Neuro-ophthalmology

OBJECTIVES:

- Not provided.

There is some difference between f1&f2 slides so make sure to cover all the different point (click here)

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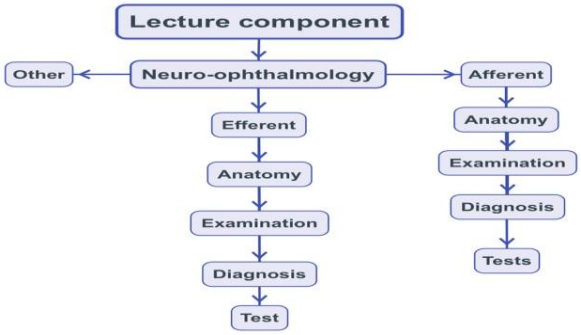
REFERENCES: lecture,
Editing file

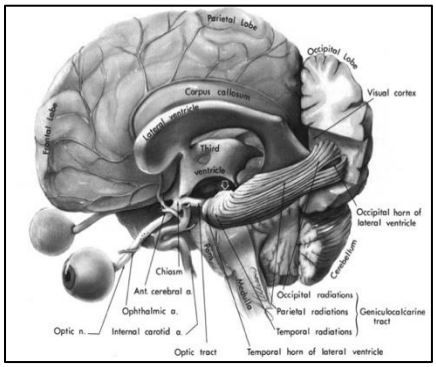
Color index: Important | Notes | Book | Extra

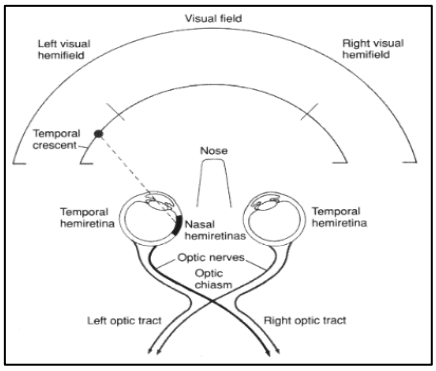
Special thanks to 436 (A) teamwork.

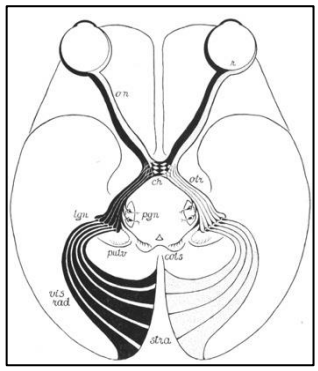


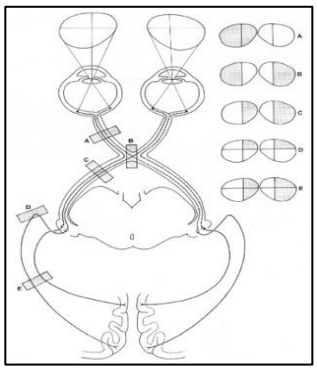
- What is neuro-ophthalmology? It is a sub-specialty that deals with the afferent & efferent visual system.

Afferent Anatomy



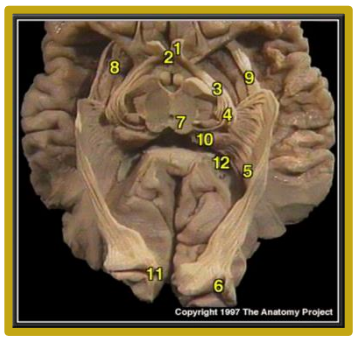






Human visual afferent system

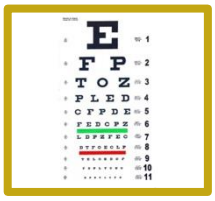
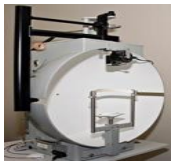
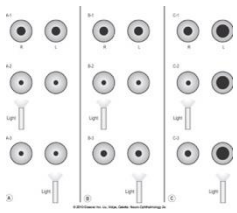
- What is afferent (sensory) visual system? optic nerve.
- What is efferent visual system? pupil, eyelid and ocular motility.
- In afferent visual system:
  - Eyeball
  - optic nerve (lazy S-shaped) going through optic canal.
  - optic chiasm.
  - optic tract.
  - optic radiation.
  - occipital cortex.
- In the visual pathway, we don't say lateral or medial, but we say nasal or temporal.
  - The temporal fibers are responsible for nasal visual field, and the nasal fibers are responsible for the temporal part of the field; There is a cross relationship.
- The afferent system starts at the retina (the nerve fiber layer of retina) forming the optic nerve then optic chiasm → optic tract → optic radiation → occipital cortex.
- There are some fibers that decussate (cross to the other side).
  - The nasal fibers cross to the other side, while the temporal fibers remain in the same side.
  - The amount of the crossed fibers is more than the uncrossed fibers; 53% of fibers cross & 47% of fibers remain uncrossed.



Name structure number 2: optic chiasm.
Name structure number 3: optic tract.
Name structure number 5: optic radiation.

Afferent Visual System Examination

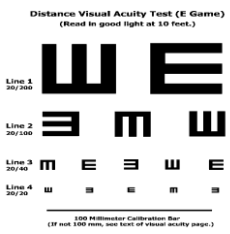
- **How to examine the visual system?**

Visual acuity	Color vision	Visual field	Pupil examination	Fundoscopy
 Uses: to measure visual acuity. 20/20 feet (6/6 meters) mean the patient can see at 20 feet what a normal person can see at 20 feet. It is used for older children & adults (less than 6 years, we use allen chart.	▪ Ishihara test 	• Confrontation test  • Haurmkphrey (automated visual filed test).  • Goldmann. 		▪ Direct ophthalmoscopy. ▪ Indirect ophthalmoscopy. ▪ Slit lamp and lens - Look at the nerve itself.

