MEDICAL UNIVERSITY – PLEVEN

Neurology Clinic

CLASS 4: Syndromes of cranial nerve lesions (I-VI-th)

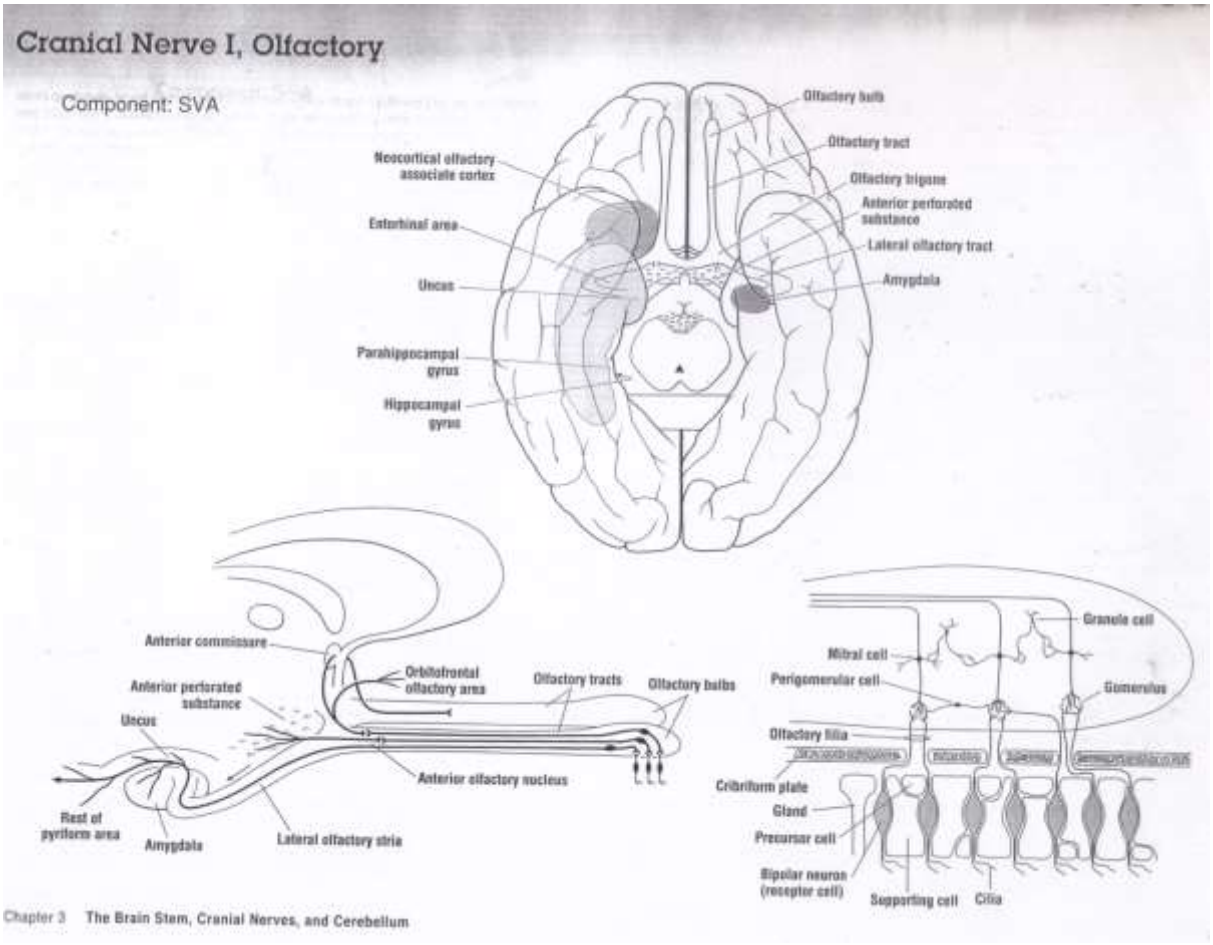
CN I Olfactory nerve

Anatomy. The olfactory mucosa occupies a small area on the roof of the superior nasal concha. Olfactory cells are bipolar sensory cells. Olfactory receptors located on the cilia are composed of specific receptor proteins that bind particular odorant molecules. The unmyelinated axons of all olfactory cells converge in bundles of up to 20 fila olfactoria on each side of the nose, pass through the cribriform plate to the olfactory bulb on the floor of the anterior cranial fossa. After a synapse in the olfactory bulb, olfactory fibers travel through the lateral olfactory striae to the amygdala and areas of the temporal lobe or by way of the medial olfactory striae to the subcallosal area and the limbic system.

