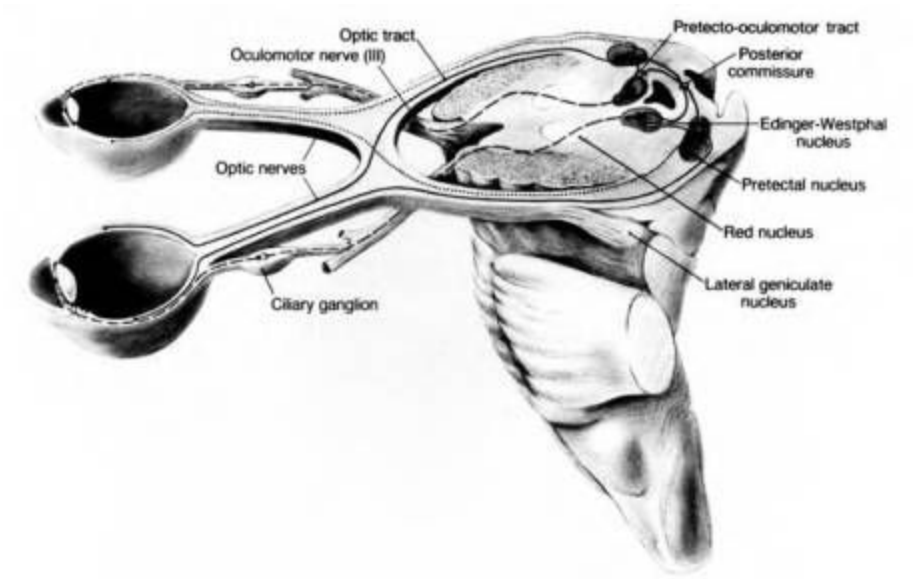


Neuro-Ophthalmology

2017-2018

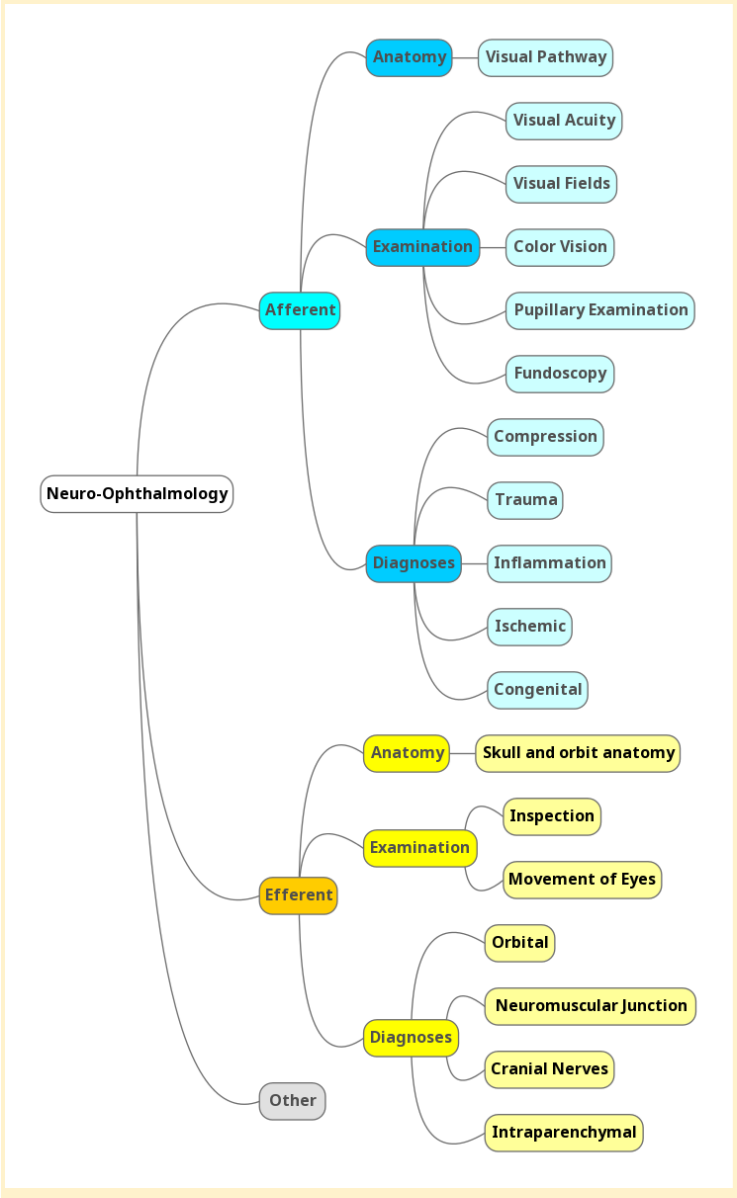
Done By: Reema AlRasheed & Nouf AlMasoud

