433 Teams

OPHTHALMOLOGY

Neuro-Ophthalmology

Color index:

432 Team – **Important** – **433 Notes** – Not important

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Neuro-ophthalmology deals with visual problems caused by disorders of the brain or the optic nerve connection.

Our eyes simply receive visual information - we actually see with our brain. In turn, the brain controls the position and focus of the eyes, directing our visual attention.

Part 1: Pupillary Disorders

Anatomy and physiology:

Pupils:

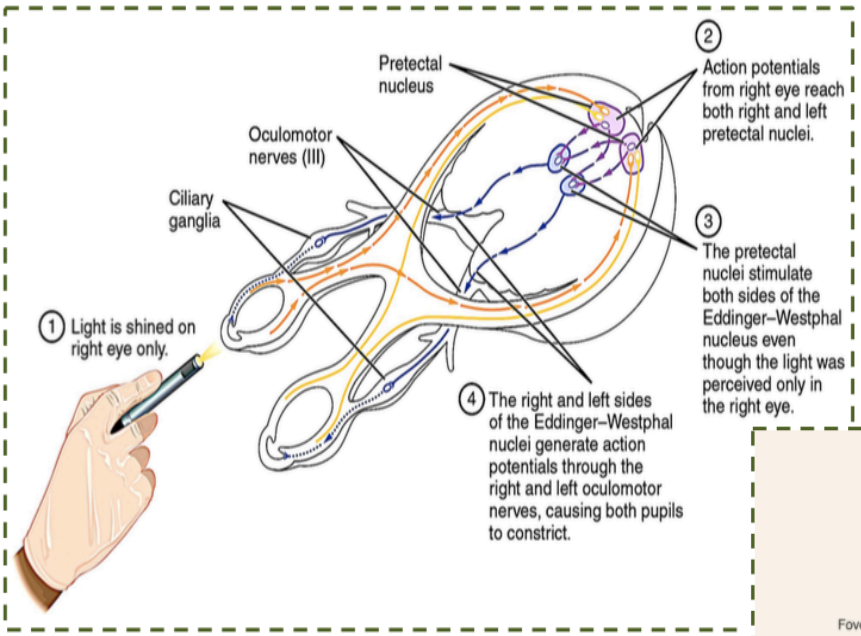
pupillary control: The physiology behind a "normal" pupillary constriction is a balance between the sympathetic and parasympathetic nervous systems.

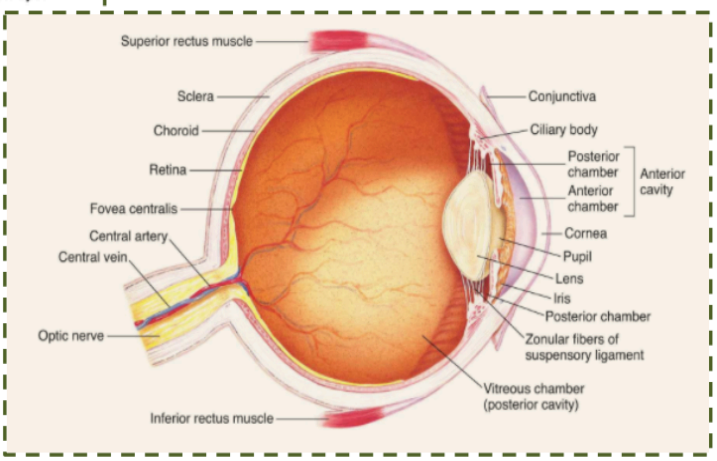
1**Parasympathetic** innervation leads to **pupillary constriction**.

1. Originate from **Pretectal nucleus at midbrain and stimulate both Eddinger-westphal nucleus**
2. **Divided into superior and inferior division**
3. **inferior division** go to **ciliary ganglia** (parasympathetic ganglia) and finally reach to muscle

Sphincter pupillae muscle: Supplied by **parasympathetic fibers** of **Oculomotor nerve** and lead to **constriction of pupil**

Pupil constrict to light and near stimuli.





2\Sympathetic innervation leads to pupillary dilation.

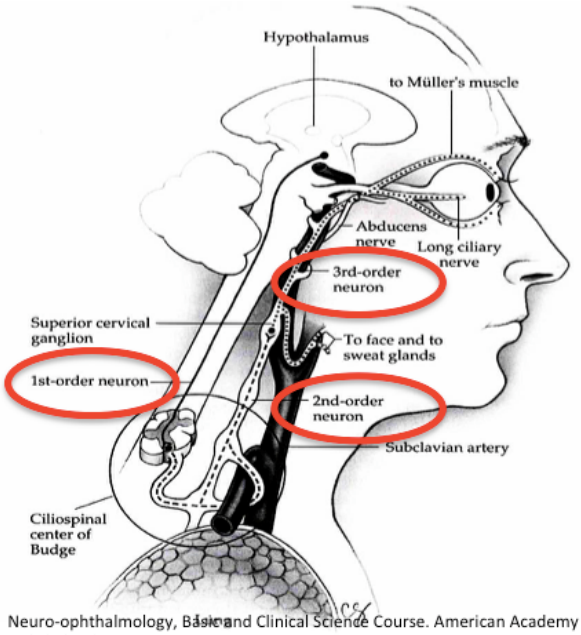
- Originate from **hypothalamus and go through superior cervical ganglia**

Dilator pupillae muscle: Supplied by **Sympathetic fibers** and lead to **Dilation of pupil,** a group of muscles in the peripheral 2/3 of the iris.

