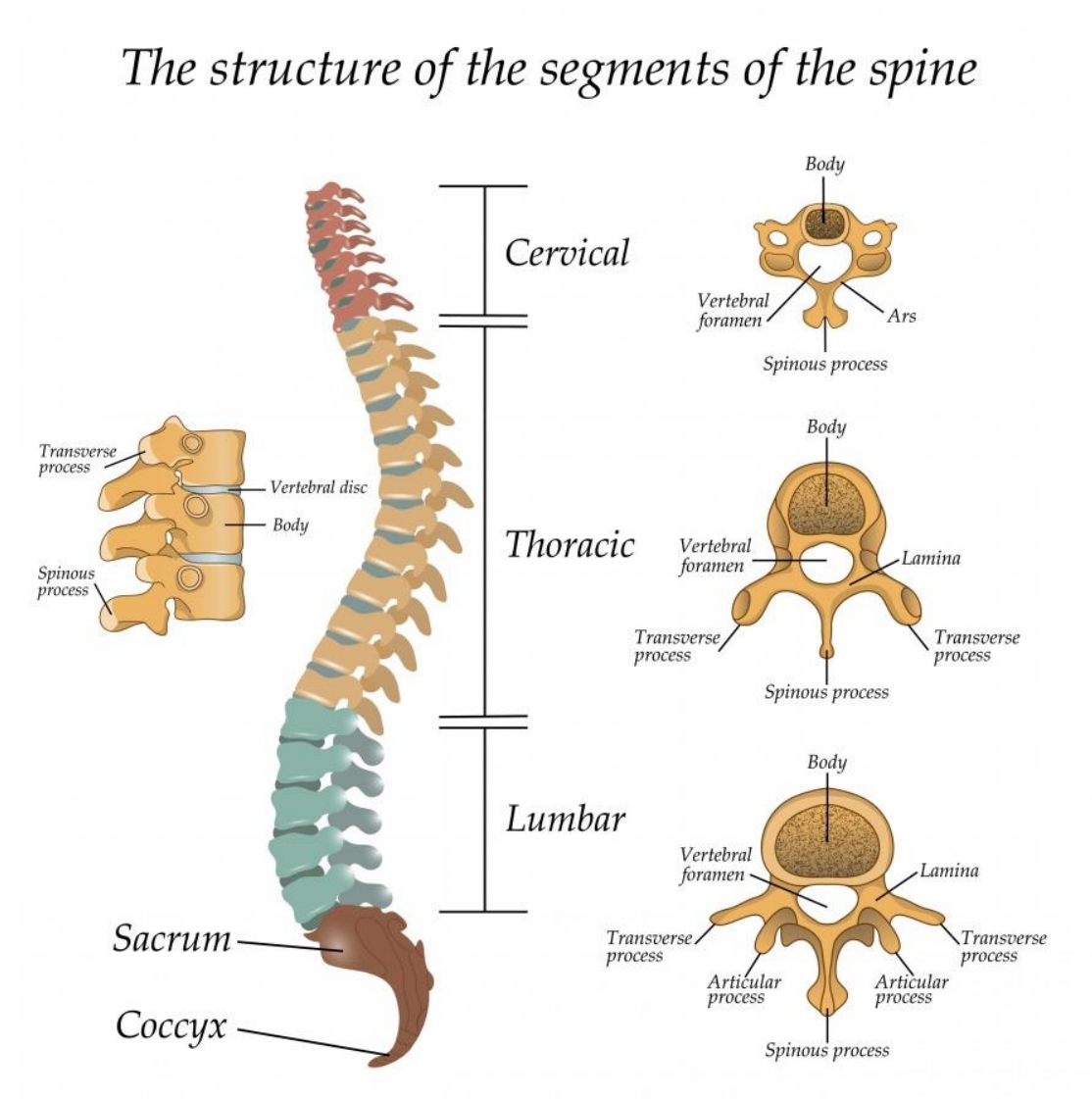
Ganong's Review of Medical Physiology - Kim E. Barrett et al., 25th Edition.

