

Diseases of the Cranial Nerves

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Diseases of the cranial nerves often lead to severe burden on patients, diverse functional disorders, and neurological deficits. Due to the diverse functions of the cranial nerves, the exact topographic localization of the clinical symptoms is very important. Diseases of the cranial nerves are a crucial part of every medical exam. In the following article, you will get an overview and a short summary of the most important diseases of all 12 cranial nerves. You can learn how to correctly examine pairs of cranial nerves here.



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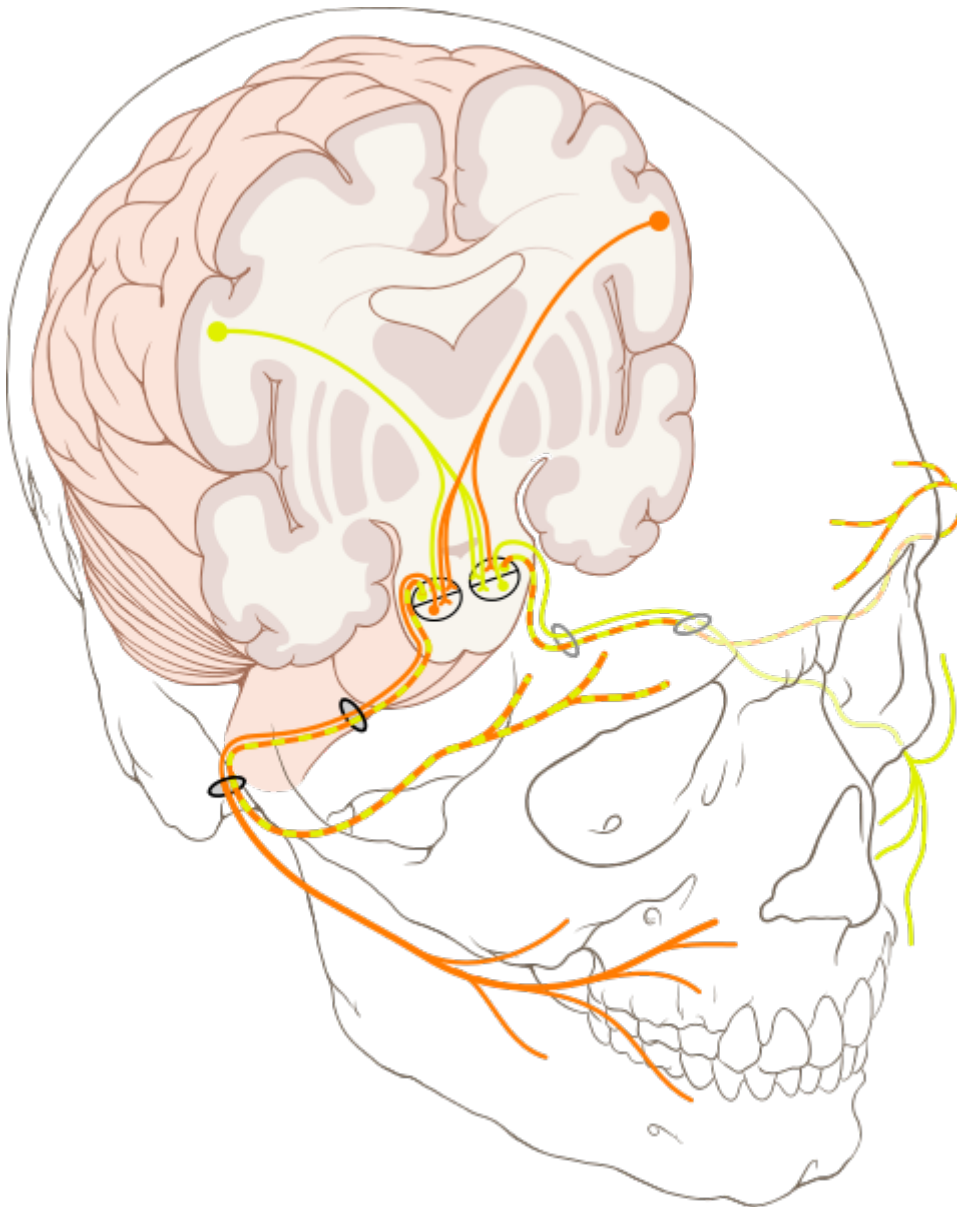