♦ Why do we need to do all the five examinations? because there might be an injured optic nerve with a good visual acuity but an abnormal visual field, or a good color vision but abnormal acuity and vice versa.

♦ **Visual acuity**

- The visual acuity test is used to determine the smallest letters you can read on a standardized chart (Snellen chart) or a card held 20 feet (6 meters) away.
- The visual acuity can be tested by projecting letters (Snellen chart)
- It can also be tested by using E game chart.
- It is used to test for far and near.



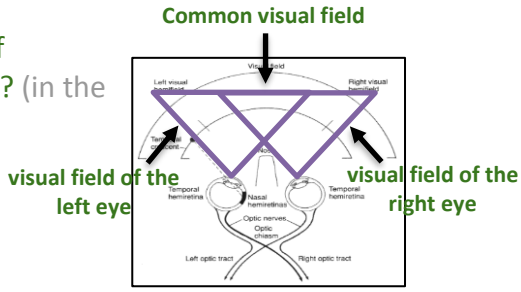
E game chart (extra)

♦ Color vision:

- Ishihara **color plate**. It is a book with multiple plates that has different numbers with colors.
  - Basically, you just name the number. It should be done mono-ocular.
  - Count how many did the patient get correct. 10\15 – 12\15
  - Good for screening of color vision defect, but it does not tell you the type of color defect.

♦ Visual field:

- Visual field means an island of vision. So, if you close one eye, you will see an island that is a triangle in shape, expanding the further it goes.
- That is why when you examine the visual field (by confrontation test), there has to be an equal distance between you and the patient.
- By closing one eye, only **1/5** of the visual field disappears. So, if someone loses one eye, 1/5 of the visual field will be lost. Why? (in the pic)
- If you draw a triangle for each eye. There is a common triangle in the middle represented by both eyes.



- Benefits of having a common visual field:
  1- 3-dimentional vision.
  2- locking mechanisim: our eyes are stright because we see the image from 2 different angels, then the brain will lock them to each other.
- So. If we lose one eye, we lose only the temporal field from one direction.
- **3 types of visual field examination**:
  1- Confrontation test:
    - Good for screening and is a good test for absolute scotoma (total blindness).
    - It is a qualitative method; it doesn't calculate the density of the defect.
    - How to perform the examination:
      - Confrontation visual field testing involves having the patient looking directly at your eye or nose and testing each quadrant in the patient's visual field by having them count the number of fingers that you are showing.
      - The examiner should be one the same level of the patient, at arm's length.
      - Cover one eye because the visual field is mono-ocular & there is a common area represented by both eyes.
      - Project your finger in all the quadrants (ask the patient how many fingers).
      - Put your fingers then take it away (so the patient does not look at it).
      - If there is a defect, you say there is a defect in superior-temporal, inferio-temporal, superior-nasal, or inferio-nasal.
    - On the other hand, you ask the patient "look at my cornea". Then ask: do you see black and white? Do you see lid margins? Do you see the eyebrow? If he\she is able to see the details, that means no central scotoma.
    - In central scotoma: patient will say, I cannot see your eyes, I can see only half of your face.
    - If peripheral, patient will not be able to count your fingers.
  2- Goldmann test: a technician will move a target, then ask the patients if they can see it.
  3- Humphrey: automated. It is a quantitative method; it measures the density of the visual field defect.

- Some patients have relative visual field defect, it is like a mesh in front of their vision; in such a case, you must do actual visual field test by using a machine (Haurmkphrey).

♦ Pupil examination:

1- Dim light.
2- Ask the patient to look at a specific target, not the wall. E.g. electric plug.
3- Shine light from down. Look at both pupils. Are they equal in size, or not?
- Unequal pupil size: anisocoria.
- Unequal refractive error: anisometropia.
- Unequal image size: Aniseikonia.
4- Shine light over one pupil, then observe pupil constriction if it is brisk or weak. Then do the same for the other pupil. Estimate the reaction if equal or not.
5- If you are not sure, do swinging flash test. Swing the light from eye to eye (you have to keep the light for 2 seconds to see the pupil unrest then you shift to the other eye).
- Normally, if you shine a light on the pupil, it constricts then relax a little (this is called pupil unrest or hippus pupil).
- If both constrict → normal.
- If one dilates → this is abnormal & it means there is relative afferent pupillary defect (RAPD+).
    - We call it relative because we are comparing one pupil in relation to the other.
    - RAPD+ tells you there is an afferent visual pathway injury (optic nerve injury), regardless of the cause (e.g. optic neuritis, ischemic optic neuropathy or optic nerve tumor).

- **What is accommodation**? is a change in the lens power or curvature to look at a near & tiny object.
    - The components of near response are:
        1- Miosis.
        2- Accommodation.
        3- Convergence.
    - Near reflex is a part of pupil examination.

♦ Fundus Exam

- Examination is done by:
    1- Slit lamp with lens.
    2- Direct ophthalmoscope (direct because we can pick it up and use it): is a handheld device with a light source & optical center used to examine the pupil. It is monocular with high magnification & image is real (upright).
    3- Indirect ophthalmoscope: is a head-mount device that is binocular, magnification is less, you need to use a lens (usually we use lens with 20 dioptric power).
    - **The Total dioptric power of the eye is 60.**
    - So, 60\20=3 so the magnification is 3 times.
- Common question in OSCE: difference between direct and indirect ophthalmoscope