Diagnosis of brain death – neurology examination

- Components of a complete neurological examination are:
  - Examination of the patient-absence of spontaneous movement, decerebrate or decorticate posturing, response to verbal stimuli, and response to noxious stimuli administered through a cranial nerve pathway.
  - During the examination spinal reflexes may be present.
  - Absent pupillary reflex.
  - Absent corneal, oculocephalic, cough and gag reflexes.
  - Absent oculovestibular reflex when tested with 20 to 50 ml. Of ice water irrigated into an external auditory canal clear of cerumen, and after elevating the patients head 30'. Labyrinthine injury or disease, anticholinergics, anticonvulsants, tricyclic antidepressants, and some sedatives may alter response.

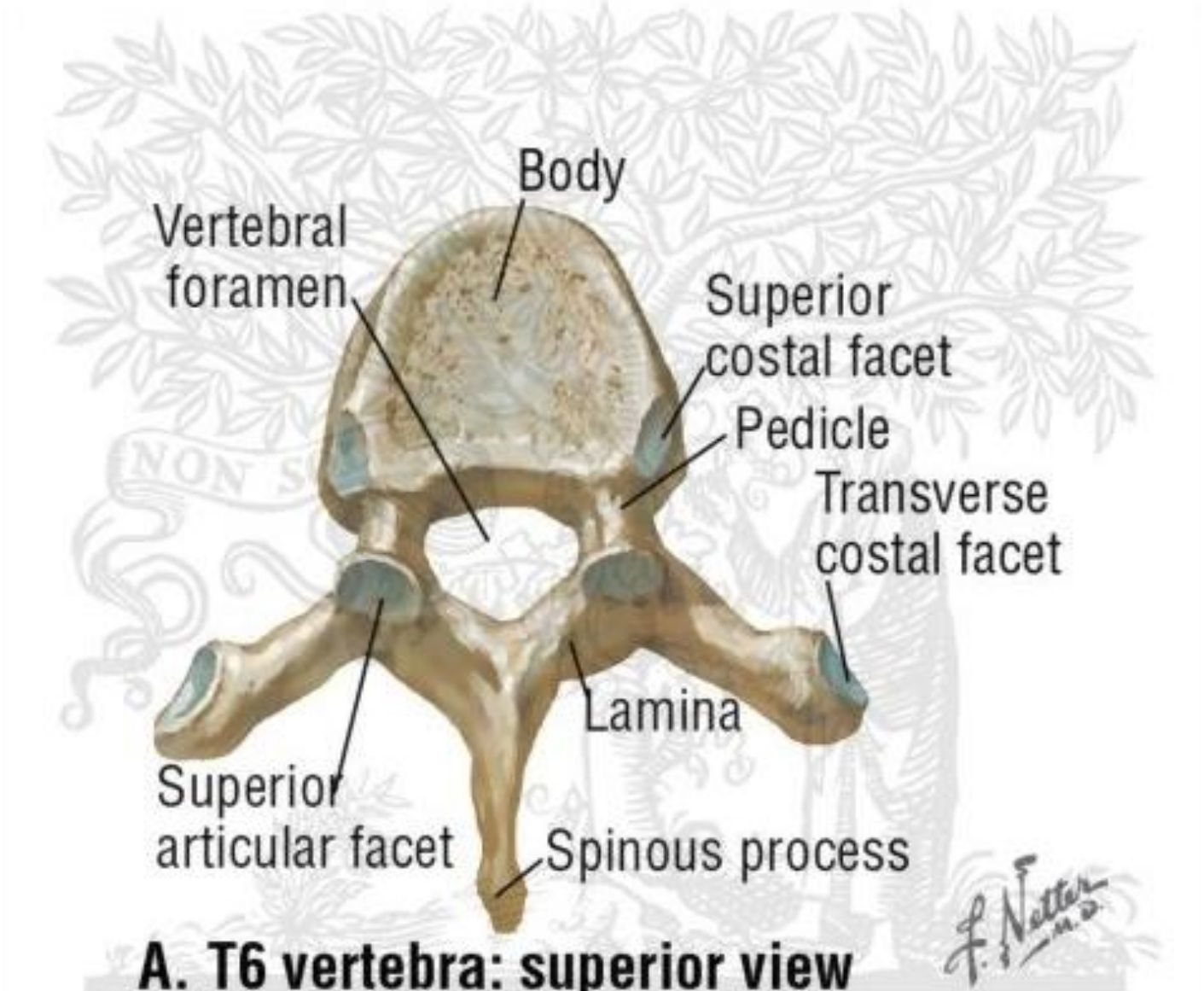


Vertebral column examination

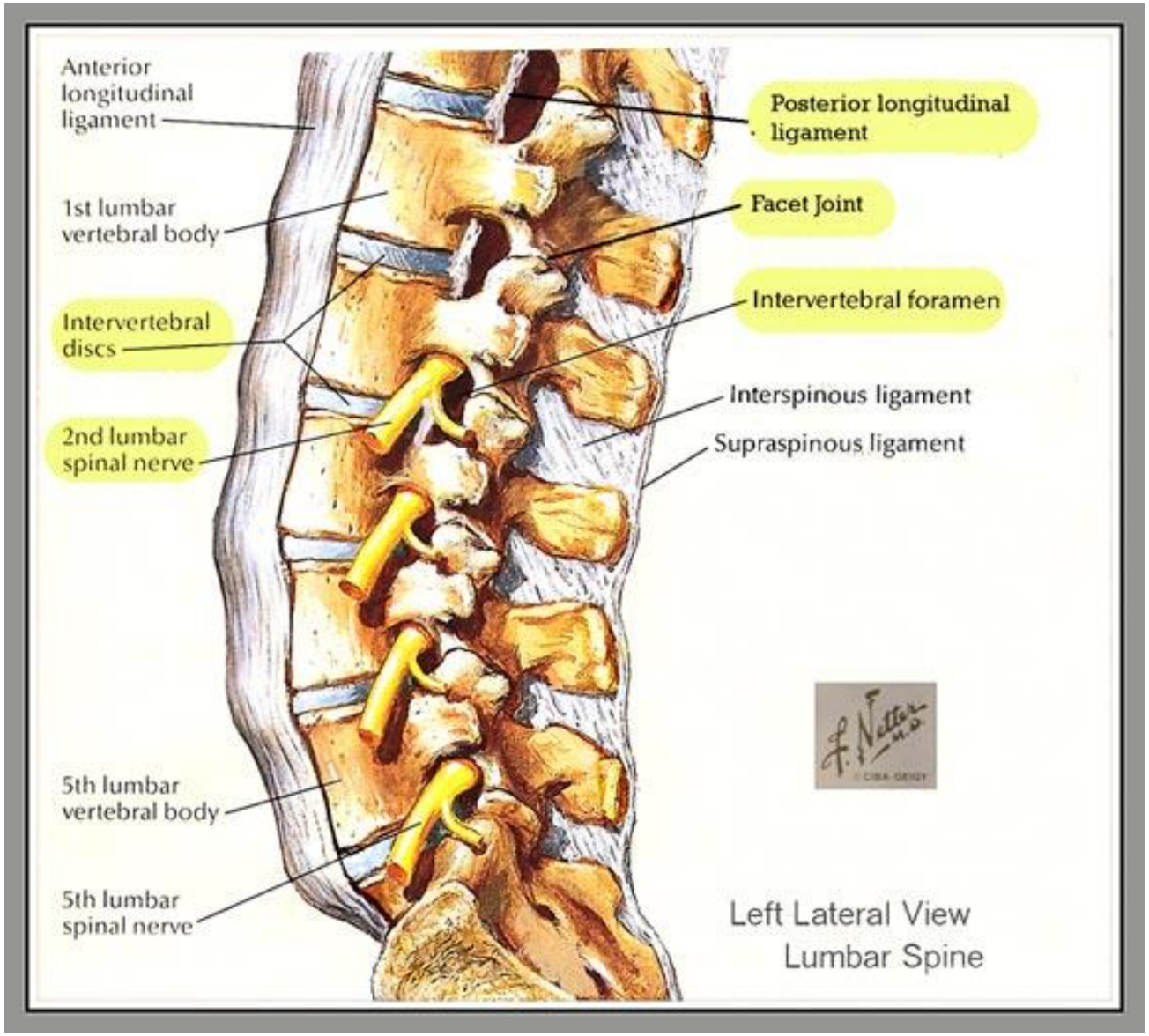


http://anatomychartee.co/

A typical Vertebra – anatomy (superior view)