(b)

Peripheral and Central Facial Nerve Palsy

Facial nerve palsy is **paresis of the muscles supplied by the facial nerve (VII) on 1 side of the face due to a lesion of the facial nerve.** The paresis generally only occurs on 1 side, but it may also occur on both sides. Usually, it is temporary. Initially, the patient suffers from non-specific dragging pain in the region of the ear before the paresis develops over several hours or days.

This occurs in 10-35 cases/100,000 inhabitants. During pregnancy, the prevalence increases 3 times, especially in the 1st trimester. This condition has a universal distribution and has no predilection for ethnicity or age.



[Image](#): Cranial nerve VII. By Patrick J. Lynch, License: [CC BY 2.5](#)

Possible causes for facial nerve paresis:

- Viral and bacterial infections
- Stroke/ischemic lesions
- Basal skull fracture
- Tumors in the petrous bone or the parotid gland
- Intracranial injury
- Toxic causes
- Idiopathic causes
- Chromosomal damage

Depending on the location of the lesion, one distinguishes between central and peripheral facial nerve palsy.

In both, rehabilitation must be started as soon as possible to avoid complications.

Peripheral facial nerve paresis

Peripheral facial nerve paresis involves lesions of the 2nd motor neuron, the anterior motor horn, the peripheral nerves, or the muscles outside the central nervous system. Peripheral facial nerve palsy is characterized by a **weakened myotatic reflex, negative pyramidal tract signs, a slack tone, and atrophy of the affected muscles.**

Idiopathic facial nerve palsy (Bell's palsy) is the most frequent peripheral cranial nerve lesion, and it is accompanied by a single-sided and acute occurrence of peripheral facial nerve palsy. This disease can occur at any age, often between the ages of 10-20 and 30-40 years. Women seem to be affected more frequently than men.

Bell's palsy **heals in approx. 70% of cases without any consequences**, but persistent defects after re-innervation may remain.

As a consequence of 1-sided peripheral facial palsy, the following actions **are no longer possible**:

- Frowning
- Raising eyebrows
- Closing the eyes
- Puffing out the cheeks
- Whistling
- Showing the teeth

These symptoms also suggest the failure of the nerve, and weakness or complete paresis of the mimic muscles may result. **Symptoms include:**

- Bell's phenomenon (incomplete closure of the eyelid)
- Upward rotation of the eyeball becomes visible
- Drooping of the labial angle and the lower eyelid
- Elapsed nasolabial fold
- Slackened platysma

Disorders of lacrimation, headache, gustatory disturbances in the anterior 3rd of the tongue, ear pain, and increased hearing sensation are other accompanying symptoms.

Causes of Bell's palsy are:

1. Herpes zoster infections
2. Otitis media
3. Guillain-Barré syndrome
4. HIV infection tumor
5. Ischemic stroke
6. Autoimmune disease
7. Lyme disease
8. Among others.

Central facial palsy

Central facial palsy is due to a **lesion of the 1st motor neuron** in the region of the brain or its descending projections to the spinal cord.

Central facial palsy involves increased myotatic reflexes, weakened multisynaptic reflexes, and positive pyramidal tract signs; a cramp-like increase in the tone of the

affected muscles also occurs, without any relevant atrophy. **Frequently, central facial palsies are caused by cerebral circulatory impairments or brain tumors.**

In contrast to peripheral facial palsy, **a patient with central facial palsy can frown and close his eyelid** since the peripheral nuclear areas (facial nucleus) lead to the fibers of the facial nerve and are interconnected to finally reach the forehead and the eye, which also receive fibers from the other side.