Quantitative olfactory disturbances (anosmia, hyposmia, hyperosmia). Anosmia (a loss of the sense of smell) can be caused by disorders of the nose - rhinitis sicca, viral infections (influenza), heavy smoking. It can be a clinical sign of a traumatic brain injury. Unilateral anosmia can be a characteristic symptom of an olfactory groove meningioma. Rarer causes of hyposmia include Paget disease, Parkinson disease, diabetes mellitus, a relative loss occurs with ageing.

Qualitative olfactory disturbances (parosmia). The perception of smell can be changed because of autonomic (hunger, stress) and hormonal changes (pregnancy) or disturbances such as ozena, depression, traumatic lesions or nasopharyngeal empyema. Olfactory hallucinations can be caused by mediobasal and temporal tumors (focal epilepsy), drug or alcohol withdrawal, and psychiatric illnesses such as schizophrenia or depression.

Tests of smell. Examination is usually performed to investigate a specific complaint rather than as a screening test. Each nostril is tested separately. Agents like peppermint, camphor and rosewater are used for identifying smells. A bottle with a test substance is held in front of one nostril. The patient is asked to inhale and report any odor perceived.

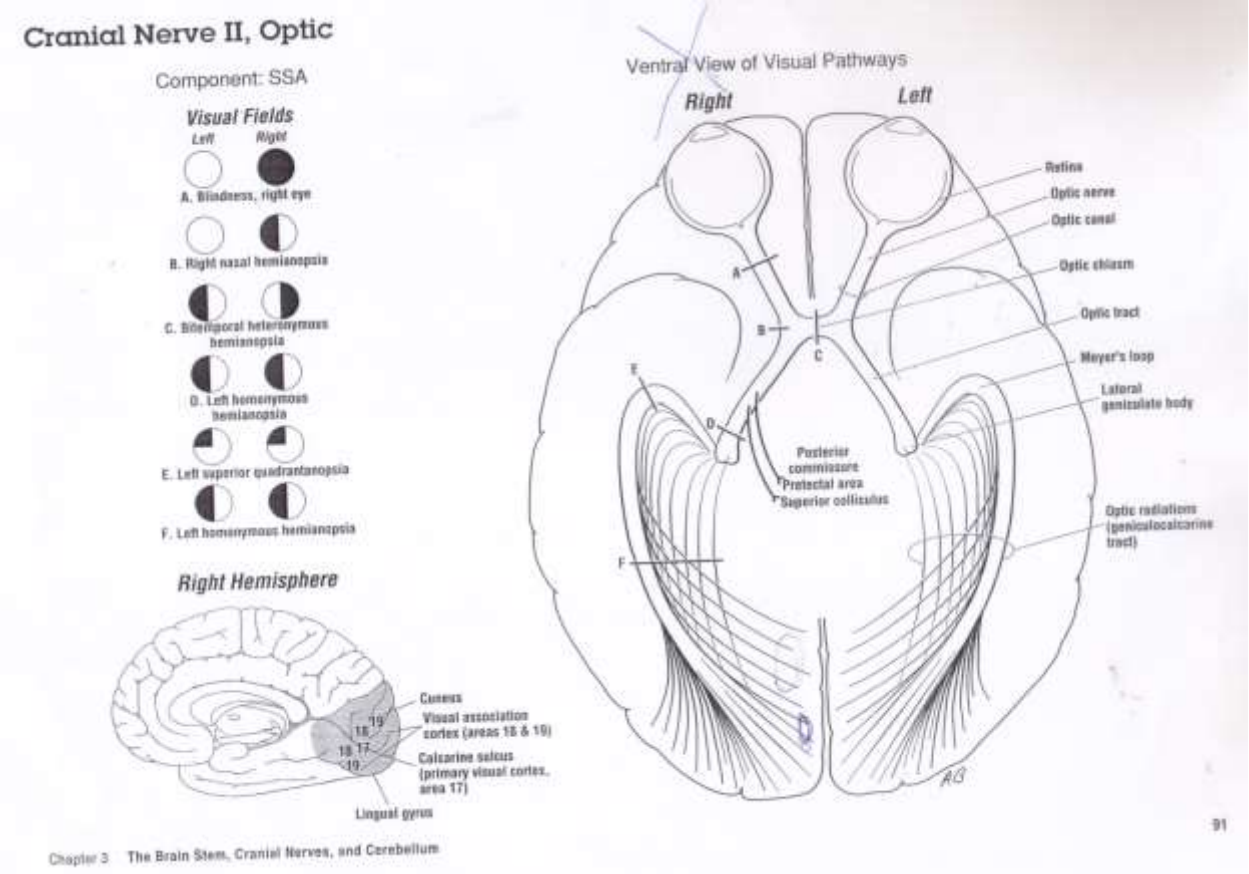


CN II Optic nerve

A Visual Field Defect - a term used for the absence of some part of the normal visual field.

Visual field defects	Clinical feature
- Monocular	- caused by unilateral retinal lesions or by partial lesions of the optic nerve
- Binocular	- caused by unilateral lesions of the visual pathway from the optic chiasm onward
Hemianopsia	- a defect occupies one half of the visual field (right or left)
Homonymous hemianopsia	- an example for a homonymous visual field defect - caused by a lesion of the optic tract, lateral geniculate body, optic radiation or visual cortex on the contralateral side
Bitemporal hemianopsia or bitemporal quadrantanopsia	- an example for a heteronymous visual field defect; - caused by a lesion of the optic chiasm; - lesions affect the decussating fibers derived from the nasal half of each retina; - a tumor compresses the optic chiasm from below (e.g., a pituitary adenoma), there is initially an upper bitemporal quadrantanopsia and later followed by bitemporal hemianopsia - a tumor compresses the optic chiasm from above (e.g., a craniopharyngioma), there is initially a lower bitemporal quadrantanopsia and later followed by bitemporal hemianopsia.
Quadrantanopsia	- a defect occupies one quarter of the visual field