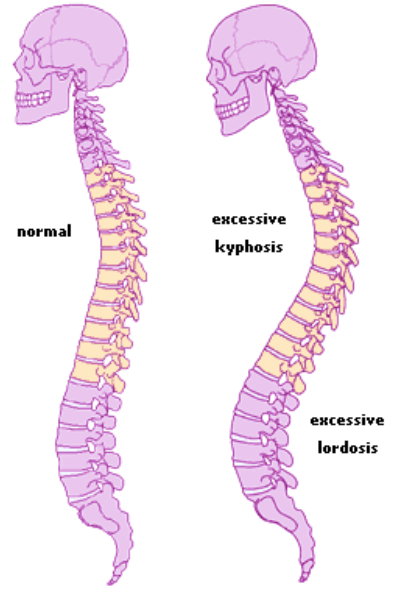
Netter's Clinical Anatomy - 4th Edition. John T. Hansen.

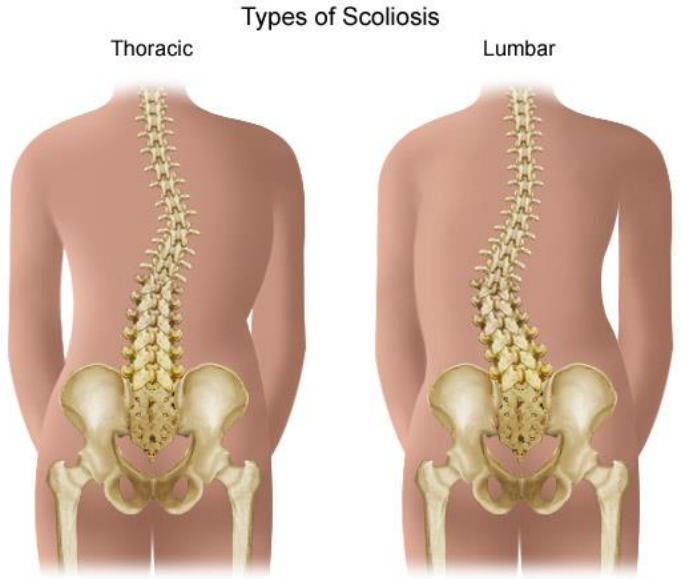


Netter's Clinical Anatomy - 4th Edition. John T. Hansen.

Vertebral column examination

- The normal spine has a cervical and lumbar lordosis, and a thoracic kyphosis. Exaggeration of these normal alignments may result in hyperkyphosis of the thoracic spine or hyperlordosis of the lumbar spine.
- Inspection may reveal a lateral curvature of the spine (scoliosis) or an asymmetry in the prominence of the paraspinal muscles, suggesting muscle spasm.
- Back pain of bony spine origin is often reproduced by palpation or percussion over the spinous process of the affected vertebrae.
- The neurologic examination includes a search for focal weakness or muscle atrophy, or signs of spine injury.

