

CLINICAL SKILLS LAB 5

SHEET



SUBJECT : Physical examination-Cranial nerves

Csl_5 sheet

It's very long, why? Because I tried to include most of what's in Macleod in shorter words, and I didn't know what to mention and what not. The doctor wasn't cooperative in this manner...forgive me

Due its long ,I couldn't check the sheet more than once, therefore I apologize for any grammar or spelling mistakes.

Note: this sheet covers all Macleod for the cranial nerves and the doctors lecture,(no need to go back to it)

References:

- Macleod's Clinical Examination, 13th Edition
- Doctors lecture (record)
- Different sources on the internet

All the boxes are for illustration and information from Macleod, which I thought to be necessary to include here, so if you are short on time skip them.

Layout of the sheet is; for each cranial nerve(1N-12N) there's it:

- Anatomy
- Abnormalities/disorders
- The examination
- Beside some illustration that are important from the doctor or the book.

Good luck for all

General CNS (cranial nerves) anatomy

The nervous system is divided to two parts: peripheral (spinal nerves and cranial nerves) and central (brain, brain stem and spinal cord)

Spinal nerves are 31 pairs all of which are mixed (both motor and sensory)

Cranial nerves are paired except the first (olfactory)

Cranial nerves are called so because they rise from the brainstem (midbrain, pons and medulla oblongata) and pass through different foramina inside the brain skull to reach the periphery (ex the ear or the eye).

Foramina names can be symbolized in **OS ROS**

O: Optic foramen, the optic nerve passes through it

S: superior orbit fissure, 3N, 4N, 5aN(ophthalmic branch) and 6N

R: rotundum, 5bN(maxillary branch)

O: ovale, 5cN (mandibular branch)

S: Spinosum, middle meningeal artery and vein

1N (olfactory) and 2N (optic) emerge from cerebrum of the brain tissue itself (not from the brain stem)

C.N from 3-12 all arise from brain stem

Two C.N ; 3rd (oculomotor) and fourth(trochlear) from the midbrain

Four C.N ; 5, 6, 7, 8 from the pons

Four C.N ; 9, 10, 11, 12 from medulla oblongata

* the doctor only explained C.N from 2 to 8 but said they are included with us. In this sheet, I (self acted) and included them as a Macleod reference

❖ Olfactory nerve (CN1)

Anatomy

Bipolar cells in the olfactory bulb form olfactory filaments with small receptors

Abnormal findings

Hyposmia or anosmia (reduction or loss of the sense of smell respectively) may result from

- ear, nose and throat disease,
 - damage to the olfactory filaments after head injury or
 - local compression or invasion by basal skull tumors.
- ✚ Disturbance of smell may also occur in the pre-symptomatic stages of Parkinson's and Alzheimer's diseases.

Hypogeusia/ageusia (altered taste) associated with anosmia.

Parosmia : when pleasant smell that is good for others is annoying for you (from trauma. tumors. or upper respiratory tract infection)

Examination (not done)

Testing of smell is of limited clinical value, and rarely performed; the pt will come complaining with problems of smelling

❖ Optic nerve (CN 2) – and the 3N 4N 6N

Physioanatomy

The **optic nerve** examination is accomplished with other related nerves; the third ,fourth and the sixth C.N all of which are concerned with the orbit and eye movement and vision

In the eye; Rod photoreceptors are responsible for night vision and detection of peripheral movement. Cone photoreceptors are responsible for color and central vision.

The optic nerve is **purely sensory**; leaving the eye through the optic disc and entering the cranium through the optic canal. The pair joins at the optic chiasm. Leaving the chiasm, the

Visual pathway is renamed the 'optic tract'. The optic tracts terminate in the lateral geniculate bodies (thalamus) and radiate to the occipital lobe,
The visual pathway consists of the retina, optic chiasm, optic tracts, lateral geniculate bodies, optic radiations and visual cortex

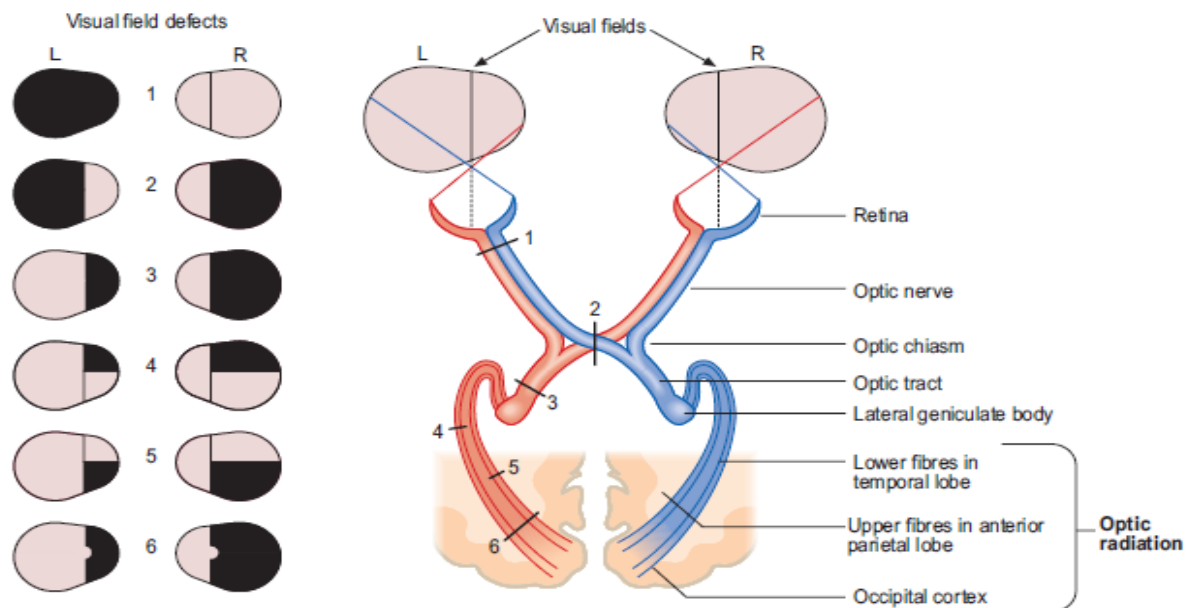


Fig. 12.3 Visual field defects. (1) Total loss of vision in one eye because of a lesion of the optic nerve. (2) Bitemporal hemianopia due to compression of the optic chiasm. (3) Right homonymous hemianopia from a lesion of the optic tract. (4) Upper right quadrantanopia from a lesion of the lower fibres of the optic radiation in the temporal lobe. (5) Lower quadrantanopia from a lesion of the upper fibres of the optic radiation in the anterior part of the parietal lobe. (6) Right homonymous hemianopia with sparing of the macula due to lesion of the optic radiation in the occipital lobe.

This Pic (visual fields) is very important and expect questions from it either in the data interpretation or the written exam

The visual field is divided into two parts; medial (the nasal part-close to the nose), and lateral (the temporal part-close to the temporal bone).

The optic nerve is also divided same in this matter; the nasal part of the optic nerve (Num 1 in Pic.) and the temporal part of the optic nerve

The nasal part of 2N takes part from the temporal visual field and the temporal part of 2N takes from the nasal visual side.

The optic chiasm, in which the nasal parts of both optic nerves cross, is susceptible to compression from the pituitary gland (ex pituitary adenoma or prolactinoma) lead to inability to see on the sides for both eyes; called **“bitemporal hemianopia”**

How does this happen?