Editing File

Resources: 433 Slides & Team (Group A), Doctor's Notes, Ophthobook

NOTE: The slides were not given by the doctor, so the teamwork is basically done by using *dr's notes, 433 group A slides and 433 teamwork* which have different pictures, good luck!

Lectures Outlines:



Before we start we need to know that the neurological part of visual system is composed of 2 parts:

1. Afferent visual system= Vision = Optic nerve
2. Efferent visual system= Motility, Pupil size, Eyelids = (CN3,4,6) + Central nuclei in the brainstem.

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

★Afferent Visual System

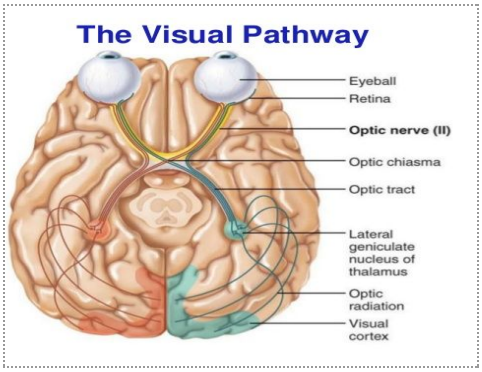
A. Anatomy & physiology: The optic nerve is formed by:

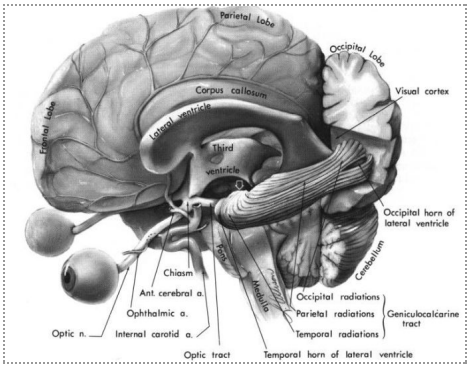
1. *Nasal* tract, which is responsible for the nasal part of the field and *temporal*, which is responsible for the temporal part of the field.
   Both fibers will run in the optic nerve
2. 53% of the fibers will decussate:
   Which fibers decussate?? Nasal fibers. Remember they're nearer to the midline, so it's easier to decussate.
3. Optic chiasm => tract => Lateral geniculate nucleus of thalamus (LGN)=> optic radiation=> visual cortex.

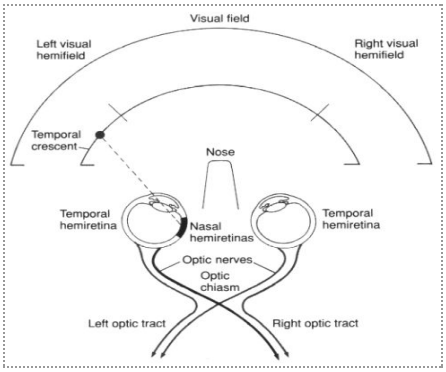
What do we mean about visual field?

Island of vision, triangular in shape because it expands the further it goes. Each eye has its own triangle , however there's a common area in the middles which is covered by both eye.. that's why when we close our eyes we only lose 1/5th of the field (20%).

This common area provides the depth perception of picture (Stereopsis). That's why you cannot watch a 3D movie with one eye..