- If there is a cut through sympathetic pathway patient will develop signs of **Horner syndrome.**

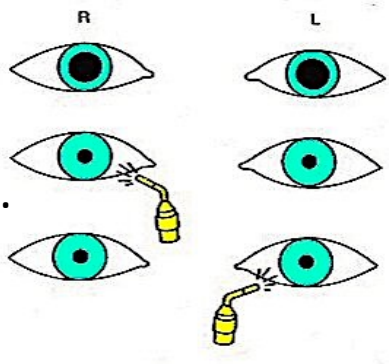
The first-order neuron descends from the hypothalamus to the first synapse, which is located in the cervical spinal cord (levels C8-T2, also called **ciliospinal nucleus of Budge**).

- The second-order neuron travels through the brachial plexus, over the lung apex (some tumor in the lung may damage the sympathetic pathway of the pupil) . It then ascends to the superior cervical ganglion located near the angle of the mandible and the bifurcation of the common carotid artery.
- The third-order neuron then ascends within the adventitia of the internal carotid artery, through the cavernous sinus, where it is in close relation to the sixth cranial nerve [1,3]. The oculosympathetic pathway then joins the ophthalmic (V1) division of the fifth cranial nerve (trigeminal nerve). In the orbit and the eye, the oculosympathetic fibers innervate the iris dilator muscle as well as **Müller's muscle**, a small smooth muscle in the eyelids responsible for a minor portion of the **upper lid elevation (2-3 mm)** and lower lid retraction. (The main eyelid muscle elevator is Levator palpebrae, supplied by 3rd nerve)



Neuro-ophthalmology, Basic and Clinical Science Course. American Academy of Ophthalmology, 2007

Examination of the pupil:



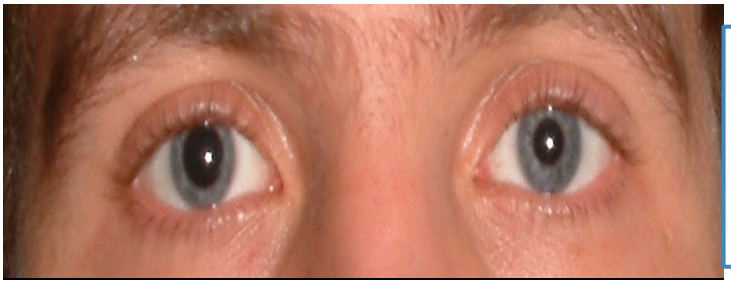
1- Best conducted in **dim light room** using a bright light
2- The patient should be relaxed and **fixing on a distant object** (to get red of accommodation because the accommodation cause miosis) .
3- The size, shape and position of each pupil should be noted in light and dark condition.
4- Check light reflex:

- **Direct pupil reflex:** When focus the light on one eye, **that eye** will constrict
- **Consensual pupil reflex:** When you focus the light in one eye, **the other eye** will constrict

5- Looking for a **relative afferent pupillary defect (RAPD)**

Do swinging light reflex (Marcus gunn reflex), both eyes should be **always constrict** when you focus the light **if Dilated** when you focus the light, this is **+RAPD and means there optic nerve damage.**

http://www.neuroexam.com/neuroexam/content.php?p=19



20% of the population has Physiologic Anisocoria.

Criteria of Physiologic Anisocoria:

1)Less 1 mm deferent sizes.
2) Same amount in the dark and light.
you don't need to investigate them.

How to know which one is abnormal? Look to the corneal light reflex (U should evaluate the patient in dim and light).

When the small pupil does not dilate as well as the large pupil in dim light, then the small pupil is abnormal.When the larger pupil does not constrict as well as the small pupil in response to a light stimulus, then the large pupil is abnormal. (This condition called **Anisocoria (unequal pupil size**)

Causes of Dilation of pupil:

- Previous ocular surgery
- Ocular trauma
- Use of medication like cycloplegics e.g. **atropine, cyclopentolate**
- **Third** nerve palsy **(mid dilated fixed pupil, not respond to light)**
- **Tonic pupil (Adie's pup**

Tonic pupil (Adie's pupil)*:

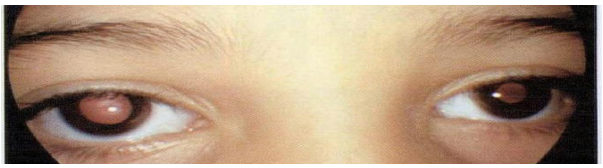
✓ Benign condition
✓ Young female , subacute onset.
✓ 80% **unilateral** dilation of pupil.
✓ It is due to *ciliary ganglionitis* which denervates the parasympathetic supply to the iris and ciliary body.
✓ **Physical Examination:**

• Sluggish, segmental pupillary responses to **light**

• Normal response to near followed by slow redilation. **called light near dissociation.**

• Instillation of weak cholinergic agents **(0. 1% pilocarpine)** will cause **constriction of the tonic pupi**l (denervation hypersensitivity**) the normal eye won't change**

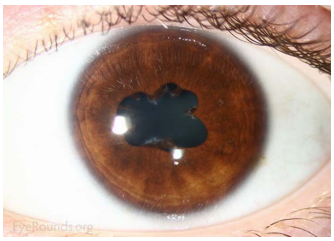
✓ Holmes-Adie syndrome:



- Includes tonic pupil, diminished deep tendon reflexes and orthostatic hypotension.