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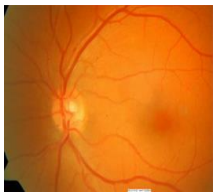
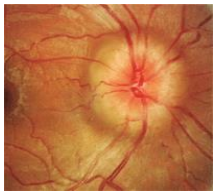
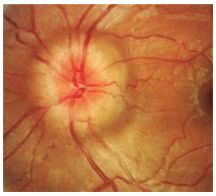
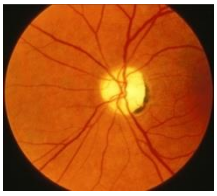
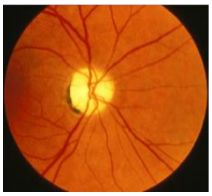
<td>   </td>
<td>   </td>
<td>  
Very yellow pale nerve that indicates damaged atrophied nerve.</td>
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<tr>
<td>♦ This is what we see when we use slim lamp and hand-held lens.
♦ We can see:
1. Optic nerve.
2. Macula.
3. Superior temporal arcade.
4. Inferior temporal arcade.
5. Superior nasal arcade.
6. Inferior nasal arcade.</td>
<td colspan="2">♦ What is the normal color of the nerve? slightly orange, reddish.
♦ The ON has a cup (depression) and neuro-retinal rim (remaining part).
♦ Can you draw the margins of the nerve with a pencil?
- Yes → normal.
- No → abnormal (indicates swelling).
♦ What do we call a bilateral optic nerve swelling? bilateral disc edema (NOT always papilledema).
- Papilledema by definition is a bilateral optic nerve head swelling because of high ICP.
• It is an emergency because high ICP might be secondary to a tumor & it requires an immediate imaging.
• If the imaging is normal, what might be the cause?
- Fever → (infection, meningitis).
- Headache and usually obese women on OCP → benign idiopathic intracranial HTN (pseudotumor cerebri), very common in our community.
♦ What are the causes of optic nerve atrophy?
- Post-optic neuritis.
- Post-increased ICP.
- Tumor.
- Inflammation.
- Compression.
♦ Thus, optic nerve atrophy requires imaging.</td>
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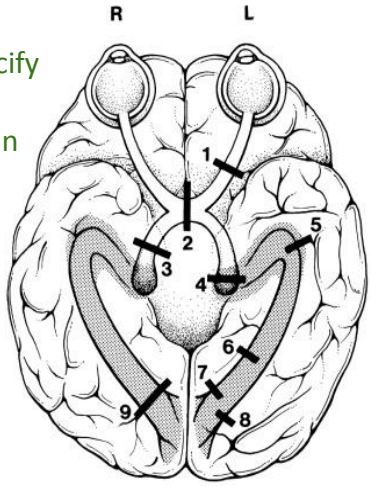
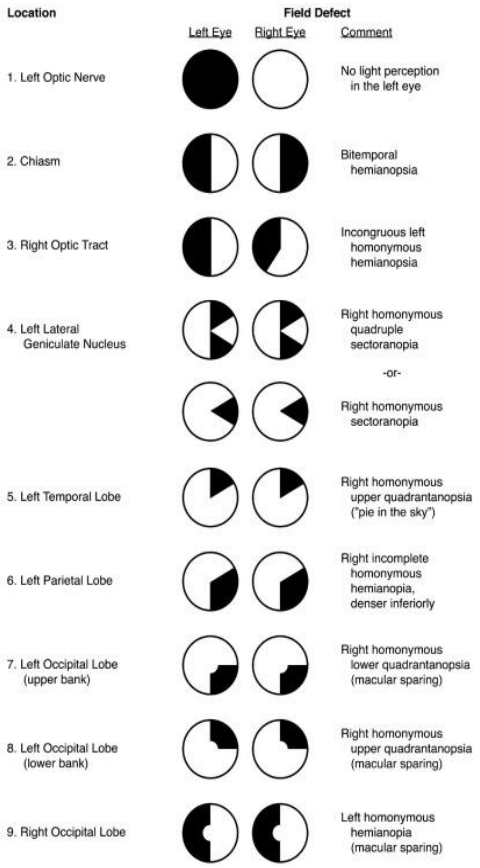
Visual pathway disorder

- **Very common question, name the defect and the location of the lesion**
- Terms:
  - Anopia/anopsia: complete visual field defect, absence of vision.
  - Hemianopia: when half of the visual field is affected.
  - Quadrantanopia/Quadranopsia: quarter of the field of vision.
    - In quadranopsia, we have to specify superior or inferior.
  - If it is more than quarter but less than half → we still call it quadrantanopia/quadranopsia.
  - Macular sparing: mouth eaten part.





- If I ask you to name the visual field defect:
  - Just look at the printout and localize black part (right or left).
  - Decide if both defects are homonymous (means both are on the same side) or not.
  - Then name the visual field defect (don't forget to specify if it's superior or inferior in case of quadranopsia).

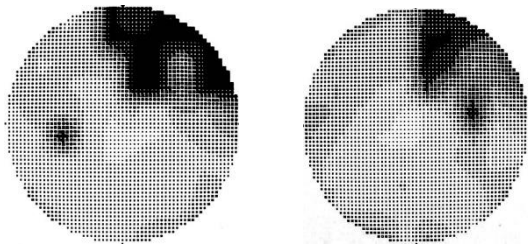
- Localization of visual field defect **(the numbers correspond to the figure above):**
  1. Optic nerve or eyeball → complete anopia.
  2. Chiasm (near pituitary gland):
     - Any mass (e.g. parasellar tumors, pituitary macroadenoma, lactenoma or acromegaly) will press against the chiasm (**binasal** decussating fibers) resulting in what is called **bitemporal** heteronymous hemianopia (we can't say right or left here).
  3. Optic tract:
     - The side of the lesion **should be opposite** to the side of the affected visual field.
     - Hemianopia visual field defect always indicates a lesion in the optic tract.
     - Therefore, in left homonymous hemianopia
       - The lesion is in the right side.
       - Since causing hemianopia it will be in the optic tract.