The musculature is no longer mobile – especially in the area of the mouth – and it is flaccid, as with peripheral facial palsy. Also, the labial angle droops are immobile and are partially open on the affected side.



[Image](#): Man suffering from right facial palsy. By St. Bartholomew's Hospital Society, License: [CC BY 4.0](#)

Note: A distinguishing feature of peripheral and central facial palsy: with central facial palsy, the patient can frown and close his eyelid.

Trigeminal Neuralgia

Trigeminal neuralgia occurs in the innervation area of the trigeminal nerve in the form of a severe, acute, and recurrent attack-like facial pain that is generally single-sided. **Three forms** are distinguished:

- Classic trigeminal neuralgia: in patients with compression of the trigeminal nerve by a presumed or demonstrated vascular loop
- Secondary trigeminal neuralgia: associated with another disease such as multiple sclerosis and tumors
- Idiopathic trigeminal neuralgia: when the cause is unknown

Other categories include several causes of facial pain such as painful trigeminal neuropathy due to herpes zoster virus, post-traumatic trigeminal neuropathy, painful trigeminal neuropathy attributed to other disorders, and idiopathic painful trigeminal neuropathy.

Classic trigeminal neuralgia

Previously called tic douloureux. The principal cause of this pain is the compression or mechanical irritation of the trigeminal nerve by blood vessel loops at the point where it

exits the brainstem. The average age of onset is between 50–79 years.

Symptomatic trigeminal neuralgia

This form of trigeminal neuralgia accompanies demyelination diseases such as multiple sclerosis, which occurs as a consequence of either tumors or Costen's syndrome (e.g., a facial pain which originates from the facial muscles due to malfunction of the mandibular joint). Inflammatory processes and, in rare cases, medical interventions can result in trigeminal neuralgia.

Symptomatic trigeminal neuralgia may be accompanied by hypoesthesia, i.e. reduced sensitivity towards tactile stimuli, in the form of numbness or tingling, in the region of the 1st trigeminal branch. Furthermore, the corneal reflex is weakened.

Patients with symptomatic trigeminal neuralgia are, on average, younger than patients with the classical form of the disease (tic douloureux). Double-sided facial pain often also occurs in such patients. The goal of therapy is to treat the underlying cause.

Symptoms of trigeminal neuralgia:

- Attacks of shooting and severe pain, which occurs repeatedly, up to 100 times a day
- Mostly, the supply area of the 2nd branch of the trigeminal nerve is affected
- It can be triggered by touch, coldness, speaking, swallowing, chewing, combing hair, touching or washing the face, spicy food, vibration at walking, etc.
- Due to the pain, patients are sad, powerless, anorexic, they sleep badly, and they feel weak

The patients try to avoid the trigger by reducing their mimic movement, not speaking, and not eating. Often, only fluid foods are taken in with a straw. In the cold seasons, most patients protect themselves from the cold and the wind with a scarf.