*The pupil will constrict with near vision, but very slowly. That's why we call it a "tonic pupil" - it's tonically slow. the parasympathetic pathway is much shorter than the convoluted sympathetic pathway, so potential causes for damage are more benign. The parasympathetic plexus sits right behind the eye and can be damaged after an otherwise benign viral infection. **It is a diagnosis of exclusion. Patient most of the time will be asymptomatic. However they might compline of photophobia because of dilated pupil. Sometimes they will have abnormality of accommodation in near vision. We can provide them pilocarpine will release the photophobia and help them with the accommodation It takes few months and the pupil will go back and constrict**

Causes of Constriction of pupil:



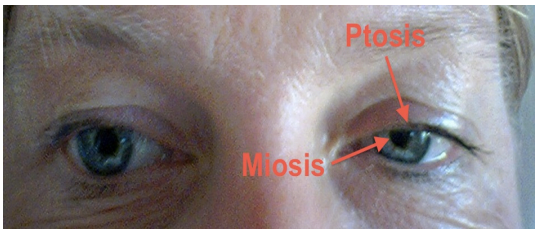
- Previous ocular surgery
- Ocular trauma or inflammation

The margin oh the pupil (iris) is attached to the lens (posterior synechia) or to the cornea (anterior synechia) (adhesion)

- Use of medication e.g. **pilocarpine**
- **Horner syndrome.**

✓ Horner syndrome:

- **Cause:** interruption of sympathetic pathway **(Carotid dissection, carotid aneurysm and tumor)!!!**
- **Signs:** at the side that affected you will see **miosis – anhydrosis – ptosis – enophthalmous**. In general no other symptoms
- **Miosis** : due to loss of dilator function
- **Anhydrosis**: lack of sweating
- **Enophthalmous** (posterior displacement of the eyeball): due to paralysis of levator palpebrae muscle.
- **Ptosis:** due to paralysis of muller's muscle



Do we need to image the patient urgently or give him the next available appointment?

1) Acute or chronic :

Acute within 2 weeks: immediate neuroimaging.

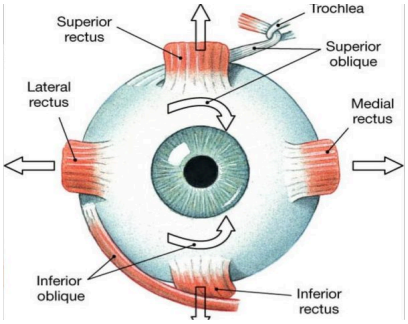
Chronic within several months or he has a surgery: follow up.

2) Painful or painless:

Painful: immediate neuroimaging. (Sometime carotid dissection presents with painful Horner syndrome)

Part 2: Neuromotility disorders

Extraocular Muscles: There are **six voluntary muscles** that run from the posterior wall of the orbital cavity to the eyeball. These are:

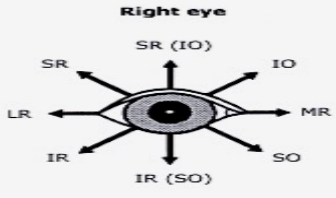


- **4 recti muscles:**

1. **Superior rectus** acts as the primary **elevator**
2. **Inferior rectus** acts as the primary **depressor** of the eye.
3. **Medial rectus** muscle is the primary **adductor** of the eye
4. **Lateral rectus** muscle is the primary **abductor** of the eye.

- **Medial and lateral rectus** muscles have only **horizontal actions.**
- Superior and inferior rectus muscles are the primary **vertical** movers of the eye.
- **2 oblique muscles: Superior and Inferior oblique muscles.**
- This vertical action is greatest with the eye in the abducted position.
- The **secondary action** of vertical rectus muscles is **torsion**. The **superior rectus** is an **incyclotorter** (inwards rotator), and the **inferior rectus** is an **excyclotorter** (outwards rotator). The tertiary action of both muscles is adduction.
  - **4th Trochlear nerve:** supplies **the superior oblique muscle**.
  - **6th Abducent nerve:** supplies **the lateral rectus muscle**.
  - **3rd Oculomotor nerve:** begins as a nucleus in the midbrain that consists of several subneclei that innervates the individual **extraocular muscles, the eyelids, and the pupils.** It supplies the superior**, inferior and medial rectus muscles and the inferior oblique muscle.**

Innervation of extraocular muscles	Primary action
Cranial nerve III	
Superior rectus	Elevation (maximal on lateral gaze)
Inferior rectus	Depression (maximal on lateral gaze)
Medial rectus	Adduction
Inferior oblique	Excyclotorsion Away from the nose
Cranial nerve IV	
Superior oblique	Incyclotorsion Toward the nose
Cranial nerve VI	
Lateral rectus	Abduction



Oculomotor and trochlear nerves exit at the of midbrain, while Abducent from pons