     → (right optic tract lesion)
  5. Temporal lobe:
     - Right homonymous superior quadrantanopsia
       - The lesion is in the left.
       - Since the defect is superior, so in the brain should be inferior (in the temporal lobe).
       - We call it **pie in the sky.**

     → (left temporal lobe lesion)

6. Parietal lobe:
   - Right homonymous inferior quadrantanopsia
     - The lesion is in the left.
     - Since the defect is inferior, so the lesion should be superior (in the parietal lobe). → (left parietal lobe lesion)
7. Occipital lobe:
   - Occipital lobe lesions give congruous visual field defect (identical visual field defect).
   - Why there is a macular sparing? because the occipital lobe has a dual blood supply: the middle cerebral artery (MCA) and the posterior cerebral artery. MCA accounts for macular fibers.
     - Macular sparing means MCA is okay.
     - If the defect is more peripheral, it indicates posterior cerebral artery involvement.

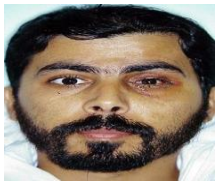
After temporal lobectomy



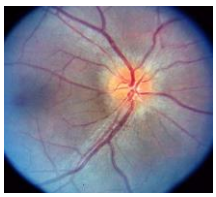
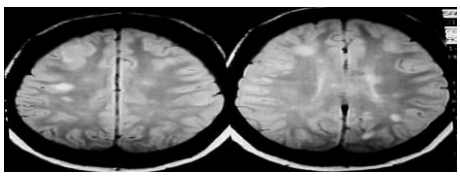
- Type of visual field defect: right homonymous superior quadrantanopsia.
- Lesion: left temporal lobe
- Black dot → normal blind spot, accounts for the optic disc.

Afferent Visual System Diagnoses

- You **do not** need to memorize it.
  - **Compression:**
    - Intraorbital ON.
    - Intracranial ON.
    - Optic chiasm.
    - Optic tract.
    - Posterior afferent system.
  - **Trauma:**
    - Globe by fire cracks, hand fest, tennis ball or door handle specially in children.
    - Intraorbital ON.
    - Optic canal.
    - Optic chiasm.
    - Occipital lobe.
    - Example
      - 25 years old man had a Firecracker exploded near left eye.
      - NLP OS (no light perception).



- **Inflammation:**
  - Orbital pseudotumor (inflammation inside the orbit).
  - Optic neuritis:
    - Optic nerve inflammation that affects young females associated with decrease in vision & pain on eye movement.
    - Inflammatory demyelinating condition associated with multiple sclerosis (not always associated with MS)!
    - The most common type in young adults (most commonly females).
    - The clinical presentation of a typical optic neuritis: sudden visual loss, pain with eye movement (inflamed optic nerve which is in close proximity with medial rectus muscle) and visual field loss (commonly central but can present with any VFD).
      - If they look horizontally, they will feel minimal pain (discomfort); If pain is severe, this is (atypical optic neuritis).
    - 20% of MS patients have optic neuritis as a first manifestation.
    - Patients with optic neuritis need an MRI of the brain and orbits to look for enhancing lesions for risk of MS.
      - If MRI is normal, risk of MS within the next 10-15 years will drop to 25%. It won't eliminate it but at least if will make it less!
    - IV steroids my speed up the recovery process but does not influence the outcome
      - It is important to let the patients know that we give them steroids not to improve the visual outcome.
      - Patients who take or do not take steroids will eventually improve if it is typical optic neuritis.
      - Oral is contraindicated because of high recurrence rates.
      - Good recovery.

Optic neuritis (common)
• 27 years old women developed blurred vision OD and mild right periorbital pain especially with eye movement. • VA 20/50. • Fundus: slightly hyperemic disc. • MRI abnormal: showing multiple white plaques, comes with MS. • Diagnosis: multiple sclerosis.    

- **Ischemic optic neuropathy:** "disease of the elderly"
  - Non-arteritic ischemic optic neuropathy.
  - Giant cell arteritis with ischemic optic neuropathy.
  - Central retinal artery occlusion.
  - Other retinal emboli.

<table>
<tr><td>Non-arteritic ION</td><td>

Patients often have DM, HTN and other vascular risk factor.
Most common cause in older patients (above 40 years).
Altitudinal visual field loss (superior or inferior).
Treatment: no treatment, ask them to control the risk factors to protect the other eye.

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<tr><td rowspan="4">Arteritis ION</td><td>

(> 55) years old (older than non-arteritic ION).
Associated with giant cell arteritis.
Present with severe irreversible visual loss (counting finger) more than non-arteritic (20/200).
Check: jaw claudication, proximal myalgia & arthralgia, scalp tenderness/pulseless, headache.
Elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP).
Temporal artery biopsy is the gold standard for diagnosis.
Treatment: systemic steroids, given immediately if suspected even before the biopsy, patient should not leave the ER without it to protect the other eye (within the first day they will go blind) .
Binocular involvement occurs in third of cases, often within the first day.

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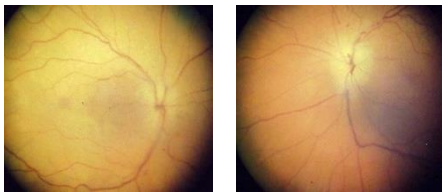
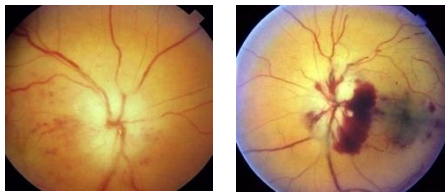
<tr><td>Giant Cell Arteritis, day 2</td></tr>
<tr><td>  </td></tr>
<tr><td>Giant Cell Arteritis, day 4</td></tr>
<tr><td>  </td></tr>
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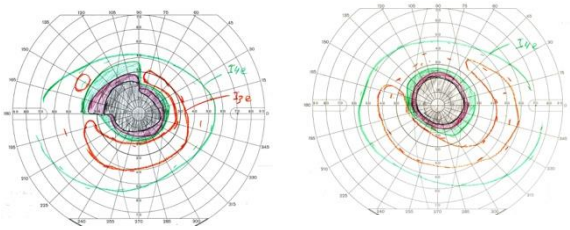
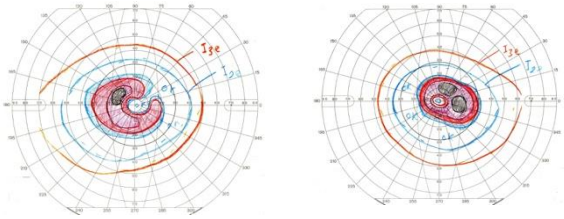
- **Congenital and genetic problems:**
  - Congenital retinal dystrophies.
  - Optic nerve hypoplasia.
  - Leber hereditary optic neuropathy.
  - Dominant and recessive optic atrophy.
  - Glaucoma.