The pituitary gland is situated in the sella turcica where the optic chiasm of the optic nerve (Num 2 in Pic.) is placed. The fibers of the nasal part of optic nerve (from the temporal visual field), pass through it and they cross to the other side “decussation”

Note: Both of the nasal part passes through the optic chiasm **but** those of the temporal part do not do decussate (meaning they stay at the same original side)

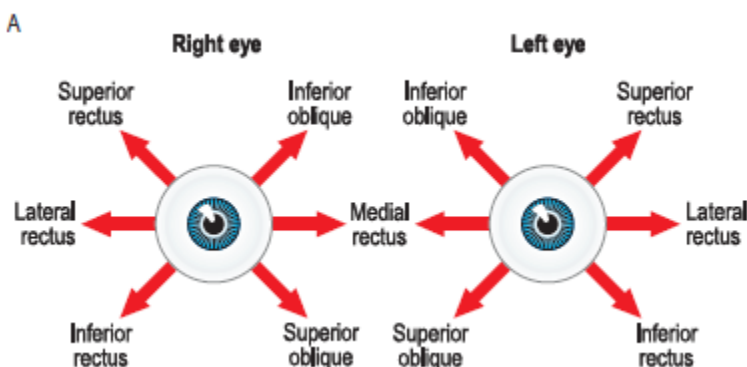
So let's say if there is a pituitary adenoma; the mass effect of it compresses the optic chiasm (compressing the nasal part of the left and the nasal part of the right carrying the temporal vision field of the right and the temporal vision of the left respectively)

Ex of clinical presentation of pituitary adenoma;

A patient comes because multiple car accidents complaining that he can see cars in front of him but all of a sudden cars hit him from his side, we scan him to see the pituitary (MRI)

The optic tract which carries both nasal and temporal parts continues the visual pathway until it reaches the cortex area (vision area 17) in the occipital lobe to get analyzed

The Optic tract (num 3 in Pic.) if injured at any place will give us special vision problem but we (csl5) are concerned on the optic chiasm



Eye muscles, functional view of the eye movement (not anatomical)

Eye muscles

Inward eye moment (medially) ; adduction

Outward eye moment (laterally); abduction

They are six eye movement muscles grouped in: rectus group and oblique group

Rectus muscle group

1. Lateral movement: abduction
2. Superior rectus :upward and lateral movement
3. Inferior rectus : downward and lateral movement
4. Medial rectus : adduction

Oblique muscle group;

1. Superior oblique :downward and medial movement (though it's in a superior anatomical position)
2. Inferior oblique :upward and medial movement (though it's in an inferior anatomical position)

Eye motor muscles are innervated by the 3N 4N 6N

The oculomotor (III), trochlear (IV) and abducens (VI) cranial nerves innervate the six external ocular muscles controlling eye movement and, also affect pupillary size through parasympathetic nerves.

Nerve(num)	Emerge	Pass through (foramen)	Function
oculomotor (III)	tentorium	superior oblique fissure	Muscles; open the upper lid (levator palpebrae superioris) Move the globe upwards (superior rectus, inferior oblique), downwards (inferior rectus) Medially (medial rectus). Parasympathetic; supplies (1)sphincter muscles of the iris, (pupil constriction) (2) ciliary muscle (focuses the lens for near vision (accommodation))
trochlear (IV)	Midbrain (the left nucleus innervates the right trochlear nerve and vice versa).	superior orbital fissure	Muscles; superior oblique muscle, which causes downward movement of the globe when the eye is adducted.
abducens (VI)	brainstem	superior orbital fissure	Muscles; lateral rectus muscle. Lateral rectus abducts the eye

All muscles of the eye **except** the superior oblique and lateral rectus are supplied by the 3rd cranial nerve (oculomotor N.)

- superior oblique from 4N trochlear
- lateral rectus from the 6N abducens

Acronym from doctor; all eye muscles take from 3N except
So4(كبريت): **S**uperior **O**blique from the **4**N) and Lr6

✚ As like all muscles of the muscular system the eye muscles have an opposite muscle (agonist and antagonist) functionally to keep the balance. So if one side is damaged the balance is disrupted and pulls the eye to the opposite of the weak muscle (towards the normal one)

Ex when the medial rectus muscle is damaged the eye will be sided to the lateral side (to the strong muscle)

Superior rectus damaged: the eye is towards the ground (downward)

Abnormal findings (disorders/diseases)

- **eye position displacement:**

Can be either caused by damage to the muscle or nerves of the eye

In a neurological born abnormal eye position, it is surgically dealt by cutting both sides of the muscles repositioning the eye correctly and then its re-stitched

Strabismus

[Case one]



As seen in Pic.1 the pt's right eye is:

1. going downward and lateral
2. the eyelid of the right is down (called ptosis)
3. the pupil is dilated

Explanation:

1. If hypothesized that the oculomotor nerve was damaged, the opposite muscles will create the movement, which are the superior oblique (pulls the eyes downwards) and the lateral rectus (pulls the eye laterally)>> so if the 3N damaged with intact 4N and 6N, the eye position will be towards downwards and laterally ..and that's what's presented in picture
2. the action of pulling the eyelid up is the levator palpebrae superioris muscle
Innervated by the oculomotor nerve >>so if damaged the eyelid will go down (called ptosis)
3. one of the functions of the oculomotor nerve is parasympathetic action, supplying the constrictor muscle of the eye (circular iris)>> so when damaged the opposing dilator muscle will be stronger leading to dilatation of the pupil

by 1-2-3 we conclude that the right oculomotor nerve is damaged

Light reflex or bilateral pupil reflex(or the pupillary reflex) : when beaming light on one of the eyes, as result of parasympathetic action, both of the eyes are constricted. This is due to inter-neuron connection in the Edinger–Westphal nucleus that receives the afferent fibers from **(2N)** .

the pupillary reflex involves the optic nerve, chiasm (where some fibers decussate) and the optic tract, bypassing the lateral geniculate nucleus to terminate in the III nerve (Edinger–Westphal) nucleus

i.e. : so if we shine a torch on the right eye, the right optic tract gives signal to both the right and left Edinger–Westphal nucleus. This nucleus by efferent fibers (by the inferior division of **3N**) cause a parasympathetic reflex action causing both of the pupils to constrict.

- The right pupil constriction is called the direct reflex (light pointed on it)
- Left pupil constriction is called indirect reflex (as a result of nucleus connection)

[Case two]



As seen in Pic.2 the pt's right eye is

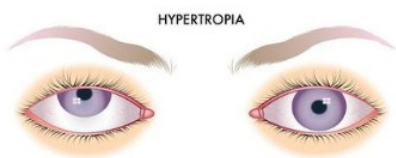
1. right eye is going inward (medially)
2. both of the eyelid are normal
3. the pupil is normal

Explanation:

The right eye is going inward (medially) meaning the opposing muscle, the lateral rectus muscle isn't functioning.. This is called right (**convergent squint or esotropia**)

- Cause : damage to the right 6N abducens nerve supplying the lateral rectus (with intact 3N and 4N)

[Case three]



In Pic. The eye is going upwards (and only) the medial and lateral are intact, called (Hypertropia)

Explanation:

Because the of eye is going upwards (and only) meaning the other muscles are functioning normally

- Cause: this abnormality is a result of a dysfunctional Superior oblique muscle therefore the damage is to the 4N

Visual disturbance

The curvature of the optic lens alters to adapt the focal length to suit the varying distance and produce a clear, focused image

Visual acuity:

Hypermetropia (long-sightedness): [بعد النظر]; can see the far away things but not the close ones

Why? Because the cornea (the lens is behind it) abnormally leveled, leading to collecting the light and focusing it behind the retina than on its normal level

- It is common in infants

Myopia (short-sightedness): [قصر النظر]; can see the close things but not far away ones.