Yoke muscles are the primary muscles in each eye that accomplish a given version (eg, for right gaze, the right lateral rectus and left medial rectus muscles). Each extraocular muscle has a yoke muscle in the opposite eye to accomplish versions into each gaze position

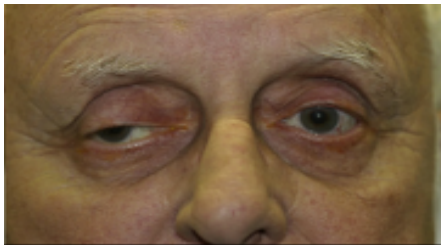
Muscle*	Primary	Secondary	Tertiary
Medial rectus	Adduction	—	—
Lateral rectus	Abduction	—	—
Inferior rectus	Depression	Excycloduction	Adduction
Superior rectus	Elevation	Incycloduction	Adduction
Inferior oblique	Excycloduction	Elevation	Abduction
Superior oblique	Incycloduction	Depression	Abduction

*The superior muscles are incycloductors; the inferior muscles, excycloductors. The vertical rectus muscles are adductors; the oblique muscles, abductors.

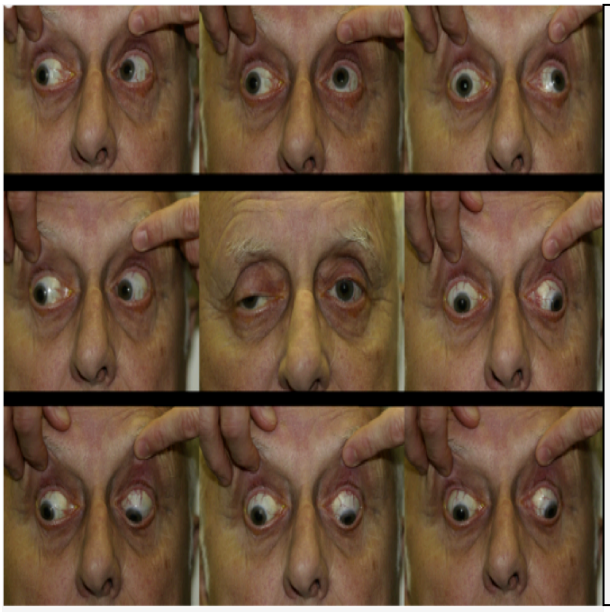
Neuromotility Disorders:

1- 3rd Cranial Nerve "Oculomotor" Palsy:

- **65 yrs old presented to ER complaining of double vision.**
- The patient presentation: the eye is deviated down and out, ptosis, pupillary dilatation and paralysis of accommodation.



Physical Examination:



Neurosurg Clin N Am 23 (2012) 607–619

- ✓ **This patient have right 3rd nerve palsy. How did we know?**

He can **abduct his right eye** only, which is **lateral rectus muscle function**

- ✓ **If you want to rule out 4th cranial nerve palsy along 3rd nerve palsy what will you do?**

Ask the patient to look **down**, if the eye **intorted** the **4th cranial nerve is intact**

- ✓ **Check for pupil involvement ?**

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.
What is the best investigation for PCA aneurysm?
Magnetic resonance angiography

Third cranial nerve palsy is caused by a lesion of the oculomotor nucleus within the midbrain or by compression of the peripheral course of the nerve by aneurysm or tumour. It leads to **drooping of the eyelid "ptosis", dilatation of the pupil that is unresponsive to light and accommodation, and an ability to move the eyeball upwards, downwards or inwards "adduction". Patient will come with horizontal diplopia.**

- ### Etiology of Third cranial nerve (oculomotor)palsy :
  - **Micro-vascular ischemia (DM and HTN)**
  - **Intracranial aneurysm (posterior communicating artery)**
  - **Trauma**
  - **Brain tumor**
- ### Medical 3rd nerve palsy:

Isolate motor part damaged due to **Vascular diseases** such as **diabetic and hypertension.**

- ### Surgical 3rd nerve palsy:

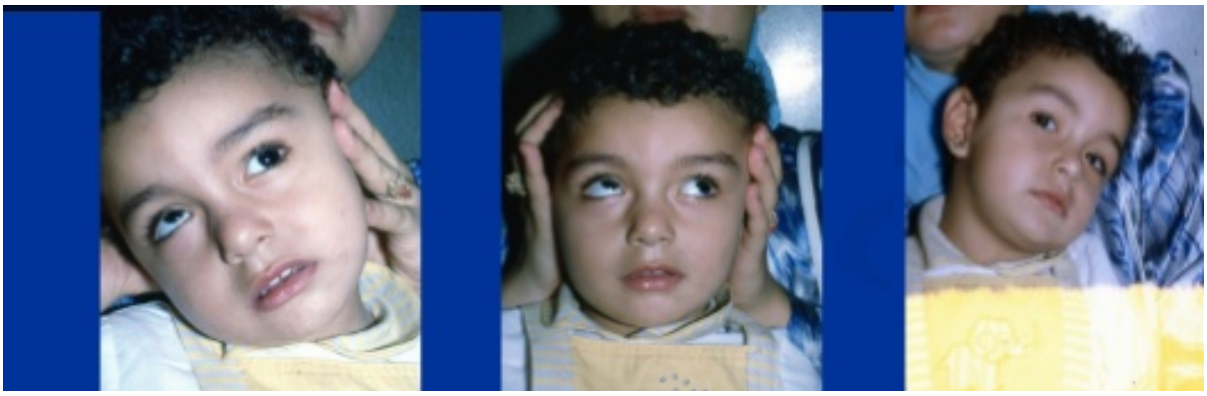
Pressure on pupil constrictor fibers of CN III due to tumor or **Posterior communicating artery aneurysm (most common cause)** lead to **Unilateral dilated pupil.** Compressive lesions usually affect the parasympathetic nerve component: a blown pupil is a potential emergency. Whenever you have pupillary involvement, you need an **MRI and angiography to rule out a dangerous aneurysm or tumour.** (PCA located in circle of Willis)