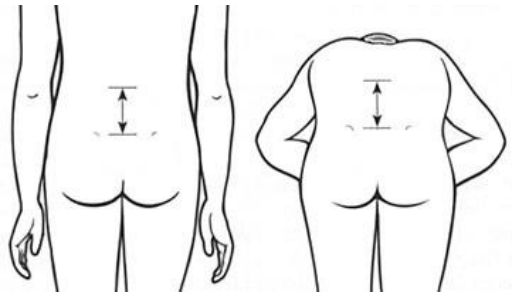


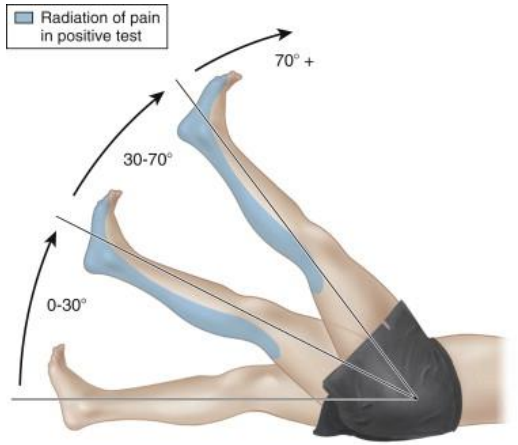


https://encyclopedia.nm.org/

Vertebral column examination

- Lasegue sign
  - Among the most helpful signs in detecting nerve root compression is passive straight-leg raising (possible up to almost 90 degrees in normal individuals) with the patient supine.
  - This maneuver is the usual way in which compression of the LS or Sl nerve root is detected.
- Thomayer test
- Schober's test
  - While the patient is in a standing position the examiner makes a mark approximately at the level of L5. Two points are marked: 5 cm below and 10 cm above this point (for a total of 15 cm distance). Then the patient is asked to touch his toes while keeping the knees straight. If the distance of the two points do not increase by at least 5 cm (with the total distance greater than 20 cm), then this is a sign of restriction in the lumbar flexion.





Cs.wikipedia.org; http://nle.nottingham.ac.uk/; George V. Lawry, Adel G. Fam, et al. Fam's Musculoskeletal Examination and Joint Injection Techniques (Second Edition), 2010.

Terminology of spinal degenerative changes

SPONDYLOSIS (IN THE BROAD SENSE) –a complex of degenerative and proliferative changes of the spinal column

SPONDYLOSIS (IN THE STRICT SENSE) – proliferative changes in ***spinal vertebrae bodies*** with formation of osteophytes

SPONDYLATRHROSIS (SPONDYLARTHROPATHY) – degenerative changes of ***intervertebral joints*** (so called facet joints)

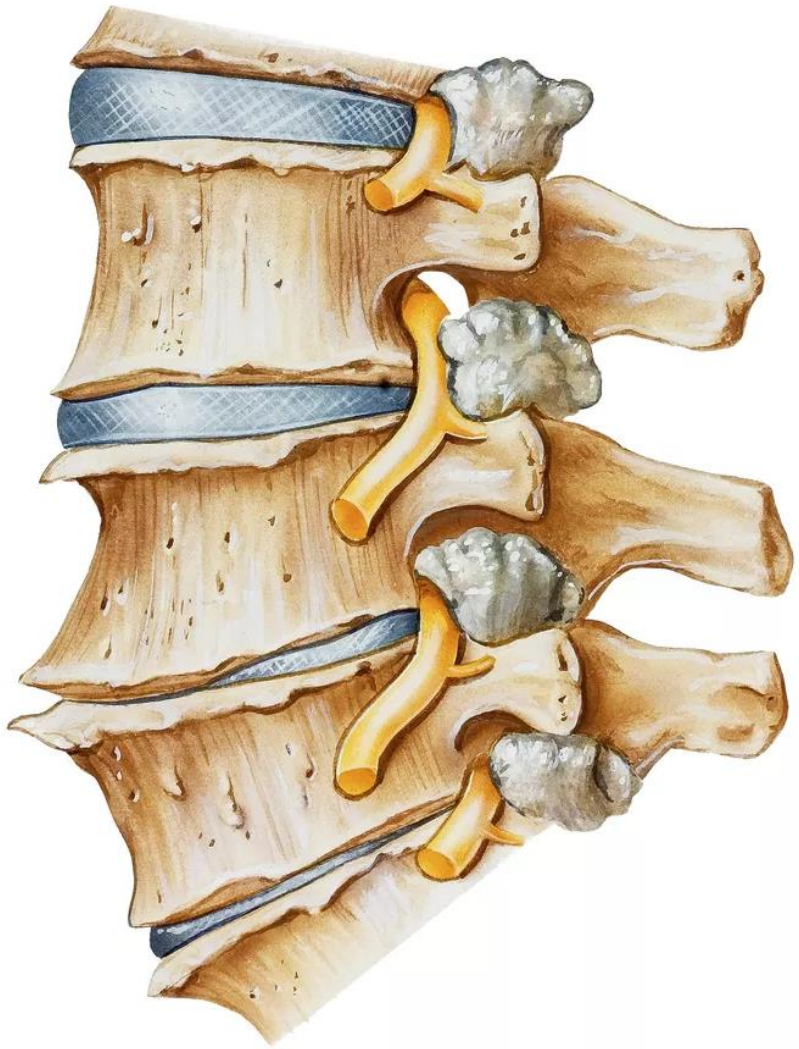
INTERVERTEBRAL DISC DISORDERS - deterioration, herniation or other dysfunction of intervetrebral disc (secondary degenerative disc disease)