Homonymous quadrantanopsia or homonymous scotoma	– caused by a lesion along the course of the optic radiation or in the visual cortex and it may affect only part of the radiating fibers or cortex – a homonymous defect that is less than a complete hemianopsia, depending on the site and extent of the lesion
Scotoma	– a defect occupies a small spot within the visual field – central scotoma is due to a lesion affecting the macula lutea and results in an impairment of central vision and a reduction of visual acuity



Loss of vision	Causes
Sudden unilateral loss of vision	– is due to a lesion of the optic nerve; can be caused by ischemia – a permanent defect, e.g., in occlusion of the central retinal a. due to temporal arteritis or embolization from an atheromatous plaque in the carotid a. – a temporary defect , called amaurosis fugax (transient monocular blindness). – a transient visual loss can be produced by a disturbance of neural function, such as migraine (retinal migraine)
Sudden bilateral loss of vision	– may be due to bilateral retinal ischemia, e.g., on standing up in a patient with aortic arch syndrome – types of intoxication can rapidly produce bilateral optic nerve lesions, e.g.,

	methanol poisoning – is due to simultaneous ischemia of both occipital lobes; often preceded by hemianopic episodes and loss of color vision as prodromal manifestations; caused by embolization into the territory of the posterior cerebral aa. on both sides simultaneously and compressive occlusion of the posterior cerebral aa. by an intracranial mass, patients deny that they cannot see (anosognosia)
Progressive unilateral impairment of visual acuity	– Retrobulbar neuritis (inflammation of the optic n. between the retina and the chiasm) and optic papillitis (inflammation of the optic n. at the level of the optic disc) cause unilateral visual loss within two days or a little longer – progressive, unilateral visual loss should be a sign for a mass: optic glioma
Progressive bilateral impairment of visual acuity	– Leber hereditary optic atrophy and tobaccoalcohol amblyopia – Vitamin B12 deficiency can cause progressive optic atrophy in combination with polyneuropathy
Optic nerve atrophy	– is a permanent residual finding after lesions of the optic nerve; the optic disc is pale – typically seen after retrobulbar neuritis or after optic nerve compression

Examination. The confrontation test, in which the examiner "confronts" the patient's visual field with his or her own. The patient and the examiner must first fixate along the same line. The examiner slowly moves a white or red object from the periphery of the visual field toward the center and determines where the patient can and cannot see it. The examiner may raise one or more fingers and ask the patient to count them (a test for small children).