Why because the lens is collecting the light and focusing it in front of the retina

Presbyopia happens in old people. They can't focus on small things in close range, why? Because the muscles responsible for accommodation are weakened (impaired power) (by age... >45) >> so it's hard for old people to focus their eye on close range objects **but they** still can see in far range.

- Pts are given reading glasses to use for close range activities
- Note: they do not necessary have abnormalities in their lens or retina therefore not necessary presented with **hypermetropea, anoopia or ... Astigmatism**

Astigmatism: the cornea, which is responsible of collecting the light on the lens, is abnormal (cornea is irregularly curved), leading to scattering the light and preventing its collection on the lens therefore the light is weakened and not focused probably on the retina.

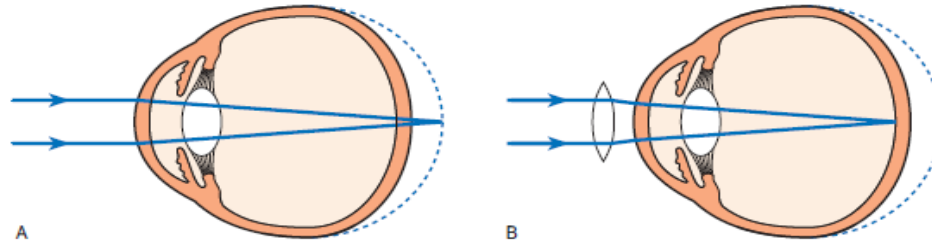


Fig. 12.9 The hypermetropic (long-sighted) eye. (A) The eye is too short and the image on the retina is not in focus. (B) The use of a convex (plus) lens brings the image on the retina into focus.

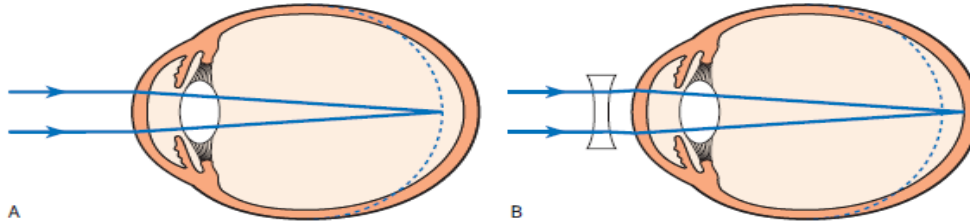


Fig. 12.10 The myopic (short-sighted) eye. (A) The eye is too long and the image on the retina is not in focus. (B) The use of a concave (minus) lens brings the image on the retina into focus.

A student asked about “كسل العين“

It's a result of a genetic defect in the optic nerve, weakening its functionality. But not weakness in the muscles of the eye

Treated by closing the strong eye and keeping the weak eye open, this will lead to hyperactivity (hypertrophy), stimulating the eye to be stronger

Addition eye abnormalities see last page in this sheet P.32

Examination (for the 2n..3N..4N..6N as a group)

*1- Introduce you self and take patient name . 2- Take permission and cooperation of patient . 3- Hand hygiene. 4- Ensure privacy(Ensure the pt that all data will be confidential between you and him/her). 5- Make sure there's a chaperone (someone with the patient). 6- Good position of patient and exposure for examining (in here mostly the pt will be sitting down) 7- explain the procedure to the pt prior to each act ...**always do bilateral**(or explain to examiner that you have to do bilaterally) 8-gain consent*

Ques.: A patient comes with a complaint of decrease visual acuity; please do a cranial nerve examination for the second third, fourth and sixth cranial nerves

1. inspection

Head position (an abnormal head posture with the head turned or tilted to minimize the diplopia)

Look at the upper eye lid if for ptosis(drooping eyelid) ,proptosis(forward bulging of the eyeball.. sclera is visible)or blepharospasm (tonic spasm in the orbicularis oris muscle).

Look for periorbital edema (the surrounding of the eye is enlarged)

- Trauma to the eye
- in nephrotic syndrome
- heart failure patients

Look at the eye ball itself if there's exophthalmos

- we look from the lateral or posterior aspect to check the eye protrusion)

Look for eye discharge

Look at the sclera for redness , discharge and color

Look at the cornea for corneal ulceration

Look at the conjunctivae (upper and lower)

- ask the patient to look down
- To examine the upper conjunctivae. We put a cotton pick swamp on the upper lid then bent the lid on the pick to be fully covered by the conjunctivae (see Pic 1).
- Look for the giant papillae(allergic eye disease) , hidden foreign body, for redness or chemosis (oedema) of the white of the eye.
 - The lower conjunctivae; same as when examining the pallor of the eye (I.e. in anemia)... ask the patient to look up (see Pic 2)

Look for lid lag (delay in moving the **eyelid** as the eye moves downwards) (see Pic 3)

- Examine the seated patient from the right.
- Hold your finger from a point 45° above the horizontal to a point below this plane.
- Watch how the upper eyelid moves with the downward movement of the eye.
- ✚ Normally, there is perfect coordination as the upper lid follows the downward movement of the eye. In lid lag, as occurs in thyroid eye disease, (the white)sclera can be seen above the iris

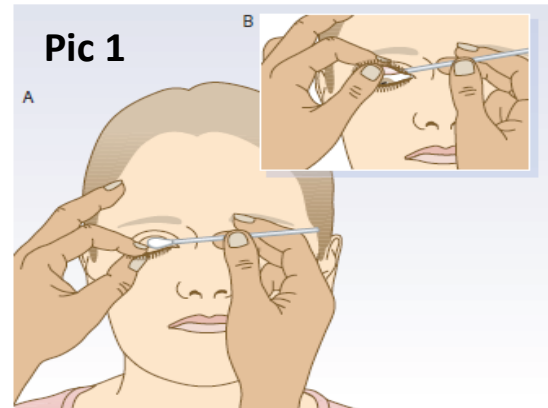


Fig. 12.13 Everting the upper eyelid to look at the conjunctiva.

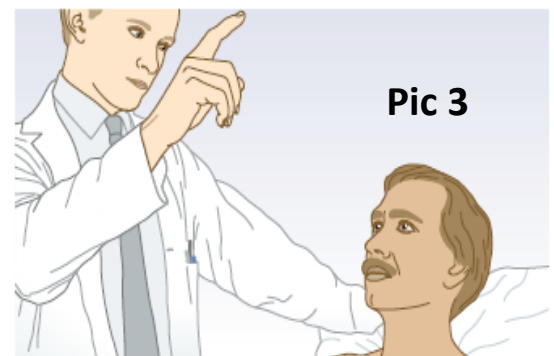
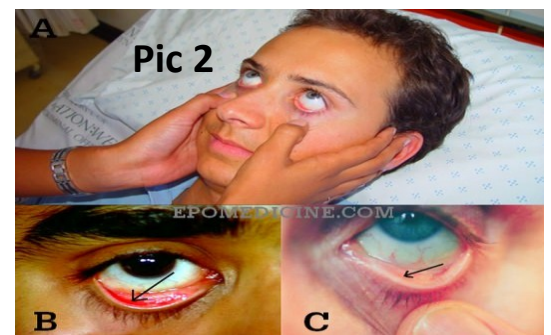


Fig. 12.12 Testing for lid lag.