- 14 years old girl, vision OS began to decline gradually without pain.
- First visit with VA OD 20/40 and OS CF at 3'
- Pale, flat optic discs OU
- VA 1-month later CF at 3' OU
- VA 10 months later 20/20 OU
- Diagnosis LHON-like optic neuropathy.

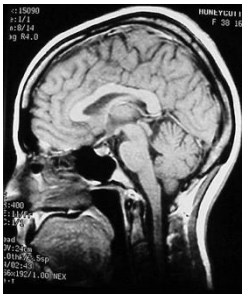
VF 1 month later	VF 10 months later
	
The goldmann visual field test Black area indicates the visual field defect (central scotoma).	

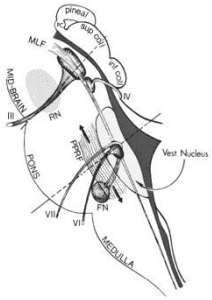
Afferent visual system tests

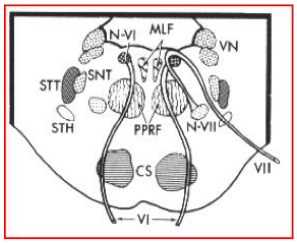
- During exam:
  - Visual field test.
- Neuroimaging:
  - CT scan
  - MRI scan.
  - ERG/VEP
- Blood test:
  - Vasculitis (ESR, CBC, ANA, VDRL).
  - LFT (SGOT[1], SGPT[2], Alkaline phosphatase).
- Urine analysis
- Creatinine, BUN
- Ultrasound:
  - Carotid doppler.
  - A and B scans (ultrasound of eyeball). If a patient has cataract, you can't see the nerve, so use US.
- Genetic evaluation.
- Electrolytes.

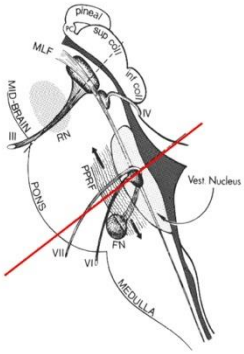
[1] Serum glutamic oxaloacetic transaminase
[2] Serum glutamic pyruvic transaminase

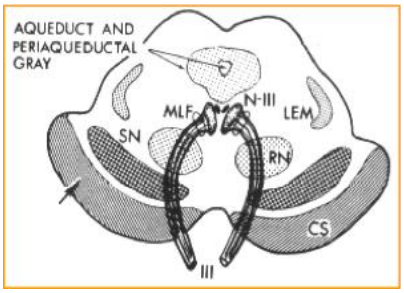
Efferent Neuro-anatomy

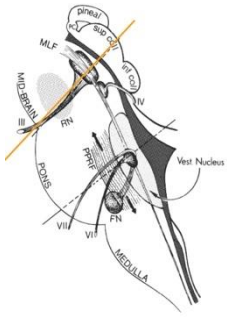






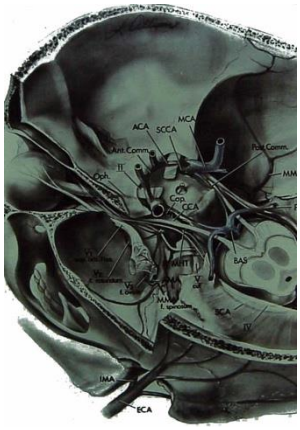


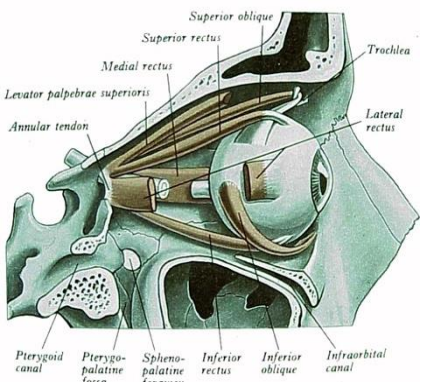




- Remember we said that the efferent visual system consists of pupil size, eyelid position and ocular motility.
- The ocular motility is controlled by cerebellum, brainstem, interneuronal nuclei as well as the peripheral cranial nerves.
- The cranial nerves controlling the ocular motility are 3 nerves which are 3rd, 4th & 6th cranial nerves.
- All cranial nerves originate from brainstem (the midbrain and pons), then goes anteriorly to the orbit to control ocular motility.
- What is the interneuron connection?
    - If I ask you to look to left, 6th and 3rd will move together, why? because of the medial longitudinal fasciculus (in midbrain).
    - If I ask you to look at something, why you look at it and not look beyond it? this is controlled by the cerebellum and the interneuron nuclei.
- There are two types of eye movement:
    1- Fast movement: shifting from one target to other.
    2- Slow movement: tracking a moving object.