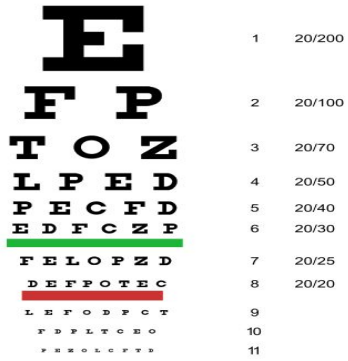






B. Examination & Test:

1. Visual Acuity: (monocular vision test)



*Distant vision:

- Snellen's chart:

Ask the patient to cover one eye and name the letters which may be one of several letters, for example E, H, or N. Subsequent rows have increasing numbers of letters that decrease in size. A person taking the test covers one eye from 6 metres or 20 feet away, and reads aloud the letters of each row, beginning at the top. The smallest row that can be read accurately indicates the visual acuity in that specific eye.

Ex: when the patient's visual acuity= 4/6, the patient can see a letter if he/she stands 4 meters far from it, however a normal patient can see the letter 6 meters far from it.

- E-game: The chart shows Es only and the patient says its direction.

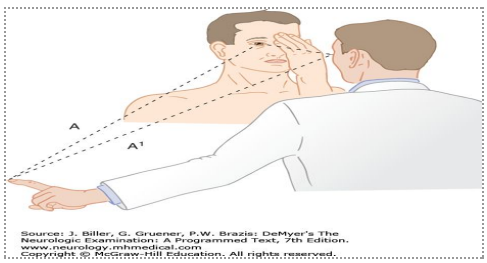
*Near vision

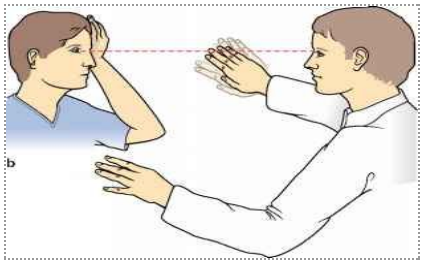
2. Visual Field: (monocular vision test)

*Directly by the doctor:

- is a qualitative test that can be done in ER, bedside or in the clinic
- **H shape is common mistake..**
- Forget about we've studied in neuro so far, the right way to examine the visual field gonna be demonstrated by our ophta doctors.

1. It's called confrontation test, because you stand in front of the patient.
2. You should be on the same level of patient's eye.
3. Your projecting fingers should be midway between you and the patient (not closer to you or to the patient), you can stretch your arm to mid periphery.
4. Four quadrants , project number in each quadrant.
5. Remember it's one number in each quadrant, Do not wave your fingers.
6. Ask the patient, do you see the details of my eye?? if he/she said: no it's really blared => the central scotoma*. (go to the next page)





*Central scotoma:

Central: in the middle of the field

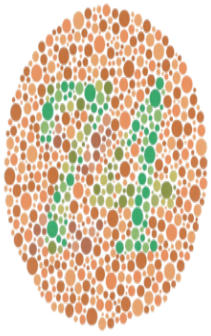
Scotoma: is an area of partial alteration in the field of vision consisting of a partially diminished or entirely degenerated visual acuity(مشوش والا فاضي)that is surrounded by a field of normal – or relatively well-preserved – vision.

Indirectly by perimetry:

- Peri=visual field, metry:to measure

1.Static(automated):	2.Manual(Goldmann):
-Uses a mobile stimulus moved by a perimetry machine. - The patient indicates whether he sees the light by pushing a button. - Computerized.	-First, a single test light of constant size and brightness is used. The test light is moved towards the center of vision from the periphery until it is first detected by the patient. -This repeated by approaching the center of vision from different directions. Repeating this enough will establish a boundary of vision for that target. -Operator dependent
-Static perimetry tests different locations throughout the field one at a time. -is a quantitative test to measure how much of the field is lost!	-Kinetic perimetry uses a mobile stimulus moved by an examiner (perimetrist), toward the center. -interactive test, will tell you if your patient is cooperative or not.

3. Color Vision:



Ishihara test: is a color perception test to role in or out color blindness, but it doesn't specify which color blindness(blue, green..) does the patient has. For clinical use.

The total score is 15=> each time the patient mistaken, count it: so if there's 5 mistakes=> 10/15.

The Farnsworth-Munsell 100 Hue Color Vision Test, or Munsell Vision Test:

Colored circles like the make plates,(blue, orange, yellow... where the patient arrange them in order from the lightest to the darkest, then flip it => put them in Excel sheet=> analyze=> will get the color blindness type. Is used for research purposes only and not used in the common practice.