2. Physical examination for visual acuity:

Before starting the test we ask the patient if he wears medical glasses, if the patient uses glasses we ask the patient to wear them

- look for four problems/signs;

Hypermetropi, Myopia, Presbyopia and Astigmatism



Fig. 12.17 Snellen visual acuity chart.

- ask pt if wears glasses if so to wear them
- We set the pt six (6) meters away from the chart (called Snellen chart)
- Cover one of the patients eyes with a card
- ask him to read from the top down until he can no longer distinguish the letters, while describing what he sees (character or number or shape)
- note down the level (distance) that he can't describe the characters

i.e., if a patient couldn't see at level 12 that means the visual acuity of the eye is 6/12, meaning what's supposed to be seen at a distance of 12 meters can only be seen 6 m from the object (normal person can see the object clearly at 12 m but the patient sees it clearly at 6

Scenario:

If the patient didn't see the first level (60/6), we ask the patient to set one (1) meter away from the chart, if still he couldn't see from one meter we wave our hand (big objective like the palm of the hand) to look for any response, if still couldn't see we flash a light on his eyes to observe his visual activity ...if the pt reaches this stage then the patient has a serious visual acuity problem

3. Visual field

Remember: the visual field is divided to nasal and temporal part

Test by four steps:

1. **Homonymous** defect

- Set the patient so that the distance between you and him is 1 meter
- Tell the patient to keep his head fixed during the examination, looking towards you and to keep his eyes fixed with your eyes (نظرك معاي)
- Hold your hands out to their full extent. Wiggle a fingertip and ask the patient to point to it when he first sees your finger movement (or to tell you)...you move your finger in x radius way (10 and 2 o' clock, and then 8 and 4 o' clock) (from each angle of a quadrante start from far and close in to the center)

By doing this the doctor is comparing the visual fields of the patient with his (normal) because both eyes are lined at same level (opposite to each other)

2. Sensory **inattention**

- Set the patient so that the distance between you and him is 1 meter
- Tell the patient to keep his head fixed during the examination looking towards you and to keep his eyes fixed with your eyes (نظرك معاي), we Test both eyes together
- Both you and the patient should keep your eyes open. (Test both left and right fields at the same time.)
- Raise your hands and move one of them, ask the patient to tell you which had you moved, do this to both sides to examine for bilateral normal visual sensation or whether the patient reports seeing only one side.

3. **Peripheral** visual field

Besides describing the visual field as nasal and temporal it can be called peripheral or central

- Set the patient so that the distance between you and him is 1 meter
- Tell the patient to keep his head fixed during the examination looking towards you and to keep his eyes fixed with your eyes (نظرك معاي), we test each eye separately.
- Tell the patient to covers his right eye...the doctor closes the contra to it (here the left) so that both of the patients eye and the doctors are at the same level
- move your finger from far (Peripheral) to center ask the patient to tell you when he first sees it and compare it to your own observation

Ex if the patient had bitemporal hemianopia the patient won't see the fingers coming from the side to center but the doctor will

4. **Central** vision (examine for scotoma)

Central field of the vision can be observed by the ophthalmoscope looking directly to the retina of the eye to check the fovea (the blind spot)

It's a black spot on the retina that doesn't collect any light, present were the optic disk originates (start of the optic nerve), normally is to have one black spot...the abnormal is to have more than one black spot on the retina (called scotoma) meaning more than one blind spot on the visual field

Set the patient so that the distance between you and him is 1 meter

- Tell the patient to keep his head fixed during the examination looking towards you and to keep his eyes fixed with your eyes(نظرك معاي), we test each eye separately.
 - Tell the patient to covers his right eye...the doctor closes the contra to it (here the left) so that both of the patents eye and the doctors are at the same level
 - **First**, Hold the hatpin (pointed pin) at the fixation point; you and the patient focus on each other's eye.
 - Ask the pt to describe the color of the pin.
A 'pale' or 'pink response implies color desaturation, usually because of a lesion affecting the optic nerve.
 - Compare the four quadrants of the visual field centrally; each time ask about color desaturation.
 - **Second**, Move the hatpin temporally and horizontally until it disappears from your visual field. Maintaining the same temporal horizontal position, move it anteriorly or posteriorly until it also disappears from the patient's visual field.
 - Compare the size of the patient's blind spot to yours.
 - **Third**, move the pin in the quadrant movement and ask the pt if the color(redness) changes at any point, if changed (called color desaturation)
-

4. **Ocular** movements (examination to the muscles of the eye, examine diplopia and Nystagmus)

- Set the patient so that the distance between you and him is 1 meter
- Tell the patient to keep his head fixed during the examination) Hold your finger vertically at least 50 cm away from the patient, (arms length)
- Ask the patient to follow your fingers as you move it in a **H movement** and if he sees your finger doubled (double image) to tell you. Called “diplopia”
 - If present, cover one of the patient’s eyes to see if diplopia is monocular or binocular.

We do the test **twice**, why? The first time to examine for diplopia (if there’s double imaging) and the second time to examine for Nystagmus (abnormal eye movement the eye doesn’t move smoothly across a wide movement, (slow drift in one direction: زي التقطيع)

Note we don’t do it twice if the patient in the first time didn’t present with diplopia, just continue to examine the eye movement for Nystagmus, but if complained about having a double vision we do another separate one to check the eye movement (Nystagmus)

5. The pupils

- Set the patient so that the distance between you and him is 1 meter
- Ask the patient to fix his eyes on a distant point straight ahead.

Do two tests:

First, **inspection:** (we dim the lights in the room)

- Bring a bright torchlight from the side to shine on the pupil and look at the symmetry and the shape of the pupils (there’s no abnormalities in the right and left pupils)

Ex. ..Pt with constricted pupils might be due morphine doses

Pt with fixed dilated pupils might be from internal bleeding, brain stem lesions or death (it’s a sign of death)

Second, **light reflex** (direct and indirect)

- Put the torchlight to shine on pupils, coming from the lateral aspect of the pt
- And look for pupil constriction on the both eyes

The other eye that didn't receive light, but had a constriction reflex is called indirect reflex or "consensual light reflex").

6. Convergence and accommodation (to examine the ciliary muscles that are responsible for the changes in the lens diameter)

- Approach the pt from his lateral aspect
- ask the pt to look forward at the wall and to fix his vision
- approach a pencil(object) to him from the side and ask the patient to focus on it

In normal response, two things occur:

- convergence (meaning both the eyes go medially inwards)



- accommodation (Look for pupil constriction); the image (pencil) gets clear for the pt ...we ask the pt if he refocused on the pencil once it came in his field of vision
-

7. Color vision examination, Squint (cover test), Oculocephalic (doll's-eye) reflex and Ophthalmoscopy test: **not acquired from us**

❖ Trigeminal nerve (5N)

Anatomy :

Is the biggest of the cranial nerves and is divided to three divisions: ophthalmic, maxillary and mandibular. Those carry both sensory and motor information (mixed)

The sensory part receives from the face and the mouth

Two types of sensation ; general sensation ex touch

And special sensation ex taste (sweet, sour, salty)

- For the face its general sensation

The motor information gives its information to muscles of mastication (three of four muscles of mastication). The masseter, the medial and lateral pterygoid and temporalis

Pic of each muscle position and nerve innervation

- for the tongue

The tongue anatomically is divided to anterior 2/3 and posterior 1/3

Anterior 2/3 take its **general sensation** from the trigeminal nerve 5N and the **special sensation** from facial 7N nerve

Posterior 1/3 gives it sensation (**both general and special**) to glossopharyngeal nerve(9N)

- Sensation; (face, mouth and part of the Dura,) ,sensory ganglion; trigeminal (Gasserian) ganglion.
- Motor; temporalis, masseter, medial and lateral pterygoid (muscles of mastication).
- Connection; pain and temperature pathways descend to the C2 segment of the spinal cord, so ipsilateral facial numbness may occur with cervical cord lesions.