2-Fourth Cranial Nerve "Trochlear" Palsy:

These patients have an **upward deviation of the affected eye and a "cyclotorsion" twisting of the eye that makes them tilt their head away to the opposite shoulder and vertical diplopia. Also the Patients have difficulty in down gaze.** The fourth cranial nerve is the skinniest nerve and runs the longest distance inside the cranial vault. This long passage makes it more susceptible to injury and neoplasm. The fourth nerve is also susceptible to being pulled from the root where it exits from the back of the brainstem.

Etiology:

- Trauma
- Idiopathic
- Congenital. More fourth palsies occur in elderly males from trauma and more congenital palsies are found in the pediatric population.

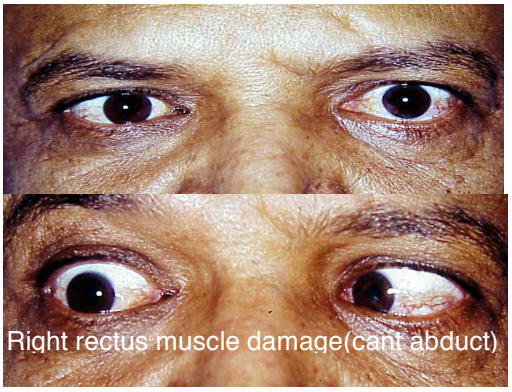


3-Sixth Cranial Nerve "Abducent" Palsy : A sixth cranial nerve palsy is caused by a lesion of the abducens nucleus in the pons or by compression of the peripheral course of the nerve by an aneurysm or tumour. It leads to **inability to move the eye outwards "abduction".** false -ve sign ? 6th due to ^ IOP you won't know exactly where is the lesion so false localizing sign

- ✓ **Horizontal diplopia (worse at distance)** why?

conversion in near you don't need lateral rectus meanwhile in distsnce it will get worse

- ✓ **Esotropia (crossed eye)**
- ✓ **Face turn in the direction of the paralyzed muscle**
- ✓ **Limited Abduction on the side of the lesion**

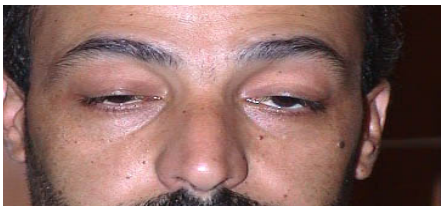


Causes:

- Intracranial tumors
- Trauma **(most common cause because it's long nerve)**
- Microvascular diseases **(mostly DM)**
- Increased intracranial pressure

Part 3: Neuromuscular Disorders

Ocular Myasthenia Gravis (OMG)



- Chronic autoimmune disease affecting the **neuromuscular junction** in skeletal muscles (nicotinic acetylcholine receptors).
- **History:** Patient is not able to stand from his bed at morning after sleeping due to muscle weakness Or he feel fatigue at the end of the day.
  Ask the patient is your double vision or ptosis worse early morning or at the end of the day? Have you notice that the double vision worse at evening?
- **Signs: Ptosis** (due exhaustion of muscle NOT due to paralysis) – **Diplopia** – fatigue – **pupil is normal –** painless condition
- **Investigations:**
  1. **Tensilon test:** inhibits acetylcholinesterase and can transiently reverse signs of weakness due to OMG, such as ptosis and extra-ocular muscle paresis. (Where you give edrophonium chloride (an anticholineresterase) and look for an improvement in symptoms as their Ach levels build up.)
  2. **Check for systemic weakness, difficulty in swallowing or breathing**. you have to ask about generlized symptoms because pt can convert to systemic
  3. **Assess orbicularis strength:** Ask the patient to close his eye strongly and open them
  4. **Blood test for: acetylcholine receptor antibodies (50% present in OMG)**

- Ocular myasthenia gravis patients might present with ptosis or diplopia or both. We have to ask if the symptoms change during the day (better in the morning or at night) and whether the diplopia is stable or not (horizontal or vertical).

- Assessing the orbicularis strength: by asking the patient to close both eyes strongly then we try to open them.

- Ach receptor antibodies in general myasthenia gravis = 60-80%, it is less in ocular MG.

- Tensilon test is diagnostic.

- Other tests for ocular MG like ice test: ask the patient to put an ice pad over the ptosis for two minutes and then check for any improvement (measure the degree of ptosis before and after).

- Sleep test: measure the degree of ptosis then ask the patient to sleep and re-measure after the patient awakes. (improvement = positive test)

- The pupils are not affected.