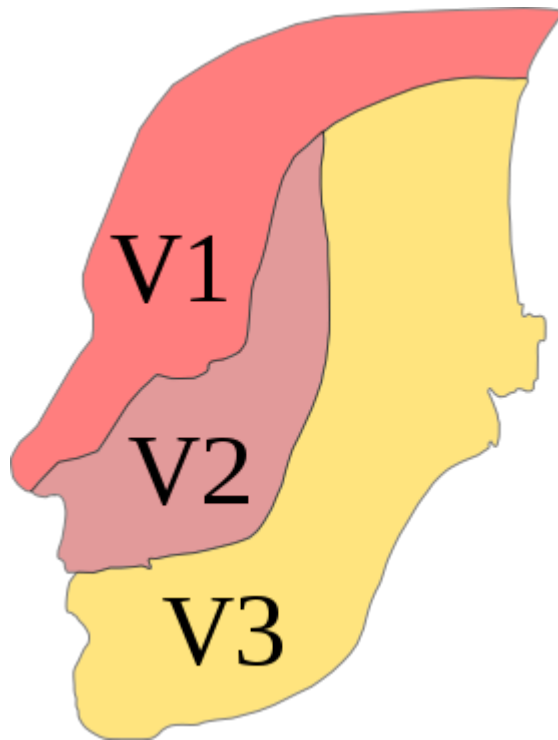


Image: Trigeminal distribution. By Madhero88, License: [CC BY 3.0](https://creativecommons.org/licenses/by/3.0/)

Eye muscle paresis

Oculomotor nerve palsy

The oculomotor nerve (the eye movement nerve) innervates several eye muscles and – along with the trochlear nerve (IV) and the abducens nerve (VI) – is responsible for the movement of the eyeball.

Roughly/3rd of all eye muscle pareses are caused by oculomotor nerve palsy, which is overall slightly rarer than abducens nerve palsy. In 60–70% of cases, oculomotor nerve palsy occurs as an isolated loss.

Lesions of this nerve can result in various types of paresis: **complete (inner and outer) oculomotor nerve palsy.**

Complete loss of the function of the nerve leads to the **following clinical picture:**

- Ptosis (drooping of the upper eyelid)
- The eyeball deviates to the outside and downwards
- Widened pupil (mydriasis) and pupils unresponsive to light (totally unresponsive pupil)
- Diplopia (double vision)

In cases of complete oculomotor nerve palsy, **the consensual reaction of the opposite eye remains**, i.e. a reflex-triggered concordant reaction occurs on the opposite side of the body. The opening of the eyelid may be possible through contraction of the frontal muscle since double vision only occurs following the elevation of the eyelid.

Causes of oculomotor nerve palsy are:

1. Tumor
2. Stroke
3. Infections of the central nervous system (meningitis or encephalitis)
4. Aneurysm
5. A local lesion in the base of the eye.

Ophthalmoplegia interna

Ophthalmoplegia interna involves **completely unresponsive pupils accompanied by the free movement of the eyeball**; the pupil does not react to either direct or indirect light nor convergence. As a result, the patient does not have a clear vision in the affected eye when looking at close objects. There is also paresis of accommodation.

Ophthalmoplegia externa

In cases of ophthalmoplegia externa, however, the **motility of the eyeball is impaired, yet autonomous innervation of the pupil and the ciliary muscle is intact**. If pupil function has been preserved, complete paresis of all the muscles innervated by the oculomotor nerve is quite rare.

Anisocoria

Another disease or deficit of the oculomotor nerve would be **pupils of unequal width**, which is referred to as anisocoria. Anisocoria is present in Claude-Bernard- Horner syndrome, which is characterized by constriction of the pupil (miosis), a drooping eyelid (ptosis), anhidrosis (decreased sweating), and posterior displacement of the eyeball (enophthalmos).

Anisocoria can also occur alongside intracranial pressure involving compression of the oculomotor nerve.



Image: Anisocoria. By Russavia, License: [CC BY 3.0](https://creativecommons.org/licenses/by/3.0/)

Trochlear Nerve Palsy

Trochlear nerve palsies are rarer than oculomotor or abducens nerve palsies. **The most frequent cause of monosymptomatic trochlear nerve palsy is a traumatic brain injury.**

Trochlear nerve palsy is characterized by **isolated paresis of the superior oblique muscle**. The function of this muscle is to depress the eyeball. In cases of paresis, the symptom increases during adduction and is virtually absent during abduction, i.e. the eye of the patient faces towards the nose and upwards, and the patient experiences double

vision (diplopia), just as in cases of oculomotor nerve palsy.

Causes of trochlear nerve palsy are tumor, demyelination, meningitis, and other.