Nerve (division)	type	Pass through	Function
ophthalmic (V ₁)	Sensory	superior orbital fissure	skin of the upper nose, upper eyelid, forehead and scalp, V ₁ supplies sensation to the eye (cornea and conjunctiva) and the mucous membranes of the sphenoidal and ethmoid sinuses and upper nasal cavity
maxillary (V ₂)	Sensory	Foramen rotundum.	Upper mouth, roof of pharynx, gums, teeth and palate of the upper jaw and the maxillary, sphenoidal and ethmoid sinuses.
mandibular (V ₃)	Sensory and motor.	foramen ovale	floor of the mouth, common sensation, i.e. not taste , to the anterior two thirds of the tongue, the gums and teeth of the lower jaw, mucosa of the cheek and the temporomandibular joint in addition to the skin of the lower lips and jaw area, but not the angle of the jaw

Abnormal findings

Cause; result

- (trigeminal neuralgia); facial numbness and pain.
- injury in association with facial fractures ;Unilateral loss of sensation (particularly V₂) or by local invasion by cancer.
- Lesions in the cavernous; loss of the corneal reflex , V₁ or V₂ cutaneous sensory loss.
- neurovascular compression(Trigeminal neuralgia); severe, lacerating pain typically in the distribution of V₂ or V₃
- VZV virus (chickenpox); any sensory nerve,(particularly, thoracic dermatome or V₁)
- Myasthenia gravis; weakness of the muscles of mastication
- pseudobulbar palsy; brisk jaw jerk.



A



B



C



D

Fig. 11.8 Herpes zoster. (A) The ophthalmic division of the left trigeminal (V) nerve is involved. **(B)** The maxillary division of the left V nerve. **(C)** Cervical spinal root left C5. **(D)** Thoracic spinal root right T8.

Examination

Introduce yourself.....same as before

1. sensory

first; general sensation of **the face

First examine the sensory part on three places on the face (skin) -- (that represent the trigeminal divisions)

When examining the sensation of the trigeminal we examine the light touch and the pain

- light touch
 - we ask the patient to close his eyes
 - tell the him that I am going to swab on your face and when you feel anything just say yes
 - we swab (by a cotton wool tip) on each area of the trigeminal divisions as shown in Pic
 - Compare both sides

- pain (superficial pain)
 - same as before; use a small sharp object (pin) and the same as light touch
 - Compare both sides

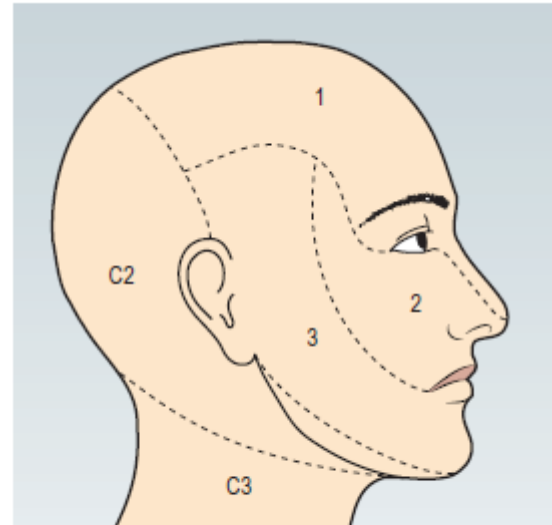


Fig. 11.5 The sensory distribution of the three divisions of the trigeminal nerve. (1) Ophthalmic division. (2) Maxillary division. (3) Mandibular division.

Second, sensory of that to **the anterior 2/3 of tongue (general sensation);

Use like an ear pick (for our exam we use wooden swab sticks)

- Ask the pt to close his eyes
- Ask the pt to protrude his tongue as much as possible, and touch it by the pick
- let him tell you when he feels the pick

For knowledge (not to be done)

Nasal tickle test: use a wisp of cotton wool to tickle the inside of each nostril and ask the patient to compare: it is an unpleasant sensation easily appreciated by the patient

2. Motor part (muscles of mastication) (signs rare)

- We inspect (**inspection**) the muscles looking for at their bulking (atrophy, muscle wasting or normal).. (Most apparent in temporalis).
- Then ask the pt to shut on his teeth (to clench his teeth);to **palpate the bulk** of the masseter muscle (feel the cheeks)
- Now to examine the medial and lateral pterygoids;
We put our hand underneath the chin and ask the pt to open his mouth against the resistance of our hand ...to **asses the power of the pterygoids muscles** (if the pt has weakness in the pterygoids he wouldn't be able to open his mouth)

3. Reflexes

There are **two** reflexes to examine; the corneal reflex and the jaw reflex

- **The corneal reflexes** (only for knowledge, not to be done...say "I'm going test... ")

Is related to the ophthalmic division as the afferent fiber. When touching the cornea the pt closes both his eyes (blinks bilaterally) as action of the facial efferent fibers.

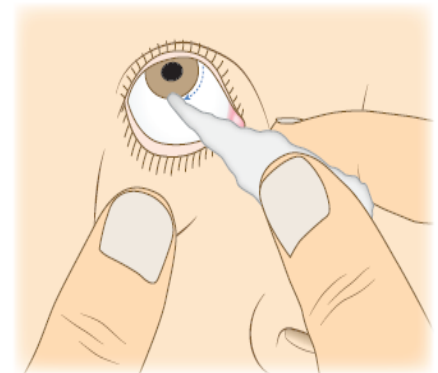


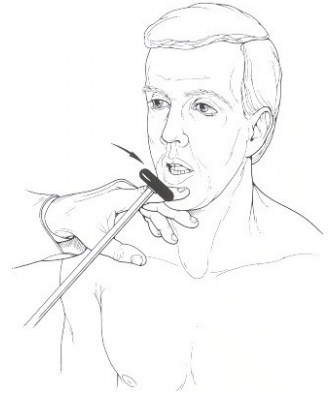
Fig. 11.6 Testing the corneal reflex.