CN: III, IV, IV CN

1. **Anatomy and physiology review**
   - **control eye movements (fig.1)**
   - **IV and VI are purely**
   - **III is motor with parasympathetic component of CN III mediating the efferent limb of the pupilary reflex via the ciliary ganglion (fig.2)**
   - **III innervates all eye muscles (inferior oblique, inferior rectus, superior rectus, medial rectus, and levator palpabrae superioris) except superior oblique (IV) and lateral rectus (VI)**
   - **pass through the cavernous sinus and the superior orbital fissure and can be involved in a lesion in either**

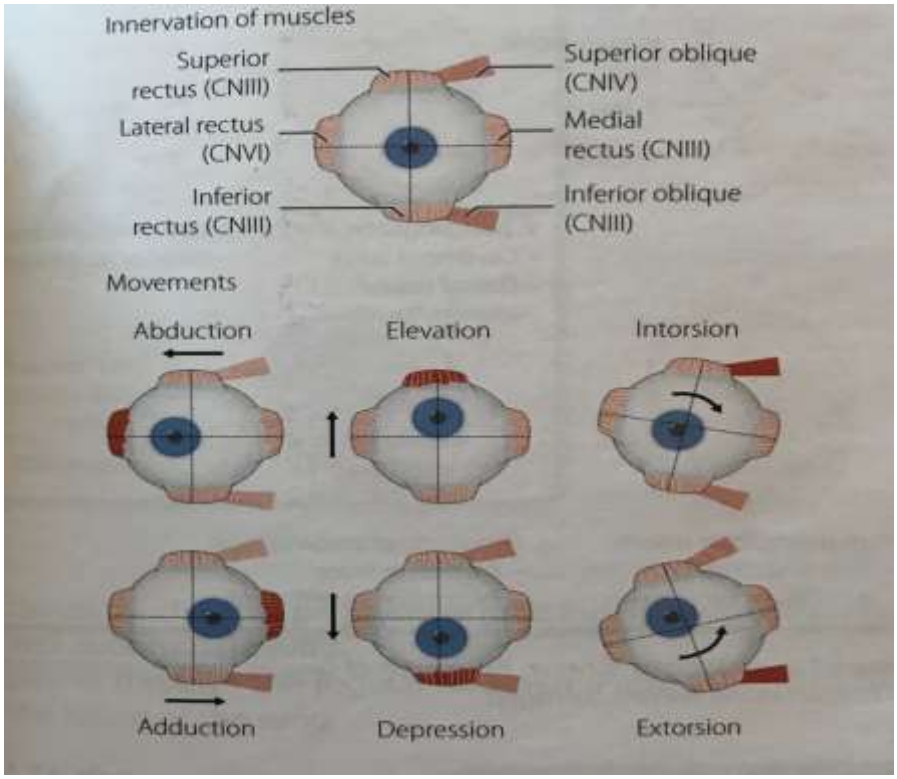


Fig. 1

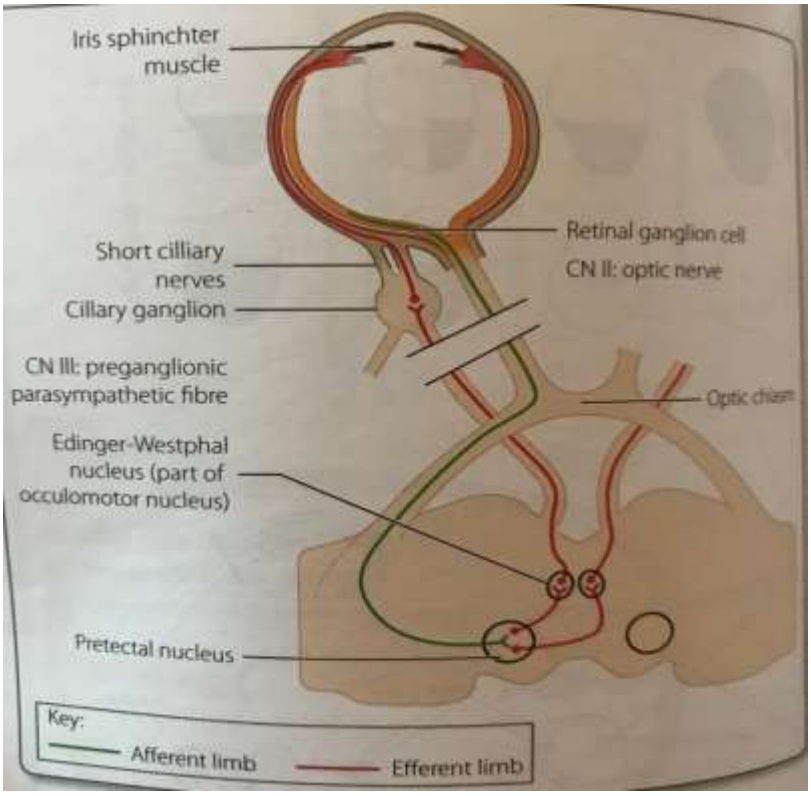


Fig. 2

2. Objective

The objective is to assess the integrity of control of eye movements. This is mainly focused on testing the motor functions of CN III, IV and VI, including the whole neuroaxis from muscle to cortical centres.

Tabl.1 The muscles and nerves of eye movements

Muscle	**Nerve**	**Primary action**
Superior rectus	III	Elevation in abduction
Inferior rectus	III	Depression in abduction
Medial rectus	III	Adduction
Inferior oblique	III	Elevation in adduction
Lateral rectus	VI	Abduction
Superior oblique	IV	Depression in adduction

3. Approach

There are three main types of eye movements: pursuit, saccadic and vestibulo-ocular:

- the occipital lobes direct control of pursuit (the slow eye movements used to track objects)
- the frontal lobes direct saccadic eye movements (the rapid movements from points of fixation)
- the cerebellar vestibular nuclei control the vestibule- ocular movements (which maintain fixation during head movements)

Ensure that you are directly in front of the patient at eye level and that you give clear instructions and ask them to report the development of any double vision.

4. Equipment

This part of the examination requires hat pin and circular card to cover each eye in turn.

5. Sequence

- Check the position of the patient’s head; straighten their head if needed.
- Check the resting position of the eyes.

- Note any deviation, resting eye movements or ptosis. Note whether they persist or worsen in each direction of gaze.
- Perform the cover test to detect latent strabismus. Cover and then quickly uncover each eye in turn, having asked the patient to fixate on your nose. If either eye has to move to fixate as it is uncovered, there is a latent strabismus (divergent if it has to adduct; convergent if it has to abduct).
- Examine visual pursuit. Remind the patient to report any diplopia.
- Note any nistagmus.
- Note whether there is full range of movement in each direction for each eye.
- Assess saccadic eye movements. Ask the patient to keep their head still and look up, down, left then right.