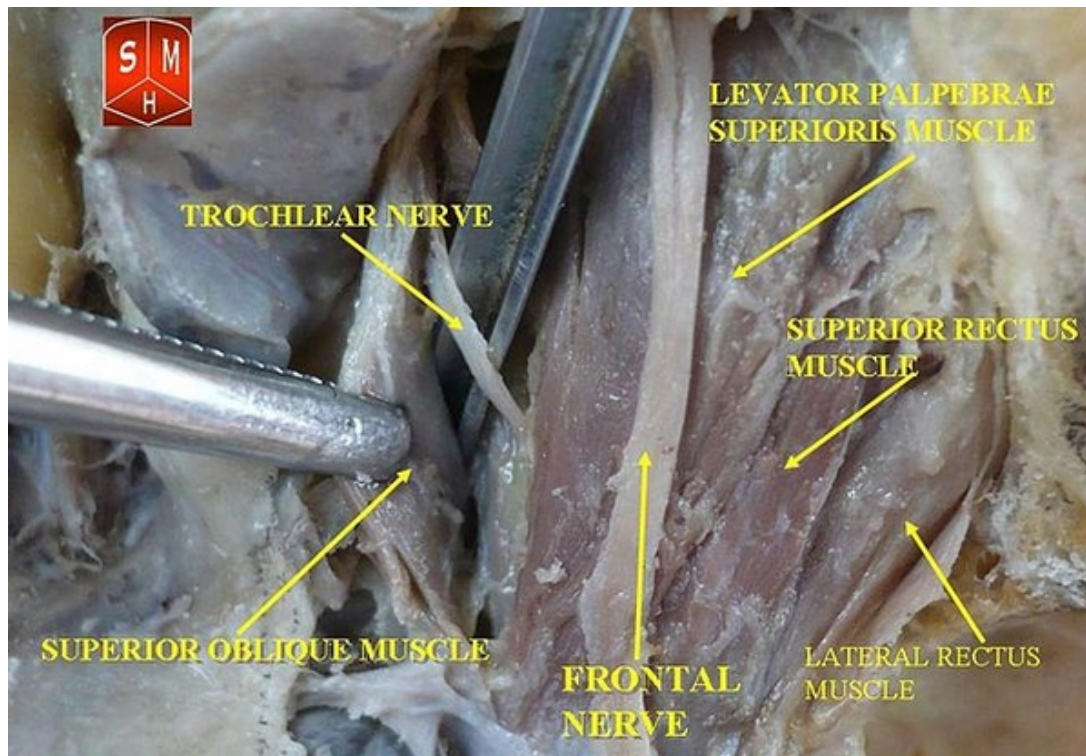


Image: Trochlear and frontal nerves. By Anatomist90, License: [CC BY-SA 3.0](https://creativecommons.org/licenses/by-sa/3.0/)

A distinction is made between double and single-sided trochlear nerve palsy.

Double-sided trochlear nerve palsy

In cases of double-sided trochlear nerve palsy, the Bielschowsky phenomenon is often positive on both sides. In this case, the diseased eye stands higher, is rotated outwards to the temple, and has a squint deviation to the nose, which creates oblique double vision. To compensate, the patient tries to rotate and lower the chin and to tilt the head to the healthy side.

Compensatory head-turning and tilting are usually not present, in contrast to 1-sided trochlear nerve palsy.

One-sided trochlear nerve palsy

One-sided trochlear nerve palsy is accompanied by a **compensatory head posture with turning and tilting towards the healthy shoulder and lowering of the chin.** The affected eye is in an abduction position and is rotated outwards. An annoying pathological rolling image is avoided since the slackened internal rotator is not utilized this way.

Abducens nerve palsy

The abducens nerve palsy is characterized by an **isolated paresis of the rectus lateralis muscle** (an externally turning muscle), which often occurs without identifiable intracranial lesions. In a high percentage of cases, the cause of these palsies remains

idiopathic. However, trauma, a diabetic metabolic state, and increased intracranial pressure due to tumor or meningitis are some causes of abducens nerve palsy.