- Explain to the patient what you are going to do, and ask him to remove contact lenses, if relevant.
- We ask the pt to look upwards we pull his upper eye lid up (pulling the conjunctiva).
- We use a swab to touch the lateral edge of the cornea of the pt eye ..normally the pt's eyes blink (both; direct and consensual) as a result of the reflex

- **the jaw reflex**

Is related to the mandibular division for the both afferent and efferent fibers

- we ask the pt to open a slight gap between his teeth (mouth hangs loosely open)
- Place your forefinger in the midline between lower lip and chin.
- Percuss your finger gently with the tendon hammer in a downwards direction



Normally; An absent, or just a slightly present reflex

Why this reflex is imp? Because in upper motor neuron lesions (like in stroke) when provoking these reflexes they will give us **hyperreflexia** (shut the teeth powerfully..Most likely to hear a snap)

❖ Facial nerve (7N)

Anatomy

- has a motor part that is related to facial expressions
- parasympathetic part related to the glands (sublingual and submandibular -that make saliva- and lacrimal gland) via nervus intermedius
- And sensation: special sensation (taste) to the anterior 2/3 of the tongue (via the chorda tympani branch)
- provides the efferent supply to several reflexes

Nerve (num)	emerge	Passes through (foramen)	function
Facial nerve (7N)	emerging from the lateral pontomedullary junction	Enter the internal acoustic meatus bone, exiting the skull Via the stylomastoid foramen.	branches to the stapedius muscle and its parasympathetic fibers, as well as being joined by the taste fibers of the chordae tympani

Abnormal findings

▪ lower motor neuron lesion (weakness of both upper and lower facial muscles).

- Bell's palsy; common in lower motor neuron lesion, preceded by mastoid pain with impairment of taste and hyperacusis

Bell's phenomenon occurs when the patient is unable to close his eye. As he tries, the eyeball rolls upwards, exposing the conjunctiva below the cornea

- Ramsay Hunt syndrome; in herpes zoster infection to (facial) ganglion
severe lower motor neuron facial palsy, ipsilateral loss

of taste and buccal ulceration, and a painful vesicular eruption in the external auditory meatus

- tumours; lower motor neuron lesion

- Synkinesis; (most commonly twitching of the corner of the mouth on ipsilateral blinking) is a sign of aberrant reinnervation

▪ upper motor neuron lesion: (weakness is marked in the lower facial muscles)

• this is because there is bilateral cortical innervation of the upper facial muscles

- The nasolabial fold; flattened and the corner of the mouth drooped.

- Involuntary emotional movements, (spontaneous smiling is preserved)

▪ Bilateral facial palsies are less common

- Guillain-Barré syndrome

- sarcoidosis

- Lyme disease

- HIV infection

- Distinct from VII nerve palsies, Parkinson's disease can cause loss of spontaneous and involuntary facial movements

Examination

Introduce yourself.....same as before

We Examine Two parts; motor and taste (note that the taste is not done because it's obvious and the pt will come complaining of loss of taste and sensation of flavors)

The motor examination (examine the facial expressions)

▪ First we **inspect** (inspection) :

1. Look at the symmetry of the face (for asymmetry or differences in blinking or eye closure on one side).
2. Eyelid (has the ability to close or not, other words if he wrinkles normally)
3. Nasolabial folds (if there's deviation or not)
4. Angle of the mouth (if there's deviation or not)

▪ Second, We test action of the muscles (**Test power**) of the eyelid and cheeks (buccinators)

- We ask the pt to raise up his eyebrows and keep like this, resulting in the wrinkling formation on both the right and left ..by this movement the pt used the Occipitofrontalis muscle that is innervated by the facial nerve
- Ask him to show his teeth with a wide smile. we examine the angle of the mouth (for no deviation in the angle of the mouth to the right or left) and for Nasolabial folds (if there's deviation or not)
- We ask the pt to close his eye and resist the action of me trying to open his eyelids, the muscle that is responsible of closing the eyes is also innervated by the facial nerve of action
- Ask him to Blow up his cheeks and try resisting the power of my fingers pushing on the check ...to examine the buccinator muscle that is innervated by the facial nerve.

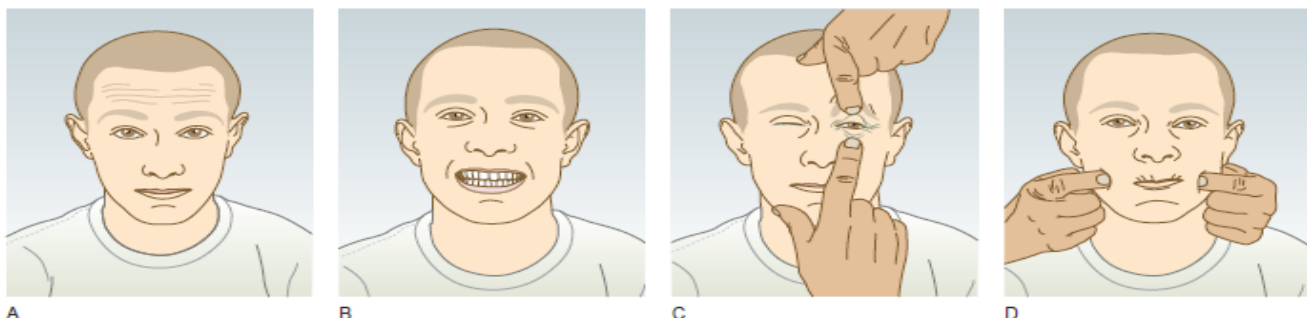


Fig. 11.11 Testing the motor function of the facial nerves. (A) Ask the patient to raise his eyebrows. (B) Ask him to show his teeth. (C) Ask him to close eyes against resistance. (D) Ask him to blow out his cheeks.

Examples: (lesions affecting the facial nerve (motor))

Remember: The motor part of the facial nerve is divided to upper and lower neurons;
The upper motor neurons control the lower half of the facial expression muscles
Meanwhile the lower motor neurons control all (both upper and lower part) of the

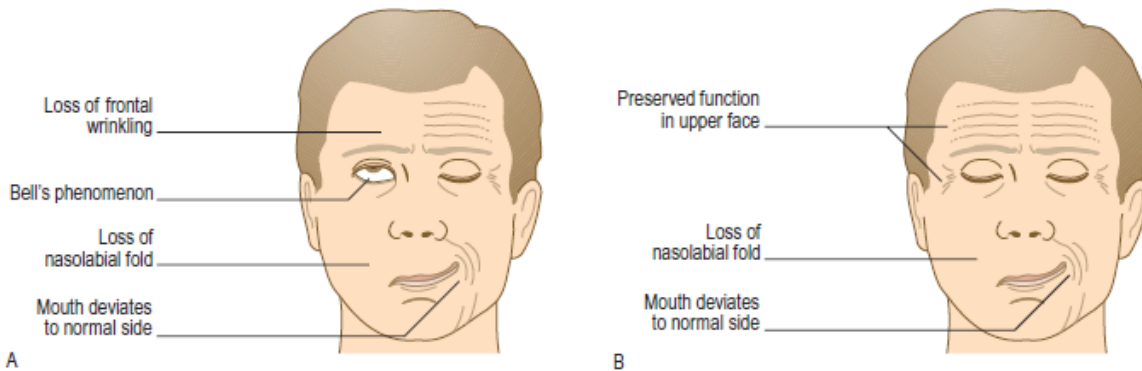


Fig. 11.12 Types of facial weakness. (A) Right-sided lower motor neurone lesion (within facial nerve or nucleus); Bell's phenomenon is also shown. (B) Right-sided upper motor neurone lesion.

