

Cranial nerve lesions

See the separate [Examination of the Cranial Nerves](#) article for a detailed reminder on how to examine the nerves.

Where is the lesion?

Try to think systematically – is it:

- Muscle (eg, a dystrophy)?
- Neuromuscular junction (eg, myasthenia)?
- Cranial nerve lesion outside the brainstem (eg, compression)?
- Cranial nerve lesion within the brainstem (eg, multiple sclerosis (MS))?

Cranial nerves may be affected singly or in groups and knowledge of which nerves are involved helps locate the lesion. Some of the causes of cranial nerve lesions are given below, after a reminder of the anatomical course of the nerve.

Conditions which can affect any cranial nerve

- Diabetes mellitus.
- MS.
- Tumours.
- Sarcoid.
- Vasculitis (eg, polyarteritis nodosa).
- Systemic lupus erythematosus (SLE).
- Syphilis.

Chronic meningitis (malignant, TB, or fungal) tends to pick off the lower cranial nerves one by one.

Olfactory (I) nerve

- **Anatomy.** Olfactory cells are a series of bipolar neurones which pass through the cribriform plate to the olfactory bulb.
- **Signs.** Reduced taste and smell, but not to ammonia which stimulates the pain fibres carried in the trigeminal nerve.
- **Causes.** Trauma, frontal lobe tumour, meningitis.

Optic (II) nerve

- **Anatomy.** The optic nerve fibres are the axons of the retinal ganglion cells. At the optic chiasm, only the fibres derived from the nasal parts of the retina decussate, join with the non-decussating fibres and pass backwards in their respective optic tracts to the visual cortex.

- **Signs and causes:**

- Visual field defects – see the separate [Visual Field Defects](#) article:
 - Field defects start as small areas of visual loss (scotomas).
 - Monocular severe sight impairment: lesions of one eye or optic nerve – eg, MS, giant cell arteritis.
 - Bilateral severe sight impairment: methyl alcohol, tobacco amblyopia; neurosyphilis.
 - Bitemporal hemianopia: optic chiasm compression – eg, internal carotid artery aneurysm, pituitary adenoma or craniopharyngioma.
 - Homonymous hemianopia: loss of the same half (left or right) of the visual field of both eyes, on the opposite side to the lesion (eg, a right side lesion causes loss of the left side of the visual field of both eyes). Lesions lie behind the optic chiasm in the optic tract, lateral geniculate nucleus, optic radiations or the occipital cortex – eg, stroke, abscess, tumour.
- Pupillary abnormalities – see the separate article on [Pupillary Abnormalities](#):
 - Optic neuritis (pain on moving the eye, loss of central vision, afferent pupillary defect, papilloedema). Causes: demyelination; rarely, sinusitis, syphilis, collagen vascular disorders.
 - Optic atrophy (pale optic discs and reduced acuity): MS, frontal tumours, Friedreich's ataxia, retinitis pigmentosa, syphilis, glaucoma, Leber's optic atrophy, optic nerve compression.

- Papilloedema (swollen discs):
 - Raised intracranial pressure (ICP) (tumour, abscess, encephalitis, hydrocephalus, benign intracranial hypertension).
 - Retro-orbital lesion (eg, cavernous sinus thrombosis).
 - Inflammation (eg, optic neuritis).
 - Ischaemia (eg, accelerated hypertension).

Oculomotor (III) nerve

- **Anatomy.** This nerve emerges from the brainstem on the medial aspect of the crus cerebri and then passes forwards between the posterior cerebral and superior cerebellar arteries, very close to the posterior communicating artery. It pierces the dura near the edge of the tentorium cerebelli, and passes through the lateral part of the cavernous sinus with the IV and VI nerves to enter the orbit.
- **Signs.** The initial sign is often a fixed dilated pupil which doesn't accommodate; then **ptosis** develops and then a complete internal ophthalmoplegia (masked by ptosis). Unopposed lateral rectus causes outward deviation of the eye. If the ocular sympathetic fibres are also affected behind the orbit, the pupil will be fixed but not dilated.
- **Causes of a single III lesion.** Diabetes mellitus, giant cell arteritis, syphilis, posterior communicating artery aneurysm, idiopathic; raised ICP if causes uncal herniation through the tentorium - this compresses the nerve. Third nerve palsies without a dilated pupil are due to diabetes mellitus or another vascular cause. Early dilatation of a pupil implies a compressive lesion. **Diplopia** from a third nerve lesion may cause nystagmus.

See also 'Combined cranial nerve lesions', below.