4. Pupillary Examination:

1st let's discuss pupillary physiology:

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

	Parasympathetic innervation:	**Sympathetic innervation**
Function	pupillary constriction.	pupillary dilation.
Origin	1. Originate from Pretectal nucleus in 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.	Originate from hypothalamus and go through superior cervical ganglia
Muscle	Sphincter pupillae muscle: Supplied by parasympathetic fibers of Oculomotor nerve and lead to constriction of pupil .	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.
Note	Pupil constrict to light and near stimuli.	If there is a cut through sympathetic pathway patient will develop signs of Horner syndrome.

	Abnormal Dilation of pupil:	**Abnormal Constriction of pupil:**
Causes	*Previous ocular surgery *Ocular trauma *Cycloplegic medication e.g. Atropine, cyclopentolate *Third nerve palsy (mid dilated fixed pupil, does not respond to light) &Tonic pupil (Adie's pupil)	*Previous ocular surgery *Ocular trauma or inflammation of the margin of the pupil (iris) is attached to the lens (posterior synechia) or to the cornea (anterior synechia)(adhesion) *Medication e.g. pilocarpine *Horner syndrome.

★ **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. Which is called light near dissociation.
- Instillation of weak cholinergic agents (0.1%pilocarpine) will cause constriction of the tonic pupil (denervation hypersensitivity), the normal eye won't change

★ **Horner syndrome:**

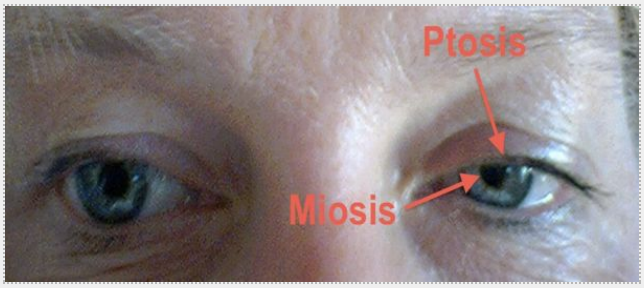
**Cause:* interruption of sympathetic pathway (Carotid dissection, carotid aneurysm and tumor)!!!

**Signs:* at the side that affected you will see:

- Miosis: due to loss of dilator function.
- Anhydrosis lack of sweating
- Ptosis: due to paralysis of muller's muscle.
- Enophthalmous. (posterior displacement of the eyeball): due to paralysis of levator
- palpebrae muscle.

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

- Acute or chronic:
  - Acute within 2 weeks => immediate neuroimaging.
  - Chronic within several months or he has a surgery => follow up.
- Painful or painless:
  - Painful=>immediate neuroimaging. (Sometime carotid dissection presents with painful Horner syndrome)



Now let's go back to the testes.

**Direct pupillary light reflex (Pupillary Reaction):*

1. Take your torch.
2. Switch off the room light.
3. Ask the patient to look to specific far target (number 12 on the wall clock)
4. Be on the right side of the patient, dont block the view from him/her.

5. Switch on the torch, wait 1-2 sec.
6. Observe the pupillary reaction, is it reactive (constricting) or not?

- The normal pupillary reaction to light reflex is Hippus pupil. Hippus, also known as pupillary athetosis, is spasmodic, rhythmic, but regular dilating and contracting pupillary movements between the sphincter and dilator muscles. click to see a video.

- What is Being Tested?

Direct response (pupil illuminated). The direct response is impaired in lesions of the ipsilateral optic nerve, the pretectal area, the ipsilateral parasympathetics traveling in CN III, or the pupillary constrictor muscle of the iris.

**Consensual pupillary light reflex (Pupillary Reaction):*

7. Compare the amount of constriction on the tested eye and the other one at the same time without moving the torch..

- Remember always to comment on, The size, shape and position of each pupil.
- What if you have found that the pupils are in-equal in size (Anisocoria)?

1st we need to know which one is the abnormal one, the big or the small one?
Do light reflex test,
in dim light:When the small pupil does not dilate as well as the large pupil, then the small pupil is abnormal. in response to a light stimulus: when the larger pupil does not constrict as well as the small pupil, then the large pupil is abnormal.