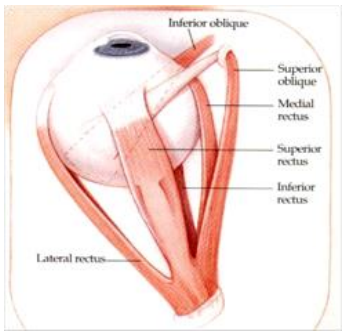
Efferent skull anatomy





The Extraocular Muscles

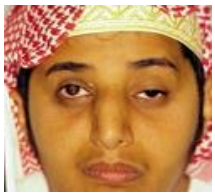
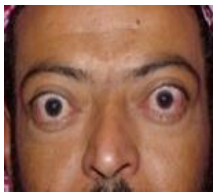
- Four recti & two oblique muscles.
- All are supplied by oculomotor nerve except, superior oblique (trochlear nerve) & lateral rectus (abducent nerve).



- Anatomy & physiology:
- The main nerve supplying extraocular muscles is oculomotor (third nerve)

EOM	Primary action	Innervation	Nucleus
Superior rectus	Elevation (maximal on lateral gaze)	Third cranial nerve, oculomotor	**Midbrain**
Inferior rectus	Depression (maximal on lateral gaze)	Third cranial nerve, oculomotor	
Medial rectus	Adduction	Third cranial nerve, oculomotor	
Inferior oblique	Excyclotorsion	Third cranial nerve, oculomotor	
Superior oblique	Incyclotorsion	Fourth cranial nerve, trochlear	
Lateral rectus	Abduction	Sixth cranial nerve, abducens	**Pons**

Efferent Examination

Just look at the patient? inspection	
▪ Are eyes straight? ▪ No, the left eye is not straight, deviated towards the nose (**esotropia**).	
▪ What are the lid positions? ▪ Left ptosis, the patient is lifting his eyebrow to compensate.	
▪ Are the eyes proptotic? ▪ Exophthalmos. ▪ Injected eyes. ▪ Scleral show, normally it is not seen.	
▪ Are there any spontaneous eye movements? ▪ Nystagmus (involuntary **rhythmic** eye movement).	

<table>
<tr><th colspan="2">Movements of both eyes in all directions</th></tr>
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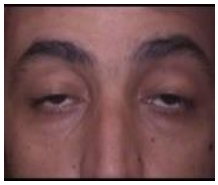
- Have the patient move eyes in all directions, not just the direction where you think there is a problem.
- In the shape of X & + **(NOT H)**.

</td><td>  </td></tr>
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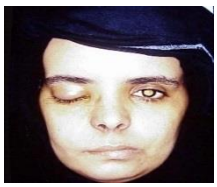
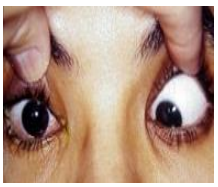
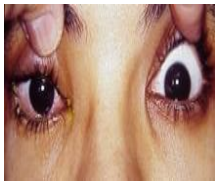
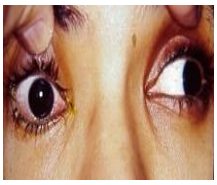
- Hold lids if necessary (only after looking first without holding lids).

</td><td> 



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- Examine each eye separately if **necessary**.
- **Ocular motility is always done binocular,** except in some cases.

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<tr><th>Smooth pursuit</th><th>Saccades</th></tr>
<tr><td>

- The reflex that helps to maintain fixation on an object in motion in the visual world while the head is stable.
- Also, the reflex that inhibits the vestibluo-ocular reflex.
- Slow eye movement.

</td><td>

- The reflex that permits a rapid refixation from one point in the visual field to another.
- Fast eye movement.
- Examined by asking the patient to look at a pen & a finger (vertically and horizontally).
- It tests two things:

Velocity:

- Both same velocity → normal.
- Slow velocity in one eye→ may be myasthenia gravis.

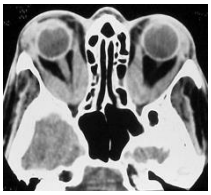
Accuracy to target:

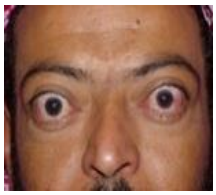
- No accuracy to target → cerebellar disease.

</td></tr>
</table>

Efferent diagnoses

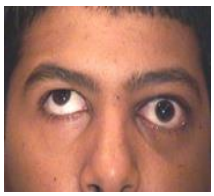
- **Orbit:**
  - ♦ Extraocular muscles:
    - In the CT, the muscles are enlarged, hypertrophied.
    - This is thyroid orbitopathy; The muscles can compress on the nerve and cause loss of vision.

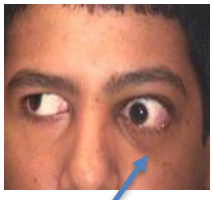


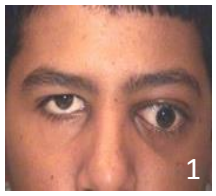


Bilateral proptosis

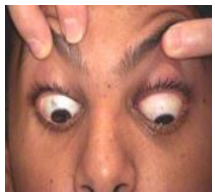
  - ♦ Trauma:
    - 14-years old boy
    - In picture 1:
      - There is malalignment.
      - Dropping of left eye (abnormal eyeball position which is called ocular dystopia).
      - If we draw a line from the lateral canthus to the lateral canthus, they **are not** in the same position; There is sagging.
    - When ask the patient to:
      - Look left, he could.
      - Look right, he could with some limitation.
      - Look down, he could.
      - Look up, he could not.
    - Very common with **orbital floor fracture**.
    - This patient had a trauma by fest, so the orbital floor got fractured.
    - Inferior rectus is entrapped in the bone; When the patient looks up, the muscle cannot relax, but looking down, it can contract.

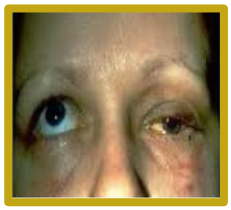






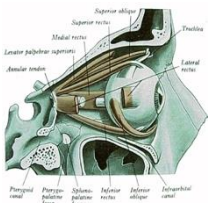
The left rim is not similar to the right rim because there is fracture.