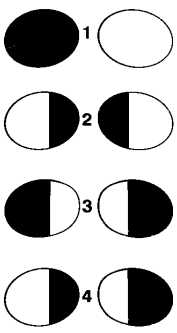
- Visual field defects: if unilateral then think about optic nerve pathology, if bilateral then the pathology is at the optic chiasm or beyond.

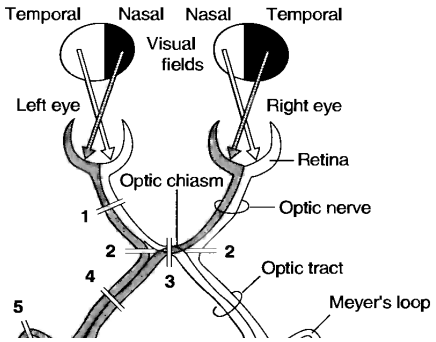
- Chiasmal defects are always bitemporal.

- Homonymous visual defects could be due to stroke or tumors.

Part 4: Visual pathway disorders

Lesions anywhere in the visual pathway will produce visual field defect.





7.macular sparing ? daul blood supply occipet is supplied by (middle + posterior cerebral artery)

ess due to Left optic nerve damage

ıopia due to bilateral carotid artery aneurysm compressed optic chiasm

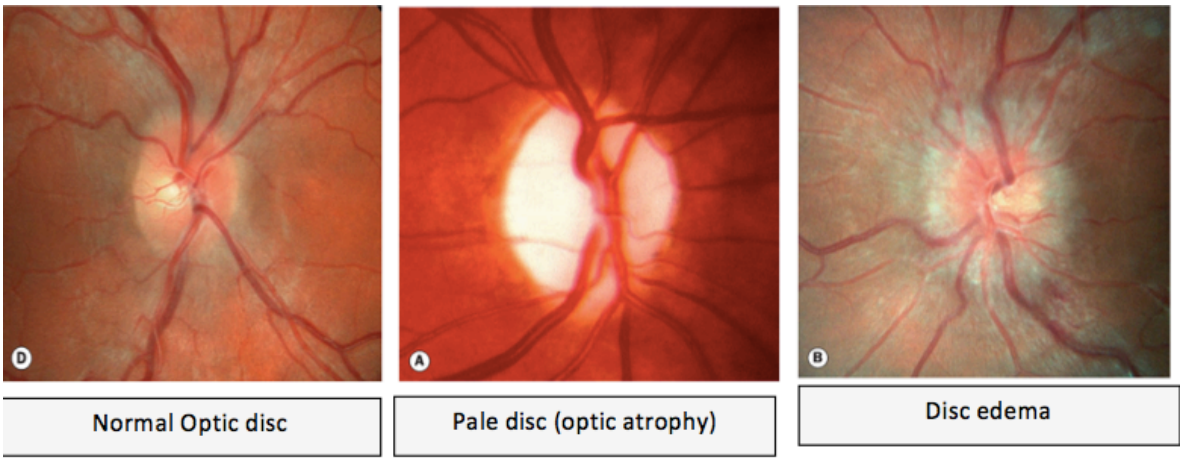
ımianopia due to pituitary tumor compressed optic chiasm

ınous hemianopia due to Left optic tract damage

quadrantic hemianopia due to Left optic radiation at temporal lobe lesion (pie in the

Optic Nerve Disease

The normal optic nerve head has distinct margins, a pinkish rim and, usually, a central, pale, cup. The central retinal artery and vein enter the globe slightly nasally in the optic nerve head, referred ophthalmoscopically as the **optic disc.** **Optic neuropathy is usually unilateral (if bilateral this is papilledema), afferent pupillary defect, central visual loss, loss of colour vision, optic disc edema or atrophy.**



Normal Optic disc | Pale disc (optic atrophy) | Disc edema

How to assess the optic nerve in the clinic: visual acuity, visual field, color test and afferent pupillary defect

1. Optic Neuritis:

- Inflammatory demyelinating condition associated with Multiple sclerosis
- Most common type in **female young adults**
- **History:** Patient will come with **sudden** visual loss with ocular pain while moving the eye
- **Why ocular pain happened?** Because **optic nerve sheath is attached to medial rectus muscle sheath**
- **Signs:** reduce visual acuity – Positive afferent pupillary defect – Optic disc edema – Pain with eye movement (optic nerve sheath is in close association with ocular muscle and because it's inflamed any movement will cause pain) – **scotoma visual field defect**
- **Treatment:** IV steroids my speed up the recovery process but does not influence the final outcome.
- Good recovery.

-A patient with optic neuritis needs an MRI of the brain and orbits to look for enhancing lesions. -Oral steroids if given alone might increase the risk of recurrence.

Relative Afferent Pupillary Defect (RAPD, Marcus Gunn Pupil) [1]:

An RAPD is a defect in the direct response. It is due to damage in optic nerve or severe retinal disease.

It is important to be able to differentiate whether a patient is complaining of decreased vision from an ocular problem such as cataract or from a defect of the optic nerve. If an optic nerve lesion is present, the affected pupil will not constrict to light when light is shone in the that pupil during the swinging flashlight test. However, it will constrict if light is shone in the other eye (consensual response). The swinging flashlight test is helpful in separating these two etiologies as only patients with optic nerve damage will have a positive RAPD.