- What is being test?

Consensual response (contralateral pupil illuminated). The consensual response is impaired in lesions of the contralateral optic nerve, the pretectal area, the ipsilateral parasympathetics traveling in CN III, or the pupillary constrictor muscle.

Ex. the left and the right pupil both are reactive, central and same size, however I think that the right pupil reflex is weak, How to be sure?? Do **swinging flash test.**

**Swinging flash test (Marcus gunn reflex):*

1. Take your torch.
2. Switch off the room light.
3. Ask the patient to look to specific far target (number 12 on the wall clock)
4. Be on the right side of the patient, dont block the view from him/her.
5. Switch on the torch, wait 1-2 sec.
6. Observe the pupillary reaction, is it reactive (constricting then dilate a little=Hippus pupil) or not?
7. Shift to the other eye, is it reactive (constricting then dilate a little=Hippus pupil) or not?

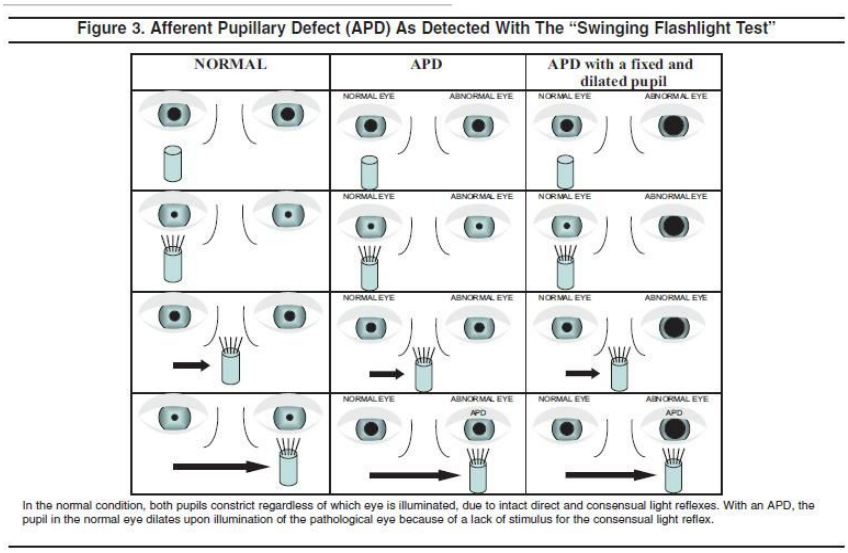
So, what happens in **relative afferent pupillary defect (RAPD) or marcus gunn pupil?**

When the optic nerve is damaged, the sensory (afferent) stimulus sent to the midbrain is reduced. The pupil, responding less vigorously, dilates from its prior constricted state when the light is moved away from the unaffected eye and towards the affected eye.

Did not get it yet?? Don't worry we're gonna simplify it by an example..

Ex.

Torch was directed to the healthy eye=> the healthy eye is going to absorb **all the stimulus (100%)** through afferent=> and then send it as an efferent to the healthy and diseased eye=> **both healthy and diseased eye are going to constrict equally 100%(2 healthy efferent)**=> now swing your torch to the other eye (diseased one)=> this eye diseased afferent is going to absorb only a few from the stimulus(ex. 50%)=> travel back through the efferent=> the diseased eye will constrict 50% only=> so going from full (100%) constriction to less constriction (50%)=> the diseased pupil will look like is dilating.



- What is being tested?

Looking for a relative afferent pupillary defect (RAPD)

**Near reflex*: Video

Take the target and while moving it closer to the patient eye observe for three things that happen while looking to a near object:

1. pupillary constriction(miosis).
2. convergence:is the ability of the eye to simultaneously demonstrate inward movement of both eyes toward each other.
3. accommodation: increase the curvature of the lens in response to looking at something moving toward the eye.

- What's light near dissociation?