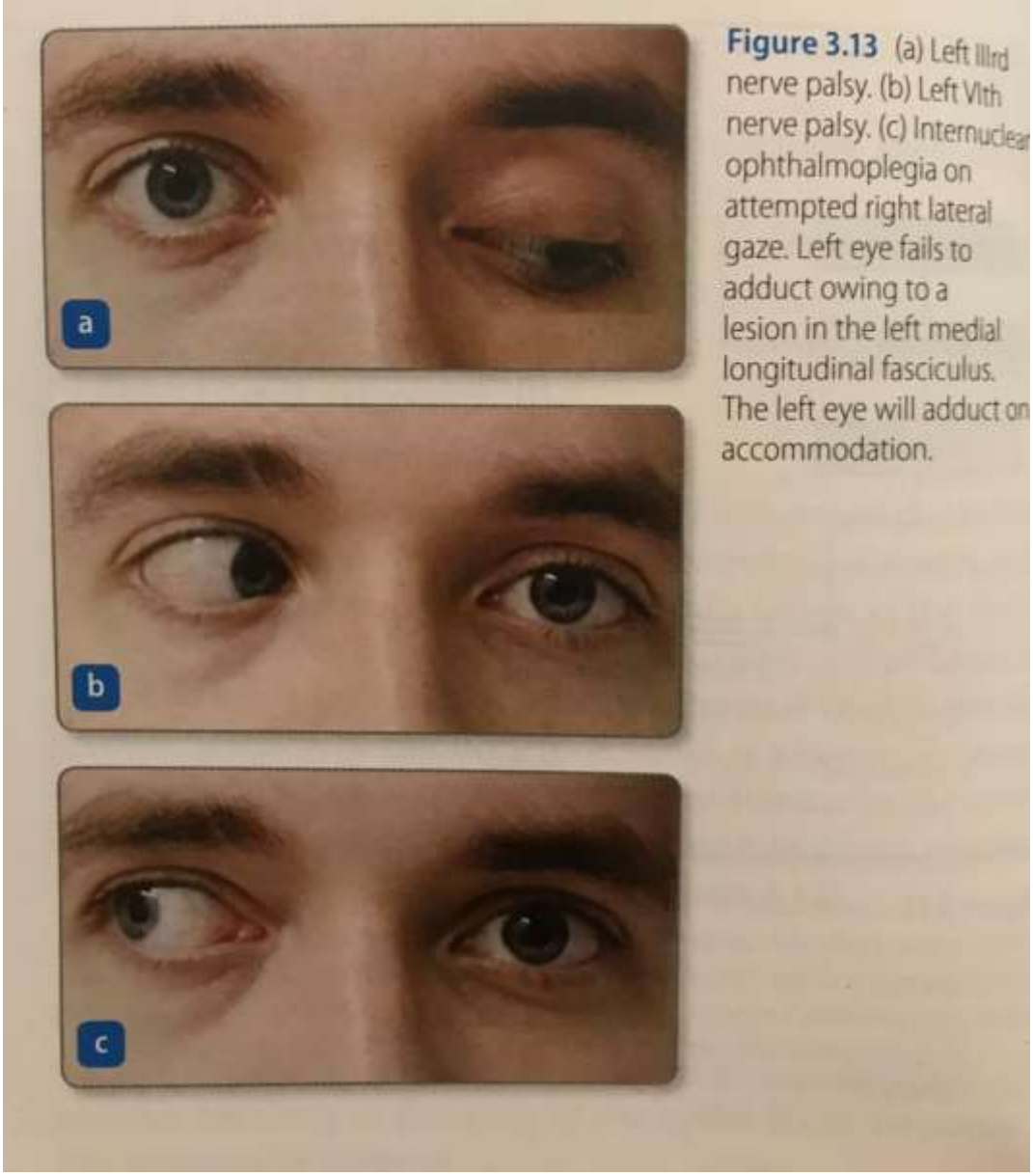


Figure 3.13 (a) Left IIIrd nerve palsy. (b) Left VIth nerve palsy. (c) Internuclear ophthalmoplegia on attempted right lateral gaze. Left eye fails to adduct owing to a lesion in the left medial longitudinal fasciculus. The left eye will adduct on accommodation.

CN V Trigeminal nerve

- consists of ophthalmic, maxillary and mandibular nerves

- is mixed motor, sensory and autonomic

– the ophthalmic nerve passes through the cavernous sinus and carries sensation from the forehead and upper eyelid and nose

– the maxillar nerve passes through the foramen rotundum and carries sensation from around the cheekbones and from the sinus and nose

– the mandibular branch passes through the infratemporal fossa and carries sensation from the lower jaw and innervates the muscles of mastication.

Examination of the sensory function of CN V

– Use a pin to assess pain perception. Test light touch and pinprick in each division on both sides

– Touch the tip of the forehead, the cheekbones and the chin on each side the patient has to describe if the feeling is sharp and to compare it on both sides.

Examination of the motor function of CN V

- Look at the side of the face, compare both sides for wasting of the temporalis muscle

- Ask the patients to clench their teeth and feel the jawfor muscle bulk

- Ask the patients to keep their mouth open while you attempt to shut it

- Test the jaw jerk. Ask the patients to open their mouth and place the tip of your finger on their chin. Then tap it softly with a tendon hammer to elicit any jaw jerk.

Patology. THE CORNEAL REFLEX (AFFERENT— OPHTHALMIC BRANCH OF V; EFFERENT—VII) - Absent corneal reflex – a failure of the blink reflex on stimulation of the cornea. If the reflex arc is intact the stimulation of the cornea will cause bilateral blinking. A lesion of CN V will cause loss of the afferent part of the reflex arc with the loss of blinking on the affected and on the contralateral sides.

Trigeminal neuralgia – a brief stabbing, shooting, electric shock-like pain in the distribution of the trigeminal nerve. It could be provoked by touching the face, chewing, drinkining. It can be idiopathic or secondary to an ectatic superior cerebellary artery irritating the trigeminal nerve rooic

CN V motor lesions cause: Wasting or weakness: muscle disease such as myotonic dystrophy, some muscular dystrophies, motor neuron disease. Positive jaw jerks: indicates an UMN lesion.

Cranial Nerve V, Trigeminal

Components: GSA, SVE