Patophysiology of degenerative changes

- **MULTIFACTORIAL**
- Usually starts in intervertebral **DISC**

- **Following FACTORS** may influence spinal degeneration development and progression :
  - age
  - smoking
  - mechanical overload
  - traumatic spinal injuries
  - genetical factors

- Clinical manifestation of spondylosis is mainly dependent of **CONGENITAL SPINAL CORD WIDTH**.



Netter's Clinical Anatomy - 4th Edition. John T. Hansen.

Clinical manifestation of spondylogenic disorders

So called SEGMENTAL SYNDROME (spine blockage) (e.g. lumbago)

- Pain + spinal dysfunction (block or hypermobility)
- = functional spondylogenic disorders

NON-RADICULAR SYNDROMES WITH IRADIATION OF PAIN ALONG THE RADICULAR ZONES

- Pain iradiading from spine to extremities or thorax, not specificaly in radicular distribution
- No objetive symptoms of radicular lesion

COMPRESIVE SPONDYLOGENIC SYNDROMES

- radiculopathy (radicular dysfunction)
- Or myelopathy (spinal cord dysfunction)
- About 1/10 of all back pains

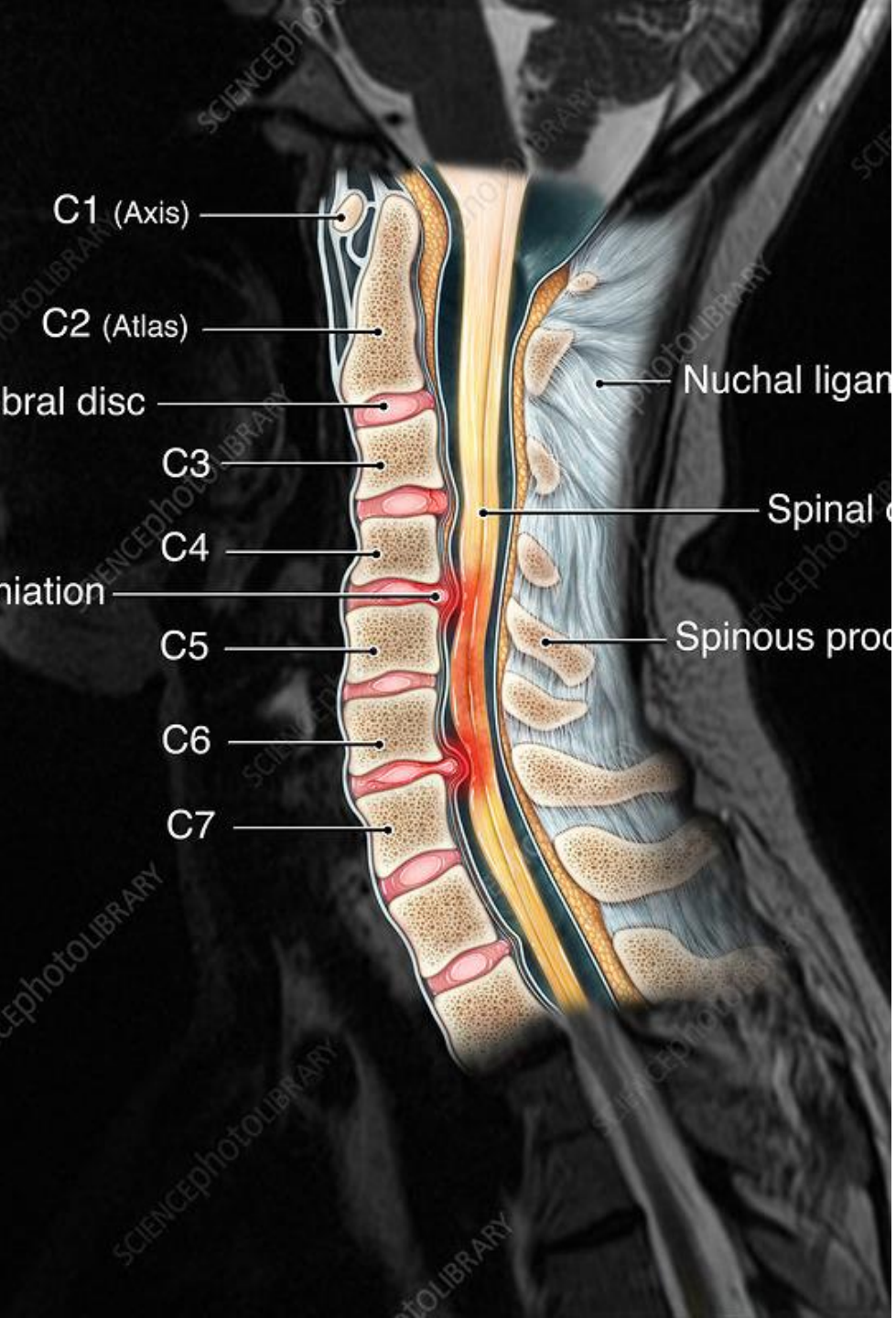
Clinical presentation of radiculopathies