Argyll Robertson pupils (AR pupils or, colloquially, "prostitute's pupils") are bilateral small pupils that reduce in size on a **near** object (i.e., they accommodate), but do not constrict when exposed to bright **light** (i.e., they do not react to **light**)
ex. **Tonic pupil (Adie's pupil).**

5. Fundoscopy (look to the nerve itself) :
**Direct fundoscopy=>* monocular=> 15 times increase in size of the disc+ erect image.
**Indirect fundoscopy=>* we use lens to increase the refractive power to 80,=> binocular=> 3 times increase in size of the disc+inverted image.
We can also use slit lamp to examine the fundus

6. Ultrasound:

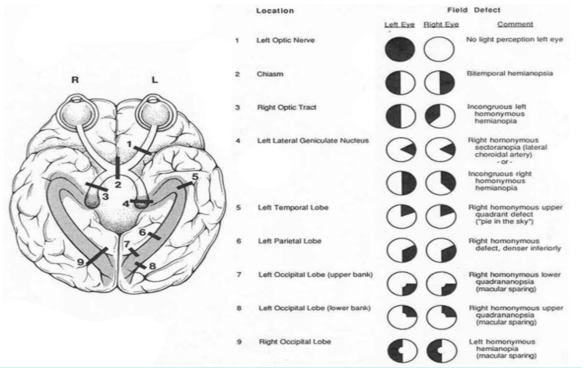
If there's opacity of the lens or the other medias which block the way of fundoscopy or slit lamp.

C. Diagnosis (dr said don't need to memorize it):

Compression	Intraorbital ON	Intracranial ON	Optic chiasm	Optic tract	Posterior afferent system
Trauma	Globe	Intraorbital ON	Optic canal	Optic chiasm	Occipital lobe
Inflammation	Optic neuritis	Orbital pseudotumor	Other		
Ischemic	Non-arteritic ischemic optic neuropathy	Central retinal artery occlusion	Other retinal emboli	Giant cell arteritis with ION	
Congenital and genetic problems	Congenital retinal dystrophies	Optic nerve hypoplasia	Leber hereditary optic neuropathy	Dominant and recessive optic atrophy	Glaucoma

*Visual pathway disorders:

1. Learn the terms
   - Anopia or Anopsia = Blindspot
   - Half of the field loss = **Hemi**anopia/psia
   - Quadrants = Quadranopia
   - Upper Quadrant = Superior Quadrantanopia
   - Lower Quadrant = Inferior Quadrantanopia
   - Same sides in the circles = Homonymous
     - Ex = Left and left.
   - Different sides in the circles = Heteronymous



        - Ex=Left and right.
    - The lesion side is opposite to the circle side=heteronomous.
    - Field of the circle = Medial = Nasal.
    - Field of the circle = Lateral = Temporal.
    - Wedge shape = LGB = Lateral geniculate
    - Central = Macular = Dual blood supply (Ms= MCA+PCA)

2. Start naming

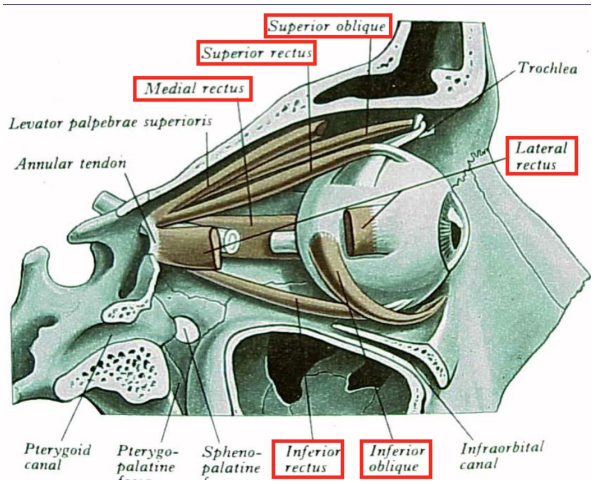
Ex: let's solve some of them together

1. Complete left eye blindness=> Left Anopia=>lesion in the Left ON(before decussating).
2. Half of the left and the right(Hemianopia, however different sides(hetro), the field in both eyes is lateral(temporal), However in this case we're going to replace hetro with (bi)=> Bitemporal hemianopia=> lesion to chiasm.
5. Quadrants of the left and the right(Quadrantanopia), the black is in both eyes on the right side(hemi), the field in both eyes is upper(superior), => Right Homonymous Quadrantanopia= > lesion to Left Temporal lobe.