From the Pic A , We see

- There's no wrinkling
- the eye lid isn't able to close (bell's phenomenon)
- The nasolabial fold is absent
- The angle of the moth has deviation to the normal side

✚ This means all upper part and lower parts are effected>> it's a lower motor neuron lesion of the right facial nerve

From the Pic B , We see

- There's wrinkling
- Eye can be closed
- No nasolabial fold
- Mouth deviation to the other side

✚ Meaning the lower part of the face is effected (upper motor neuron lesion of the right 7N)

❖ Vestibulocochlear nerve (8N)

Anatomy

Nerve (division)	emerge	Passes through (foramen)	function
the cochlear nerve	Lateral floor and wall of the fourth ventricle in the pons and medulla.	internal acoustic meatus	carries auditory sensory information from the cochlea of the inner ear directly to the brain
The vestibular nerve.	Lateral floor and wall of the fourth ventricle in the pons and medulla.	internal acoustic meatus	travels the vestibular system of the vestibule of inner ear , reflects gravity and linear accelerations of the head

Abnormal findings

13.1 Symptoms and definitions in ear disease		
Symptom	Definition	Common cause
Otalgia	Pain	Otitis media or externa, referred from pharyngitis, trauma or, rarely, cancer
Pruritus	Itching	Otitis externa
Otorrhoea	Discharge	Eardrum perforation with infection Otitis externa Eardrum perforation, severe trauma causing leak of cerebrospinal fluid Granulation tissue from infection, trauma
	Purulent	
	Mucoid	
	Blood-stained	
Hearing loss	Deafness	
Tinnitus	Noise in the absence of an objective source	Presbycusis, noise damage
Vertigo	Hallucination of movement	Inner-ear disease
Unsteadiness		Vestibular or central disease

Hearing loss

Hearing loss may be due to failure;

- of the VIIIth nerve or its endings(sensorineural)
- the conduction mechanisms of the middle ear by fluid, fixation or drum perforations or wax obstructing the external auditory meatus.

Nystagmus:

an involuntary rhythmical oscillation of the eyes, The commonest form;

- Horizontal jerk nystagmus, a slow (pathological) drift of both eyes in one direction, then a fast correction in the opposite direction,(due to a central lesion).
- Pendular nystagmus (oscillations equal in rate and amplitude about a central point) occurs with central vision defects.

Examination

Introduce yourself.....same as before

To testing hearing ..Three methods are included

The function of hearing consist of two parts; the conduction and the nerve part, the conduction pathway is:
air- tube - membrane - bones(3)- receptors

- Check for the conduction of the ear, Conduction problems (ex. Wax, membrane rupture, inflammation in the bones (osteitis)
 1. Whispering test:
 - Stand behind the patient
 - close one of the pt's ear (ex by a cotton)
 - And at a distance of **15 cm** of the pt's ear, whisper and tell the pt to repeat what you've said
 - repeat the action but each time get away from the pt
 - ✚ (normally the person can hear your whisper till you're 60 cm away from his ear
 2. Weber's test:
 - using the tuning fork (either type doesn't matter) hit it then put it on the middle of the forehead
 - ask the pt if he can locate were he hears the vibration more (on the left or right ear)
 - ✚ normally the pt hears in both equally, if there's a problem the pt will hear more in the normal ear not the obstructed one
 3. Rinne's test :

Note that air conduction is better that bone conduction(AC > BC)

 - We Place the vibrating fork at the patient's external auditory meatus; ask if he can hear it.
 - Place the still-vibrating base on the mastoid process.
 - Ask: 'Is it louder in front, or behind your ear?'
 - ✚ If air pathway is obstructed, the bone conduction is heard better than air conduction

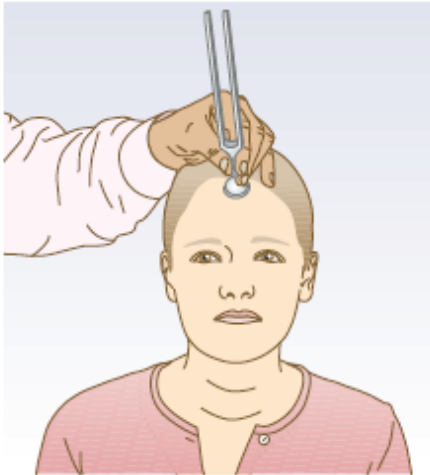
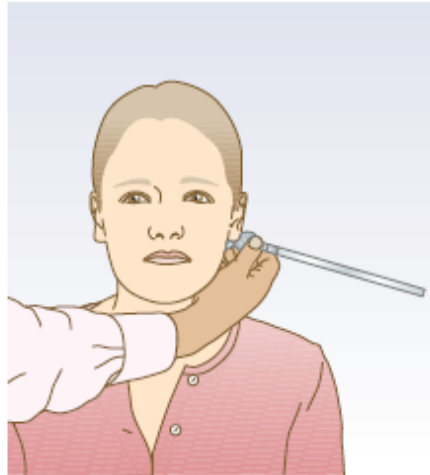


Fig. 13.8 Weber's test.



A



B

Fig. 13.9 Rinne's tests. (A) Testing bone conduction. (B) Testing air conduction.

▪ Check for nerve problems:

1. Weber's test:

- ✚ The vibrating sound is more localized to the normal better ear (not the one with the problem) -sound is heard better in the better-hearing ear.
- ✚ In symmetrical hearing loss it is heard in the middle.

2. Rinne's test :

- ✚ is the same (normally, air conduction is better than bone conduction)

❖ The glossopharyngeal (9N) and Vagus (10N) nerves

Anatomy

Both contain sensory, motor and autonomic components. Main functions of IX and X are swallowing, phonation/articulation and sensation from the pharynx/larynx.

Nerve (num)	Emerge	Passes through (foramen)	Function
The glossopharyngeal (IX)	from the lateral medulla	via the jugular foramen	Carries sensation from the pharynx and tonsils, and sensation and special taste from the posterior one-third of the tongue. Supply the stylopharyngeus muscle, Parasympathetic fibers to the parotid gland.
The vagus (X)	from the lateral medulla	via the jugular foramen	Carries important sensory information but also innervates upper pharyngeal, soft palate and laryngeal muscles.

Abnormal findings

- Unilateral X nerve damage (rare) leads to;
 - ipsilateral reduced elevation of the soft palate when the patient says 'Ah', (with deviation of the uvula)
 - dysphonia and a 'bovine' cough, from Damage to the recurrent laryngeal branch (due to lung cancer, thyroid surgery, mediastinal tumours and aortic arch aneurysm)
 - Bilateral X nerve lesions
 - cause dysphagia and dysarthria.
 - nasal regurgitation of fluids and nasal air escape when the cheeks are puffed out

Examination Introduce yourself.....same as before

- **Assess** the patient's **speech** for dysarthria or dysphonia.
- Ask him to say 'Ah'; **look at the movements** of the palate and uvula using a torch.
 - ✚ Normally, both sides of the palate elevate symmetrically and the uvula remains in the midline.
- Ask the patient to puff out his cheeks with the lips tightly closed. **Listen for air escaping** from the nose.
 - ✚ For the cheeks to puff out, the palate must elevate and occlude the nasopharynx. If palatal movement is weak, air will escape audibly through the nose.
- Ask the patient to cough; assess the strength of the cough.
- Testing pharyngeal sensation and the gag reflex is unpleasant and has poor predictive value for aspiration. Instead, and in fully conscious patients only, use the swallow test. Administer 3 teaspoons of water and observe for absent swallow, cough or delayed cough, or change in voice quality after each teaspoon. If there are no problems, watch for the same reactions while the patient swallows a glass of water.