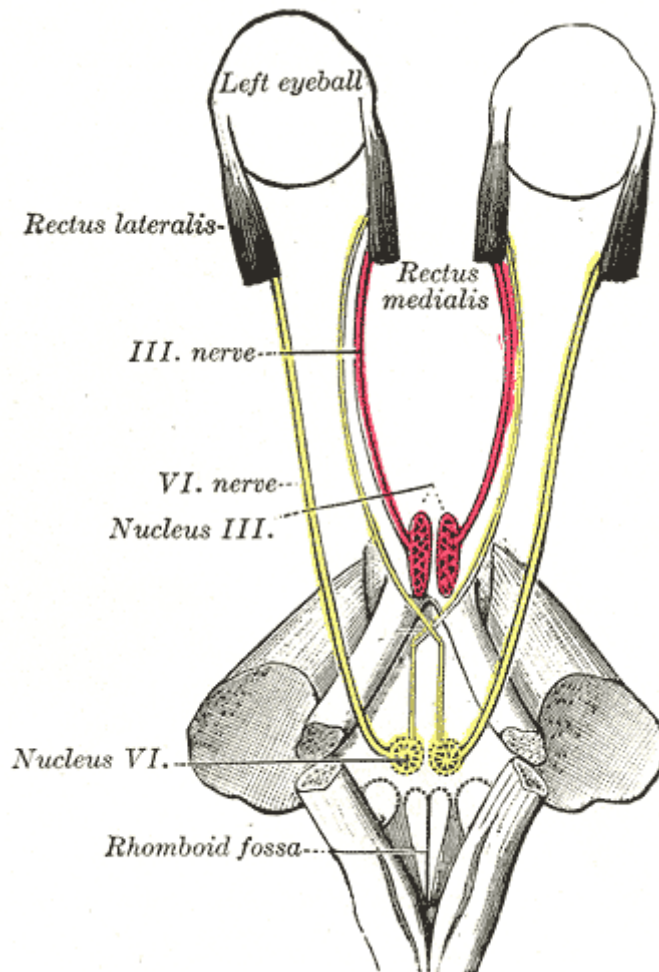


Image: Isolated paresis of the rectur lateralis muscle. By Henry Gray, License: Public domain

In cases of abducens nerve palsy, **convergent paralytic strabismus occurs even in the primary position**, i.e. the affected eye deviates towards the inside and the paralyzed eye is impaired or inhibited if it tries to turn to look to the side or to look up.

Furthermore, a slight adduction position can occur when looking up or down. Undisturbed binocular movement is, however, observed when looking to the healthy side.

Horizontally parallel double images (i.e. double vision) are usually perceived even in the primary position. The deviation of the images of objects on the retina increases on the paralyzed side, but when the affected eye is covered, the image corresponding to the respective side disappears.

However, the annoying phenomenon of double vision causes a compensatory head posture, which leads to the head being turned towards the side of the paralyzed muscle – a position that does not require the rectus lateralis muscle.

Lesions of the visual pathway

Each optic tract consists of '2 half, former' optical nerves. They conduct the lateral part of the visual information coming from the same side and also the medial sensations of the

opposite side.

The visual pathway crosses the whole brain. **Very differing deficits in the visual field may arise depending on the location of the lesion.** Therefore, when there is a lesion in the visual pathway, the visual field is examined, and pupil reaction and the appearance of the papilla are examined.