★Efferent Visual System

- **Anatomy** doctor mainly focused on orbital anatomy and skipped other parts

Skull and Orbit anatomy



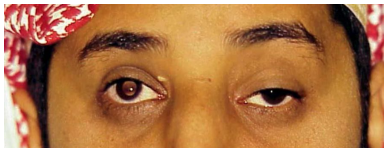
The space behind the globe is called "muscles con" any mass here can compress the optic nerve causing optic nerve damage, if it's intraconal mass it will push the globe forward (axial), if the mass is up the globe will go down.

B. Examination

- Just look at patient
  - i. Are eyes straight? the alignment.

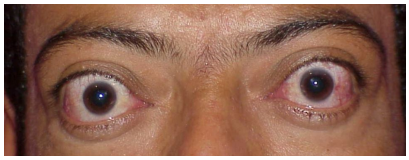

picture shows esotropia in left eye

  - ii. What are the lid positions? ptosis or retraction.



Differential diagnosis of ptosis:
"Neural causes"
- Myasthenia gravis
- Third nerve palsy
- Horner's syndrome

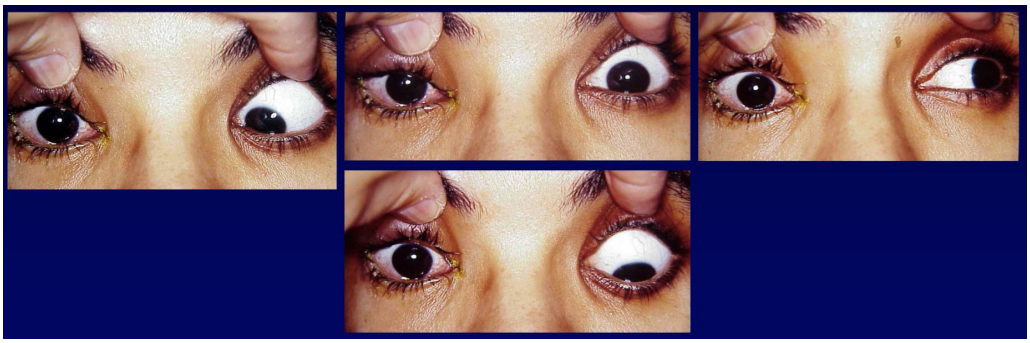
  - iii. Are the eyes proptotic?



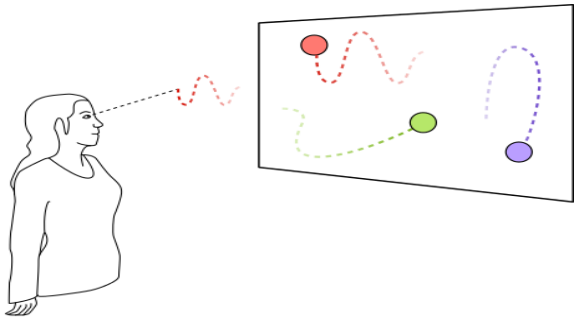
Picture shows: proptosis, lid retraction and conjunctivitis
Most likely diagnosis:
Thyroid orbitopathy

  - iv. Are there any spontaneous eye movements? Nystagmus

- Movements of both eyes in all directions (binocular test)
  - i. Have the patient move eyes in all directions, not just the direction where you think there is a problem. "in + X directions"
  - ii. Hold lids if necessary (only after looking first without holding lids)
  - iii. Examine each eye separately if necessary

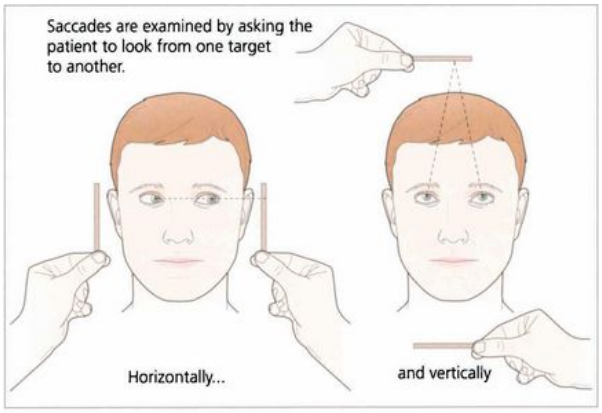


- Smooth pursuit the ability to track a moving object.
  i. The reflex that helps to maintain fixation on an object in motion in the visual world while the head is stable
  ii. Also the reflex that inhibits the vestibulo-ocular reflex
  iii. We look if it's smooth or not, for example parkinson's patients have cogwheel movement called "saccadic pursuit", patient mostly complain of the inability to watch football games or playing video games..etc