2. Ischemic Optic Neuropathy

The anterior optic nerve may become ischemic if the posterior ciliary vessels are compromised as a result of degenerative vaso-occlusive or vasculitic disease of the arterioles, which result in an anterior ischemic optic neuropathy.

A. Non-arteritic IOP:

- **History:** Old patient known to have DM and HTN come with sudden visual loss
- **Signs:** Optic disc edema and **Altitudinal (either upper or lower field) visual field loss**

B. Arteritic IOP :

Seen in patient **older than 55 years** and mostly associated with **giant cell arteritis (GCA).** They present with **sudden loss of vision, scalp tenderness, headache, pain on chewing "jaw claudication", proximal myalgia and arthralgia.**

Signs:

- ✓ Reduction in visual acuity
- ✓ Field defect
- ✓ Swollen and hemorrhagic disc with normal retina and retinal vessels – in AIOP the disc may be pale.
- ✓ Small normal fellow disc with a small cup in NAIOP.
- ✓ Tender temporal artery, suggestive of giant cell arteritis.

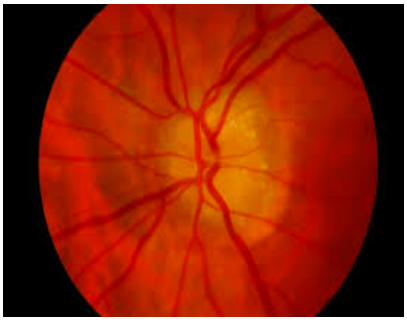
Investigation:

1. If giant cell arteritis is present, **the erythrocyte sedimentation rate (ESR) and C- reactive protein (CRP) are usually grossly elevated.**
2. **Temporal artery biopsy is the gold standard for diagnosis.** **(it should be long enough because it has skipping lesion criteria (2.5 length)**

Treatment: Systemic steroids should be given immediately if GCA is suspected. **To safe the other eye**

3. Congenital Disc Elevation: <1%

it is a rare disease. Optic disc margins are **blurred and the cup is absent but no edema or haemorrhage can be observed**. May be associated with hyperopia "farsightedness" or drusen "yellow deposits under the retina made up of fatty proteins". B-scan ultrasound can discover drusen (lipid collections)



Other causes of optic neuropathy:

- Infection e.g. viruses, TB, Cryptococcus and syphilis.
- Systemic connective tissue diseases e.g. SLE.
- Genetics: Leber's optic neuropathy*.
- Toxic and nutritional deficiencies.
- Trauma

* It is mitochondrial DNA mutation. Usually they present in young age with sudden drop of vision unilaterally or bilaterally . ask about another family member suffering from the same condition.

Papilledema

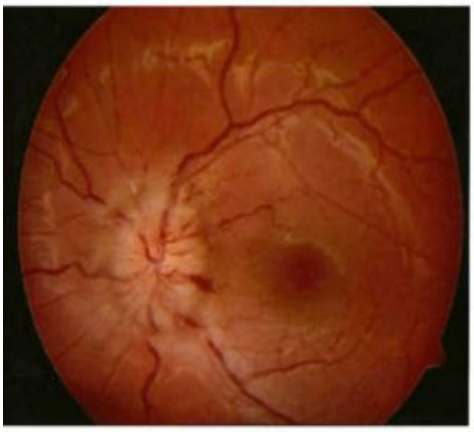
It is a bilateral swelling of the optic disc secondary to **raised intracranial pressure.** Could be caused by **intracranial mass, severe systemic hypertension, or idiopathic intracranial hypertension (pseudotumor cerebri): Female – Obesity - Tetracycline**

Symptoms:

- Headache, worse on awakening
- Nausea and vomiting if the raise in ICP is severe, may be followed by loss of consciousness, pupillary dilatation and death.
- Pulsatile tinnitus
- Visual symptoms often are absent

Signs:

- **Disc hyperemia**
- **There is no spontaneous venous pulsation of the central retinal vein.** If venous pulsations can be visualized, the cerebrospinal fluid pressure is typically less than 200 mm of water.
- **Blurring and elevation of the disc margins**
- **Peripapillary flame shaped haemorrhage**.
- **6th nerve palsy**.



- Papillitis: edematous or inflamed optic disc.

- Important signs in optic nerve disease: blurring of the margins, splinter hemorrhage in the peripapillary area, edema and elevation of the disc.

- The presence of hemorrhage = acute raise in the pressure.

- Papilledema is a diagnosis of exclusion should be confirmed by lumbar puncture.

DDx:

- Adult optic neuritis
- Hypertension
- Idiopathic intracranial hypertension
- Pseudopapilledema

Investigations:

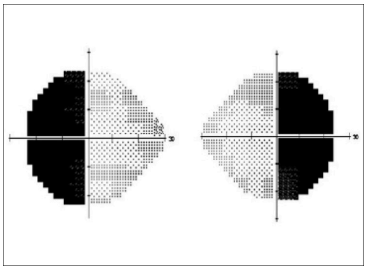
- CT or MRI followed by lumbar puncture (to measure the ICP and rule out meningitis.)
- B-scan ultrasonography to rule out buried disc drusen.
- Fluorescein angiography

Treatment:

Medical: Diamox, diuretics.
Surgical: if not controlled by medication:
- optic nerve fenestration: slit cut of optic nerve sheath > fluid will come out and release the compression
- Shunt: for patient who has sever headache and blurred vision

MCQs:

1. A patient presented with this visual field defect. Which one of the following diagnosis is the most Likely?



A. **Optic neuritis**
B. **tilted discs**
C. **pituitary tumor**
D. **6th nerve palsy**

2. You have a patient with diplopia. His left eye is turned down and out and his lid is ptotic on that side. What nerve do you suspect and what should you check next?