❖ The accessory (11N) nerve

Anatomy

The accessory nerve has two components:

A cranial part closely related to the vagus nerve

A spinal part which provides fibers to the upper trapezius and the sternocleidomastoid muscles, responsible for elevating (shrugging) the shoulders, and head turning/flexing.

Nerve (num)	Emerge	Passes through (foramen)	function
The accessory (XI)	spinal nuclei arise from the anterior horn cells of C1-5.(spinal cord)	Ascend through the foramen magnum, and exit via the jugular foramen	Responsible for elevating (shrugging) the shoulders, and head turning/flexing.

Abnormal findings

XI nerve lesions are uncommon,

- Wasting of the upper fibers of trapezius; displacement of the upper vertebral border of the scapula away from the spine, while the lower border is displaced towards it
- Wasting and weakness of the sternocleidomastoids; myotonic dystrophy.
- head drop; myasthenia, motor neurone disease and some myopathies.

Causes:

- during surgery in the posterior triangle of the neck,
- penetrating injuries
- local invasion by tumour.

Examination Introduce yourself.....same as before

- Face the patient and inspect the sternocleidomastoid muscles for wasting or hypertrophy; palpate them to assess their bulk.
- Stand behind the patient to inspect the trapezius muscle for wasting or asymmetry.
- Ask the patient to shrug the shoulders, then apply downward pressure with your hands to **assess the power** (Fig. 11.14A).
- Test power in the left sternocleidomastoid by asking the patient to turn the head to the right(opposite) while you provide resistance with your hand placed on the right side of the patients chin.



Fig. 11.14 Testing the trapezius and left sternocleidomastoid muscles. (A) Trapezius. (B) Left sternocleidomastoid.

❖ The hypoglossal (12N) nerve

Anatomy

Nerve (num)	Emerge	Passes through (foramen)	function
The hypoglossal (XII)	emerges anteriorly from the brainstem	exits the skull in the hypoglossal canal	passing to the root of the tongue innervates all the extrinsic and intrinsic muscles of the tongue, except for the palatoglossus

Abnormal findings

11.16 Comparison of bulbar and pseudobulbar palsy		
	Bulbar palsy	Pseudobulbar palsy
Motor lesion	Lower motor neurone	Upper motor neurone
Speech	Dysarthria	Dysarthria and dysphonia
Swallowing	Dysphagia	Dysphagia
Tongue	Weakness, wasting and fasciculation	Spastic, slow moving
Jaw jerk	Absent	Present/brisk
Emotional lability	Absent	May be present

upper motor nerve lesions (pseudobulbar palsy) result from;

- vascular disease,
- motor neurone disease(multiple sclerosis, Parkinson's disease)
- dyskinesias

Examination Introduce yourself.....same as before

- Ask the patient to open his mouth. Look (**inspect**) at the tongue at rest for wasting, fasciculation or involuntary movement.
- Ask the patient to put out his tongue. Look for deviation or involuntary movement.
- Ask the patient to move the tongue quickly from side to side.
- **Test power** by asking the patient to press the tongue against the inside of each cheek in turn while you press from the outside with your finger.
- **Assess speech** by asking the patient to say 'yellow lorry' .
- Assess **swallowing** with a water swallow test.



11.17 Summary of all 12 cranial nerves

Nerve	Examination	Abnormalities/symptoms
I	Sense of smell, each nostril	Anosmia/parosmia
II	Visual acuity Visual fields Pupil size and shape Pupil light reflex Fundoscopy	Partial sight/blindness Scotoma; hemianopia Anisocoria Impaired or lost Optic disc and retinal changes
III	Accommodation reflex	Impaired or lost
III, IV and VI	Eye position and movements	Strabismus, diplopia, nystagmus
V	Facial sensation Corneal reflex Muscles of mastication Jaw jerk	Impaired, distorted or lost Impaired or lost Weakness of chewing movements Increased in upper motor neurone lesions
VII	Muscles of facial expression Taste over anterior two-thirds of tongue	Facial weakness Ageusia
VIII	Whisper and tuning fork tests Vestibular tests	Impaired hearing/deafness Nystagmus and vertigo
IX	Pharyngeal sensation	Not routinely tested
X	Palate movements	Impaired unilaterally or bilaterally
XI	Trapezius and sternomastoid	Weakness of neck movement
XII	Tongue appearance and movement	Dysarthria and chewing/swallowing problems



19.14 Glasgow Coma Scale



Eye opening	
Spontaneous	4
To speech	3
To pain	2
No response	1
Verbal response	
Orientated	5
Confused: talks in sentences but disorientated	4
Verbalises: words, not sentences	3
Vocalises: sounds (groans or grunts), not words	2
No vocalisation	1
Motor response	
Obeys commands	6
Localises to pain, e.g. brings hand up beyond chin to supraorbital pain	5
Flexion withdrawal to pain: no localisation to supraorbital pain but flexes elbow to nail bed pressure	4
Abnormal flexion to pain	3
Extension to pain: extends elbow to nail bed pressure	2
No response	1
Record the GCS as a total and its three separate components: e.g. GCS 9/15: E3, V2, M4	

Cortex damage:

A lesion, affecting only the posterior tip of the occipital lobe, may produce central homonymous hemianopia with sparing of peripheral vision. A lesion, affecting only the anterior occipital lobe, may cause homonymous hemianopia involving peripheral vision but sparing central macular vision. Damage to secondary visual areas causes visual agnosia (inability to recognise visual stimuli) and distorted as macropsia (seeing things larger) or micropsia (smaller than reality)

pain:

1. with 'white eye'

Blepharitis

Severe dry eyes are a feature of Sjögren's syndrome.

Migraine visual disturbance associated with headache or eye pain

Cluster headaches may present as ocular pain.

raised intraocular pressure), patients describe seeing haloes around lights.

Optic neuritis or scleritis: Pain on eye movement usually indicates

2. with red eye

uveitis).

scleritis or conjunctivitis. Diffuse redness .

Entropion (an inverted eyelid) leads to painful corneal

Erosion

ectropion (everted eyelid) causes

dryness in the exposed eye.

glaucoma with high intraocular pressure (with cloudy

Corneal oedema clouds the underlying iris,

Acute iritis(The inflamed iris becomes stuck to the underlying lens). The pain is not

as severe

Corneal ulceration may be due to Virus: herpes simplex(mostly), or bacterial infection

scleritis : Pain on moving a red eye

Diplopia (double vision):

caused by imbalance of eye movements(Binocular) or intraocular disease.(unocular)

Blurred vision: an ocular problem,

Sudden-onset visual loss:

- Transient due to the aura of migraine or unilateral amaurosis fugax
- Permanent: due to vascular occlusion. Or indicates occipital lobe infarction.(in the absence of hemiparesis or dysphasia)
- demyelinating optic neuritis (Uthoff's

phenomenon). (Visual impairment)

Gradual-onset visual loss: cataract, age-related, optic atrophy, tumours

Distortion of vision: disruption of the photoreceptors

Flashes and floaters: caused by vitreous degeneration,(>65 years and in myopia), lead to retinal tears.

Haloes: due to corneal oedema: coloured lights seen around bright lights, occur with glaucoma.

Oscillopsia:, objects appear to oscillate, causes mild blurring to rapid and periodic jumping (common in acquired nystagmus)

Nystagmus: is an involuntary oscillation of the eyes that is often rhythmical, with both eyes moving synchronously.

Anisocoria:Anisocoria is inequality of the pupil sizes.