SENSORY SYMPTOMS

- Positive:
  - Pain „radiated“ along the dermatome (evaluate by NRS)
  - Other: paresthesia, dysesthesia
- Negative
  - Hypesthesia

MOTOR SYMPTOMS

- Paresis in particular myotome
- hypo-areflexia of particular deep tendon reflexes
- Muscle hypotrophy



Cervical spine Clinical syndromes

SEGMENTAL SYNDROME – cervical spine blockage

NON-RADICULAR IRADIATING PAIN

- CERVICOBRACHIAL SYNDROME
- Very frequent, emotional accompaniment
- The most frequent cause = intervertebral joint affection

COMPRESIVE SPONDYLOGENIC SYNDROMES

- CERVICAL RADICULOPATHY (see below)
- SPONDYLOGENNIC CERVICAL MYELOPATHY
  - Cervical spinal cord dysfunction
  - Usually quadriparesis (quadriplegia)
  - Most frequently upper motor neuron affection in LE
  - + upper and/or lower motor neuron affection in UE
  - Spastic gait, clumsy hand
  - Cervical spinal pain – may not be present

http://mauritius-images.com/

Thoracic spine: Clinical syndromes

SEGMENTAL SYNDROME – thoracic spine block

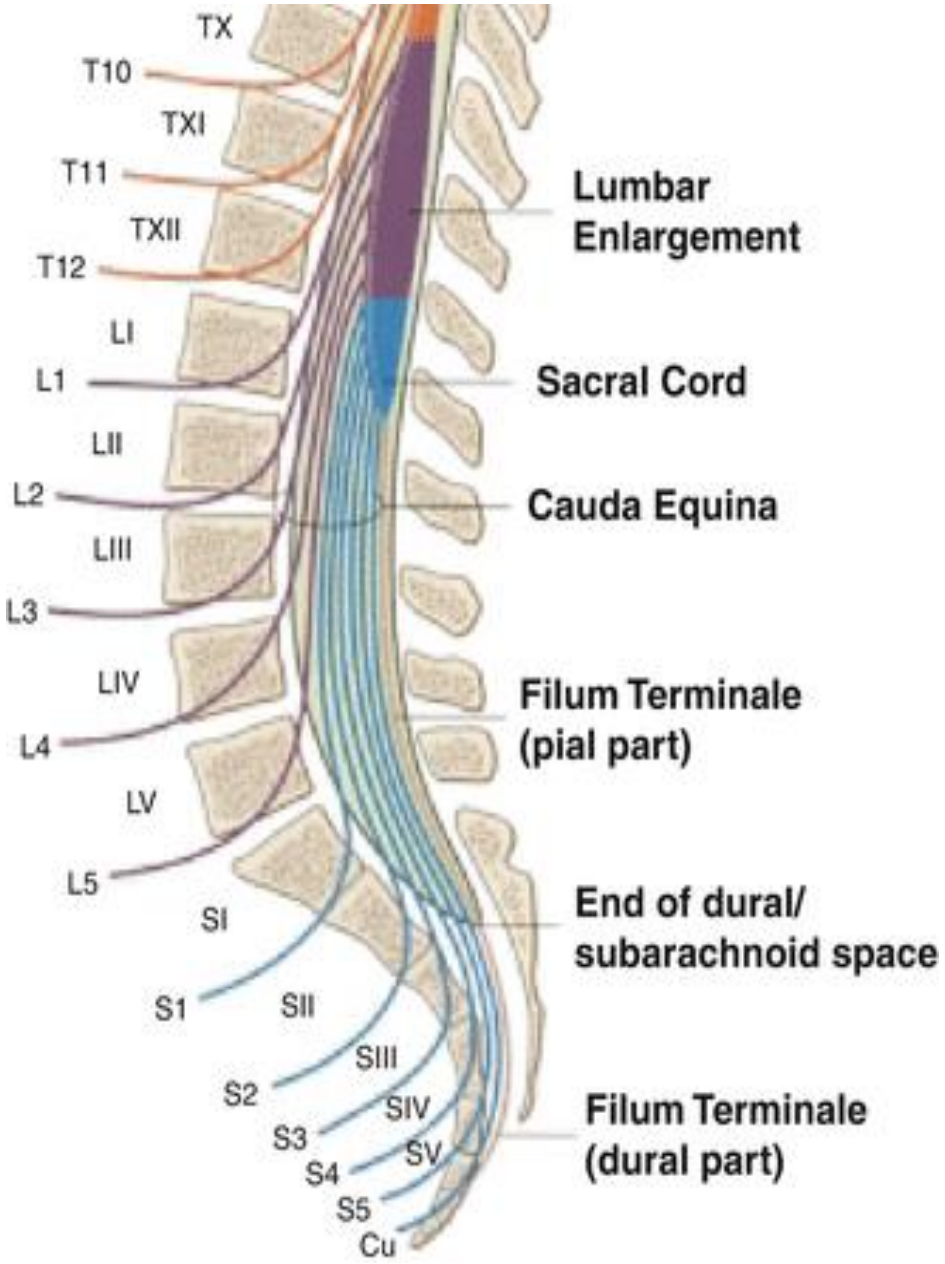
- Quite frequent

INTERCOSTAL NEURALGIA

- Usually caused by intervertebral joint blockage in thoracic spine region **WITH NON-RADICULAR IRADIATION**
- **RARELY RADICULOPATHY** (mostly secondary – radiculiris, postherpetic neuralgia).

SPONDYLOGENNIC THORACIC MYELOPATHY

- Very rare
- Presenting by spastic paraparesis:
  - upper motor neuron signs (weakness, spasticity, cltered tonus)
  - pathological hyperreflexia and Babinsky sign
  - sensory loss – sensory level (Nipples T4, umbilicus T10)
  - bowel/bladder symptoms and sexual dysfunction



Lumbar spine: Clinical syndromes

- Very frequent !!!
- **SEGMENTAL SYNDROME:** LOW BACK PAIN – very frequent
- **NON-RADICULAR IRADIATING PAIN**
  - Low back pain
  - Pain radiates into the buttocks and legs
  - No objetive symptoms of radicular lesion
- **RADICULAR SYNDROMES**
  - SCIATICA: any pain starting in the lower back and going down the leg
  - In Czech literature (not used in English)
  - LUMBOISCHIALGIC SYNDROME – L5 or S1 radiculopathy
    - Most frequent
  - LUMBOFEMORAL SYNDROME -L3 or L4.radiculopathy

Bell S.E., Beauchamp K.M. (2012) Conus Medullaris Syndrome. In: Vincent JL., Hall J.B. (eds) Encyclopedia of Intensive Care Medicine. Springer, Berlin, Heidelberg

Resources

- Harrison's Neurology in Clinical Medicine - 3th Edition. Stephen Hauser, S. Andrew Josephson.
- Lange Clinical Neurology - 10th Edition. Roger P. Simon, David Greenberg, Michael J. Aminoff.
- Adams and Victor's Principles of Neurology - 10th Edition. Allan Ropper, Martin Samuels, Joshua Klein.
- Netter's Clinical Anatomy - 4th Edition. John T. Hansen.

Thank you for your attention

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