This sounds like a CN3 palsy, and you should check his pupillary reflex. Pupillary involvement suggests the lesion is from a compressive source such as an aneurysm.

3. A 26 year old woman presents with decreased vision in her left eye that has gotten progressively worse over the past week. The eye seems to ache and the vision worsens with exercise. On exam she is found to have 20/200 vision, trace APD, and markedly decreased color vision in the affected eye. The optic nerve is mildly swollen on that side. What does this patient most likely have?

This patient's age, color vision, and progression are all classic symptoms of optic neuritis. She also describes the classic Uthoff phenomenon of worsening symptoms with increased body-temperature (exercise or shower). Many of these patients describe minor pain with eye-movement; the optic nerve is inflamed and any tugging on the nerve with eye movement is going to irritate it.

4. An 84-year-old man was out golfing with his buddies and developed sudden vision loss in his right eye. He has no past ocular history, no medical problems. No complaints of flashes or floaters, just that things "look dimmer" in his right eye. What other questions should you ask about his symptoms?

There are many questions you should ask … but with any elderly person with vision loss, be sure to ask about the symptoms of temporal arteritis. Specifically, scalp tenderness, jaw claudication, and polymyalgias (muscle aches in the shoulders and arms). This sounds like a central retinal artery occlusion, and in a patient this old you need to rule out life- and visionthreatening causes like GCA (giant cell arteritis).

Summary

Pupillary disorders:

Tonic pupil (Adie's pupil): - enlarged, is poorly reactive with light, slow sustained miosis on accommodation, Constricts to dilute Pilocarpine , unlike the normal eye, this is a diagnostic test.
Systematically: the disorder is associated with loss of tendon reflexes.

Horner's syndrome: Small pupil, enophthalmos, anhydrosis, ptosis.

Neuromotility disorders:

3rd: drooping of the eyelids "ptosis", dilatation of the pupil that is unresponsive to light and accommodation, and an inability to move the eyeball upwards, downwards, or inwards "adduction".

4th: an upward deviation of the affected eye and a "cyclotorsion" twisting of the eye that makes them tilt their heads away from the lesion.

6th: inability to move the eye outwards "abduction". Patients will go crossed-eyed "Esotropia = one or both eyes turn inward".

Neuromuscular disorders:

MG: ptosis with or without diplopia, the pupil is intact.

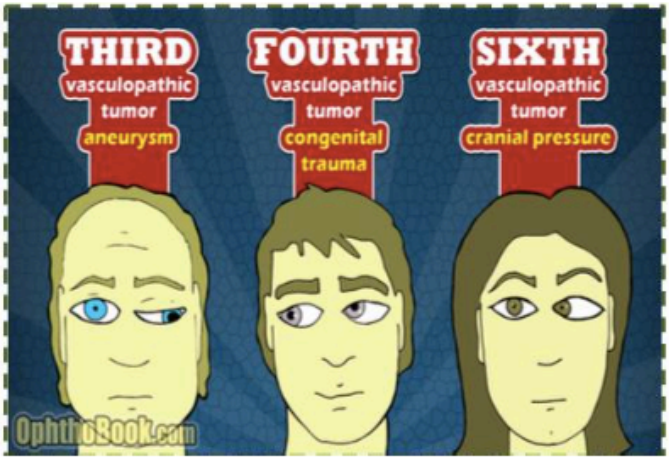
Visual pathway disorders:

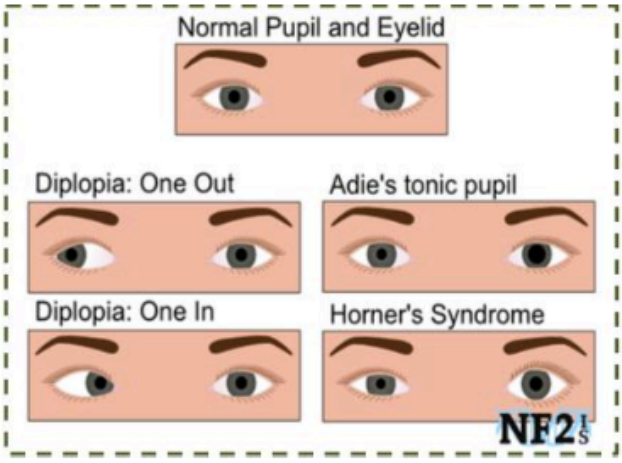
Optic neuritis: disc edema except if retrobulbar neuritis, reduced visual acuity and color vision, central scotoma, RAPD.

Papilledema: swollen disc, blurred margins, capillary dilated, no venous pulsation.

Ischemic optic disease: field defect, swollen disc with normal margins, tender temporal artery if (Giant cell arteritis).

Congenital disc elevation: optic disc margins are blurred and the cup is absent but no edema or hemorrhage can be observed. May be associated with hyperopia.





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