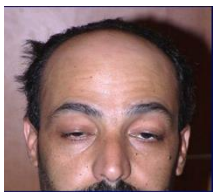
Diagnosis: blowout fracture of the left eye.
Modality for investigation: CT scan

  - ♦ Mass:



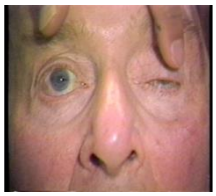
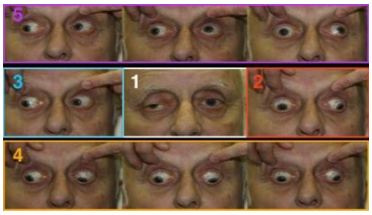
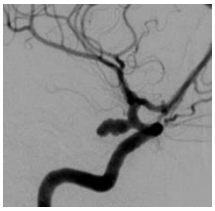
- **Neuromuscular junction:**
  - Ocular myasthenia gravis:
    - Myasthenia gravis could be an ocular condition affecting the ocular motility of the eye only without systemic manifestations.
    - Definition: chronic autoimmune disease affecting the neuromuscular junction.
    - Clinical presentation:
      - **Symptoms:**
        - **PAINLESS** ptosis (bilateral).
        - Diplopia (can present alone or together).
        - **Fatigability and variability** are characteristic (usually worse at the end of the day) unlike other palsies which are constant.
        - Ask the patient if symptoms are worse early in the morning or at the end of the day?).
        - Ask about systemic weakness, difficulty in swallowing or breathing.
        - Majority present with ocular MG then eventually convert to general MG.

      - **Examination:**
        - **Pupil is not affected.**
        - **Assess orbicularis strength (Ask patient to close eyes strongly & open them)**
    - If painful ptosis or there is pupil involvement don't say myasthenia gravis with your differential diagnosis.
      - **Investigations:**
        - Blood test for acetylcholine receptor antibodies. 50% present in OMG.
        - Tensilon test: inhibits acetylcholinesterase and can transiently reverse signs of weakness due to OMG, such as ptosis and extraocular muscle paresis. Look for improvement in symptoms.
    - Diagnostic but we don't have it here.
      - **Treatment: you don't need to know the details, but we can give steroids or Acetylcholinesterase inhibitors.**

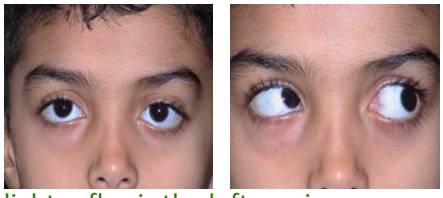
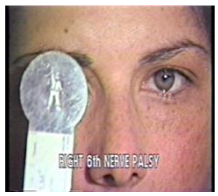


Bilateral ptosis compensated by lifting the eyebrows (absence of forehead wrinkles indicates loss of frontalis muscle tone that could be secondary to myasthenia gravis).

- **Single cranial nerves:**

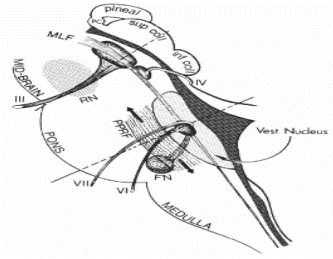
Third (oculomotor) nerve palsy	
Case	
 This is a video played by the doctor.	▪ Left eye: - Ptosis. - Loss of adduction, infraduction & supraduction (3rd nerve affected). - The abduction is intact. ▪ 65 years old presented to ER complaining of double vision (typical presentation of CN III palsy always keeps it in your head!). - Ptosis and Eyes are (down & out). - Pupillary dilatation & no accommodation. - The eye rests in a position of abduction, slight depression, and intorsion.
 	▪ Examination of extraocular movement: 1- Primary position: looking straight ahead. 2- Looking to his left: abnormal. 3- Looking to his right: normal. 4- Mild infraduction limitation. 5- Mild **spraduction** limitation. ▪ **Check for pupillary involvement:** - To differentiate between **surgical** (urgent, compression, pupil involved, needs neuroimaging) and **medical** (pupil sparing) third nerve palsy. - Absence of pupillary involvement suggests a benign process that can be observed over a couple of weeks. A fixed, dilated pupil requires extensive neurologic evaluation.  Diagnosis: 3rd nerve palsy Clinical manifestation: ptosis, failure of adduction, elevation & depression of the eye. Causes: DM (commonest); surgical
	▪ Diagnosis: posterior communicating artery aneurysm (life threatening). - Right internal carotid artery injection reveals a right posterior communicating artery aneurysm with a tubular configuration. A very small anterior communicating artery aneurysm is also identified. - Magnetic resonance angiography (MRA) is the best investigation for PCA aneurysm. **You always have to rule out aneurysm, why?** Usually parasympathetic fibers go the outside (superficial) with CN III pathway, so any compression will lead to CN III palsy with pupil involvement.
▪ **Pupils**: innervated by parasympathetic fibers which is not part of oculomotor nerve, they run together and have the same pathway. The parasympathetic pupil-constrictor fibers from Edinger-Westphal nucleus travel within CNIII, and their loss gives you a "blown pupil".	

Etiology
▪ Microvascular ischemia (DM, HTN, DLP). If risk factors are controlled, the palsy will resolve by itself. ▪ Intracranial aneurysm (posterior communicating artery) (not the most common but to ophthalmologists). ▪ Trauma (neuroimaging is indicated). ▪ Brain tumor (neuroimaging is indicated).