The following deficits in the visual field can occur as a result of nerve lesions:

- If the optic nerve is severely damaged on 1 side, the patient **is blind on the affected eye**, and their sight is not impaired on the other side.
- When there is a lesion of the medial part of the optic chiasm, it is mainly the fibers that cross to the other side that are damaged. The patient can suffer from a bitemporal (heteronymous) hemianopsia, i.e the patient does not get any visual information relating to what happens in his lateral field of vision. This is referred to as hemianopsia, or 'blinker vision.' The lateral section, which does not cross and leads to the optic tract, remains intact, meaning sight in this part of the visual field is not impaired.
- Visual loss can also affect the optic tract. For example, the patient may have a lesion in the right optic tract which **leads to a deficit in the left half of the visual field of both eyes**. This failure is referred to as bilateral homonymous hemianopsia (left). A lesion of the left optic tract would lead to a corresponding opposite deficit in the right half of the visual field. Depending on where the optic tract is damaged, this may lead to a complete or an incomplete deficit. Once such deficits have arisen, they do not usually disappear.

Further lesions can arise in the realm of visual radiation, and such lesions have diverse consequences since visual radiation spreads a fan-like manner. Tumors and strokes are the most frequent triggers for this visual disorder.

Deficits in the visual field can be observed in the following form:

- First: Pathology to the right part of the visual radiation produces left homonymous hemianopsia
- Second: Pathology to the leftward side of the visual radiation produces right homonymous hemianopsia



Image: Homonyme hemianopsia. By Nunh-huh, License: [CC BY-SA 3.0](https://creativecommons.org/licenses/by-sa/3.0/)

Quadrantanopsia occurs whenever there is only partial damage to the visual radiation such that a quadrant of the visual field is missing rather than an entire half.

Deficits in the upper part of the visual radiation are more severe than in the lower part. Deficits in the area of the visual radiation likewise do not regress either.

Amaurosis

This term refers to a complete loss of vision without apparent lesions in the eye; it can occur in 1 or 2 eyes. Amaurosis can be congenital (Leber's congenital amaurosis) or secondary. What this means for the optic nerve is that injury is commonly secondary to compression of the nerve by tumor (commonly from the pituitary), trauma, or ischemic events.

Summary of the Important Diseases of All 12 Pairs of Cranial Nerves

Olfactory nerve (I)

- Anosmia (inability to smell)
- Hyposmia (weakened ability to smell)

Optical nerve (II)

- Anopsia or amaurosis (blindness in 1 or both eyes)
- Hemianopsia
- Quadrant anopsia
- Blinker-phenomenon
- Papilledema

Oculomotor nerve (III)

- Anisocoria (unequally wide pupils)
- Miosis (narrow pupils)
- Mydriasis (wide pupils)
- Gaze palsy
- Diplopia (double vision)
- Ptosis (drooping upper eyelid)

Trochlear nerve (IV)

- Strabismus
- Diplopia

Trigeminal nerve (V)

- Trigeminal neuralgia/tic douloureux
- Paresis of the muscles of mastication
- Loss of the sensation of touch and temperature

Abducens nerve (VI)

- Diplopia

Facial nerve (VII)

- Bell's palsy (paralysis of the the facial muscles)

- Hyperacusis (sounds are perceived too loud)
- Loss of gustatory sensation in the anterior tongue
- Burning eye sensation due to dehydration of the conjunctiva/cornea

Vestibulocochlear nerve (VIII)

- Hypacusis (hearing loss)
- Deafness
- Tinnitus (permanent aural noises)
- Ataxia (instability regarding movement)
- Rotatory vertigo
- Nystagmus (eye twitching)

Glossopharyngeal nerve (IX)

- Difficulty swallowing
- Diminished salivation
- Loss of gustatory sensation in the posterior part of the tongue
- Loss of sensation in the throat

Vagus nerve (X)

- Hoarseness
- Difficulties with swallowing and at phonation
- Posticus paralysis (severe respiratory distress when a particular muscle of the larynx fails)
- Changes in heart rate (quicker or slower)
- Less gastric acid and intestinal peristalsis

Accessory nerve (XI)

- Inability to lift the shoulder
- Weakness in turning the head

Hypoglossal nerve (XII)

- Speech disorders
- Difficulty swallowing

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