Trochlear (IV) nerve

- **Anatomy.** Passes backwards in the brainstem, decussates in the anterior medullary velum and emerges to pass round the cerebral peduncle between it and the temporal lobe, passing over the tentorium to enter the cavernous sinus with II and VI, and enters the orbit to supply the superior oblique muscle.
- **Signs.** Diplopia due to weakness of downward and inward eye movement. The most common cause of a pure vertical diplopia. The patient tends to compensate by tilting the head away from the affected side.
- **Causes of a single IV lesion.** Rare and most commonly due to trauma to the orbit. It may also occur in diabetes or infarction secondary to hypertension.

See also 'Combined cranial nerve lesions', below.

Trigeminal (V) nerve

- **Anatomy.** Forms three trunks: ophthalmic, maxillary and mandibular divisions. The latter contains both sensory and motor fibres. There may be considerable individual variation in the exact areas of skin supplied:
 - Ophthalmic division lies with III, IV and VI in the cavernous sinus and supplies the skin over the medial nose, forehead, and eye (including corneal reflex).
 - Maxillary division passes through the inferior part of the cavernous sinus and the foramen rotundum and joins with parasympathetic fibres to form the sphenopalatine ganglion (lacrimation). It then enters the orbit as the infraorbital nerve, eventually supplying the skin of the upper lip, cheek and triangle of skin extending from the angle of eye and mouth to an apex in the mid-temporal region.
 - Mandibular division leaves the skull through the foramen ovale carrying sensory fibres from the skin of the lower lip and chin up to and including the tragus and upper part of the pinna; mucous membranes of the floor of the mouth, cheek and anterior two thirds of the tongue (taste fibres joining it from the chorda tympani branch of the facial nerve). Motor fibres supply the masseter, temporalis, and pterigoids.
- **Signs.** Reduced sensation or dysaesthesia over the affected area. Weakness of jaw clenching and side-to-side movement. If there is a lower motor neurone (LMN) lesion, the jaw deviates to the weak side when the mouth is opened. There may be fasciculation of temporalis and masseter.
- **Causes of a single V lesion.** See also 'Combined cranial nerve lesions', below:
 - Sensory: [trigeminal neuralgia](#), [herpes zoster](#), [nasopharyngeal carcinoma](#).
 - Motor: [bulbar palsy](#), [acoustic neuroma](#).

Abducent (VI) nerve

- **Anatomy.** From the nucleus in the floor of the fourth ventricle, fibres pass forward in the pons and emerge to follow a long extracerebral course on the base of the brain, across the apex of the petrous temporal, through the posterior fossa near the dorsum sellae to enter the cavernous sinus and thence to the orbit and lateral rectus muscle.
- **Signs.** Inability to look laterally. The eye is deviated medially because of unopposed action of the medial rectus muscle ^[1].

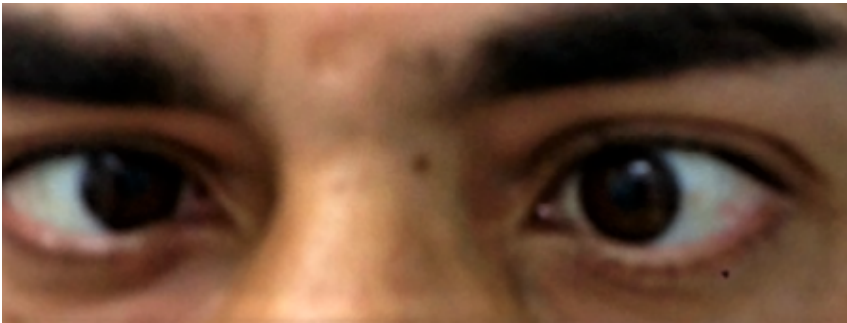


Image source: Open-i (Fadda GL et al) - see Further reading below

- **Causes of a single VI lesion.** MS, pontine CVA. It is considered a false localising sign (because of long extracerebral course) in raised ICP.

See also 'Combined cranial nerve lesions', below.

Facial (VII) nerve

- **Anatomy.** Mainly motor (some sensory fibres from external acoustic meatus, fibres controlling salivation and taste fibres from the anterior tongue). Fibres loop around the VI nucleus before leaving the pons medial to VIII and passing through the internal acoustic meatus. It passes through the petrous temporal in the facial canal, widens to form the geniculate ganglion (taste and salivation) on the medial side of the middle ear, whence it turns sharply (and the chorda tympani leaves), to emerge through the stylomastoid foramen to supply the muscles of facial expression.