Fourth (trochlear) nerve palsy	
Clinical presentation	▪ It is usually difficult to be diagnosed by non-ophthalmologist. ▪ Patient will complain of vertical double vision only. ▪ **Vertical diplopia** (**characteristic** unlike CN III palsy which can have different types). ▪ They mostly complain about actions that require downgaze vision; like: going down the stairs, eating, reading and writing. ▪ **Head tilt** to the opposite shoulder. - You might think the kid is shy and he doesn't want to interact, but actually he is just trying to avoid his double vision. - If you try to correct his head, you'll notice some **hypertropia** (a condition of misalignment of the eyes (strabismus). - If you move his head to the same side of the affected nerve it will be worse.  The light reflex is the left eye is lower than the right eye which means the right eye is shifted up.
Etiology:	▪ Congenital (commonest). ▪ Trauma even minor ones not only severe. ▪ Idiopathic.
Sixth (abducent) nerve palsy	
Clinical presentation	▪ If someone has 6th nerve palsy, the lost movement is abduction (lateral rectus muscle movement). ▪ If a patient has loss of lateral rectus in the right eye, the patient will have esotropia (medical recuts pulls over). If we cover the left eye, the patient will focus on the right eye (deviated eye), then it will go back straight. ▪ Horizontal diplopia → two images beside each other (worse at distance).  - لما امسك خط، لما اسوق السيارة او أكون مسافر تكون worse ليه؟ - Because with near vision you need convergence you don't need CN VI, but "when looking far you need it for divergence (taking the eye out). ▪ Esotropia - When you do **cover-uncover test**, esotropia is more at distance than near. - Face turns in the direction of the paralyzed muscle to avoid its action.

	- Limited Abduction on the side of the lesion. **(1)** Primary position **(2)** Looking to his right, right lateral rectus is affected. **Diagnosis: right** sixth nerve palsy.
Etiology:	▪ You have to rule out: - Microvascular diseases (most commonly) - Intracranial tumors. - Trauma. - Increased intracranial pressure (we call it false localizing sign because you don't know exactly where the lesion is. • The nerve passes through the Dorello canal at 90-degree angulation, this makes it susceptible to pressure due to any lesion in the brain. ex. a frontal tumor will cause pressure on this canal leading to 6th nerve palsy.

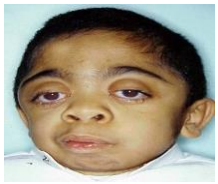
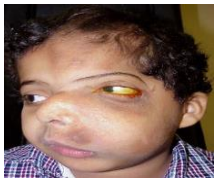
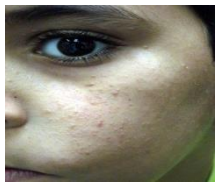
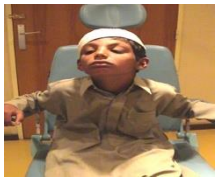
- **Multiple cranial nerves.**



- **Intraparenchymal problem:**

Internuclear ophthalmoplegia	Gaze palsy
▪ If you ask the patient to look right or left, the lateral rectus should abduct, and medial rectus should adduct; What connect this movement together? MLF (medial longitudinal fasciculus), it is an interneuron between 3rd and 6th. ▪ This interneuron connection can be injured by MS (most common cause in young patients), stroke (most common cause in old patients) or brainstem mass. ▪ What will happen if you have injured interneuron connection? The adducting eye cannot adduct, and the abducting eye will have an abducting nystagmus.	▪ The ocular complication of facial palsy is paralysis of orbicularis muscle → patient can't close the eye resulting in dryness. ▪ If you leave the dryness → abrasion → melting → perforation and eye fluid will come out. ▪ So, the cornea will perforate if the patient can't blink → iris will come out. ▪ Ointment is important, and during sleep, patient must tape the eye.  Right facial palsy  Diagnosis: right facial nerve paralysis Ocular complications: keratoconjunctivitis, exposure keratitis.

Other ……

Unusual faces	Unusual skin lesions	Unusual postures
 	 ▪ This patient has a benign tumor of the skin. It is called adenoma sebaceum, indicating a disease called tubular sclerosis. ▪ If we see it, we have to scan with MRI for brain tumor. Those patients are prone to develop gliomas.	 ▪ The patient is trying to compensate for ptosis. ▪ He could develop vertebral degeneration.

Questions:

1. **Sympathetic pathway mediated through ___**
   A. 3rd order neuron.
   B. 2nd order neuron.
   C. 4th order neuron.
   D. 5th order neuron.

2. **Horner syndrome leads to all but:**
   A. Ptosis.
   B. Miosis.
   C. Mitosis.
   D. Anhidrosis.

3. **In a patient with ptosis and diplopia, the following findings is suggestive of myasthenia gravis:**
   A. Symmetrical involvement.
   B. Increased serum creatinine phosphokinase.
   C. Improved lid movement after applying ice cube to the lid.
   D. Absent tendon reflexes.

2. **The following are true about isolated fourth (trochlear nerve) palsy:**
   A. Head trauma is the most common cause in children.
   B. Aneurysm is a common cause in adult.
   C. The head is usually tilted away from the palsied side.
   D. Vertical prism is useful in correcting the torsional diplopia.

3. **Bitemporal hemianopia may be seen in all but:**
   A. Tilted disc.
   B. Bilateral ischemic optic neuropathy.
   C. Dermatochalasia.
   D. Sectorial retinitis pigmentosa.

4. **The following findings make benign intracranial hypertension unlikely:**
   A. Bilateral sixth nerve palsy.
   B. morning headache and nausea.
   C. increased protein in the cerebrospinal fluid.
   D. hard exudate in the macula.

5. **In non-arteritic ischaemic optic neuropathy:**
   A. Embolism is the cause in the majority of cases.
   B. A small cup-to-disc ratio is a risk factor.
   C. Visual loss is usually more severe than arteritic ischaemic optic neuropathy.
   D. Optic nerve fenestration is useful in improving final visual outcome.

Answers: 1:A ,2:C , 3:C , 4:C , 5:B , 6:C , 7:B , 8: B

Good luck!