- **Signs.** Facial weakness. In an LMN lesion the forehead is paralysed – the final common pathway to the muscles is destroyed; whereas the upper facial muscles are partially spared in an upper motor neurone (UMN) lesion because of alternative pathways in the brainstem. There appear to be different pathways for voluntary and emotional movement. CVAs usually weaken voluntary movement, often sparing involuntary movements (eg, spontaneous smiling). The much rarer selective loss of emotional movement is called mimic paralysis and is usually due to a frontal or thalamic lesion.
- **Causes of a single VII lesion:**
 - LMN: [Bell's palsy](#), polio, otitis media, skull fracture, cerebellopontine angle tumours, [parotid tumours](#), herpes zoster ([Ramsay Hunt syndrome](#)), [Lyme disease](#).
 - UMN: (spares the forehead – bilateral innervation): stroke, tumour.

See also 'Combined cranial nerve lesions', below.

Vestibulocochlear (VIII) nerve

- **Anatomy.** Carries two groups of fibres, those to the cochlea (hearing) and to the semicircular canals, utricle and saccule (balance and posture). They pass, together with the facial nerve, from the brainstem across the posterior fossa to the internal acoustic meatus.
- **Signs.** Unilateral sensorineural deafness, [tinnitus](#). Slow-growing lesions seldom present with vestibular symptoms as compensation has time to occur.
- **Causes of a single VIII lesion.** Loud noise; [Paget's disease of bone](#), [Ménière's disease](#), [herpes zoster](#); neurofibroma, [acoustic neuroma](#), brainstem CVA, lead, aminoglycosides, furosemide, aspirin. See also 'Combined cranial nerve lesions', below.

Glossopharyngeal (IX) nerve

- **Anatomy.** Contains sensory, motor (stylopharyngeus only) and parasympathetic fibres (salivary glands). Passes across the posterior fossa, through the jugular foramen and into the neck, supplying tonsil, palate and posterior third of tongue.
- **Signs.** Unilateral lesions do not cause any deficit because of bilateral corticobulbar connections. Bilateral lesions result in pseudobulbar palsy. These nerves are closely interlinked.
- **Causes (single nerve lesions exceedingly rare).** Trauma, brainstem lesions, cerebellopontine angle and neck tumours, polio, [Gullain-Barré syndrome \(GBS\)](#).

Vagus (X) nerve

- **Anatomy.** The vagus nerve, 'the wanderer', contains motor fibres (to the palate and vocal cords), sensory components (posterior and floor of external acoustic meatus) and visceral afferent and efferent fibres. It leaves the skull through the jugular foramen, passes within the carotid sheath in the neck (giving off cardiac branches, and the recurrent laryngeal nerves supplying the vocal cords), through the thorax supplying the lungs, and continues on via the oesophageal opening to supply the abdominal organs.
- **Signs.** Palatal weakness can cause 'nasal speech' and nasal regurgitation of food. The palate moves asymmetrically when the patient says 'ahh'. Recurrent nerve palsy results in hoarseness, loss of volume and 'bovine cough'.
- **Causes (single nerve lesions exceedingly rare).** Trauma, brainstem lesions, tumours in the cerebellopontine angle, jugular foramen and neck; polio, GBS.

Spinal accessory (XI) nerve

- **Anatomy:** motor to sternocleidomastoid and trapezius.
- **Signs:** weakness and wasting of these muscles.
- **Causes:** as 'Vagus (X) nerve', above.

Hypoglossal (XII) nerve

- **Anatomy.** It passes briefly across the posterior fossa, leaves the skull through the hypoglossal canal and supplies motor fibres to the tongue and most of the infrahyoid muscles.
- **Signs.** An LMN lesion produces wasting of the ipsilateral side of the tongue, with fasciculation; and on attempted protrusion the tongue deviates towards the affected side, but the tongue deviates away from the side of a central lesion.
- **Causes of a single XII lesion:** rare. Polio, [syringomyelia](#) tuberculosis, median branch thrombosis of the vertebral artery.

Combined cranial nerve lesions

- **VII, VIII, then V and sometimes IX:** cerebellopontine angle tumours.
- **V, VI (Gradenigo's syndrome):** lesions within the petrous temporal bone.
- **Combined III, IV, VI:** stroke, tumours, Wernicke's encephalopathy, aneurysms, MS, myasthenia gravis, meningitis, muscular dystrophy, myotonic dystrophy, cavernous sinus thrombosis, GBS, cranial arteritis, trauma and orbital pathology.

Further reading

- [Finsterer J, Grisold W](#); Disorders of the lower cranial nerves. J Neurosci Rural Pract. 2015 Jul-Sep;6(3):377-91. doi: 10.4103/0976-3147.158768.
- [Gutierrez S, Warner T, McCormack E, et al](#); Lower cranial nerve syndromes: a review. Neurosurg Rev. 2020 Jul 8. pii: 10.1007/s10143-020-01344-w. doi: 10.1007/s10143-020-01344-w.

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