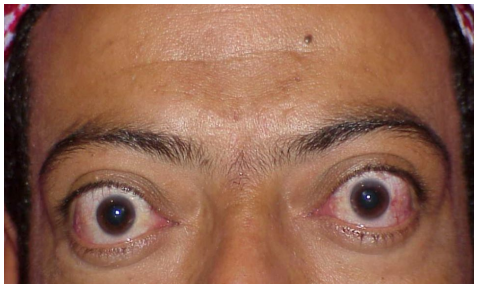
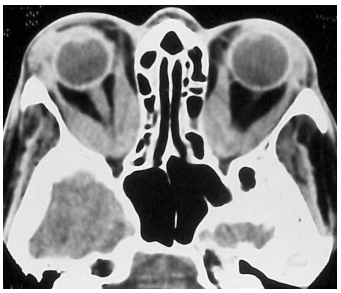
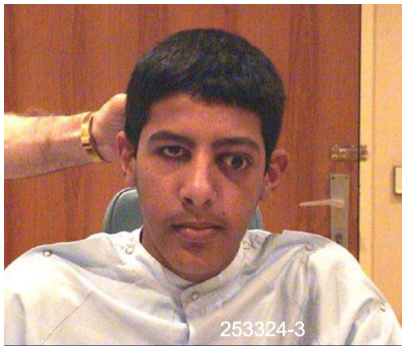
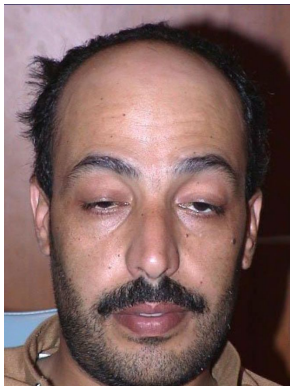
VIDEO

- Saccades the ability to fix form one target to another target.
  i. The reflex that permits a rapid refixation from one point in the visual field to another.
  ii. Basically testing how fast and accurate of movement to the target.Just like dysmetria where they overshoot the target.



VIDEO

C. Diagnoses what can affect the ocular motility? can be either:

<table>
<tr><th colspan="2">1. Orbit</th></tr>
<tr><td>a. Extraocular muscles
Thyroid Orbitopathy:
CT showing spindle shaped enlargement of the muscles which pushes the globe anteriorly.
Ideally if you draw a line from the temporal bone brims you should only have 1 third of the globe anterior to your line.</td><td>   </td></tr>
<tr><td>b. Trauma:
Blowout or Orbital floor fracture:
The maxillary bone fractures downward and the orbital contents can herniate down into the underlying maxillary sinus > thus entrapment of inferior rectus muscle > when patients look up the IR can’t relax > “pt present with enopthalmia (a sunken-in eyeball)</td><td>   </td></tr>
<tr><td>c. Mass</td><td></td></tr>
<tr><th colspan="2">2. Neuromuscular junction</th></tr>
<tr><td>a. Myasthenia gravis
Picture showing Ptosis and no obvious wrinkles despite raising the eyebrows.
-The diplopia and ptosis is usually worse on prolonged upgaze: you can test this by having your patient look at your raised finger to see who tires out first.</td><td>   </td></tr>
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3. Single cranial nerves injury

a. Oculomotor (CN3) palsy:

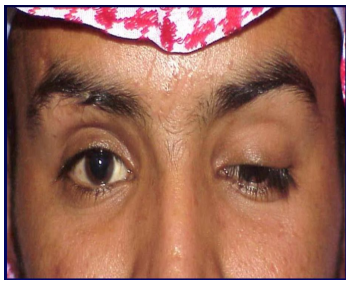
- *Clinical features:*

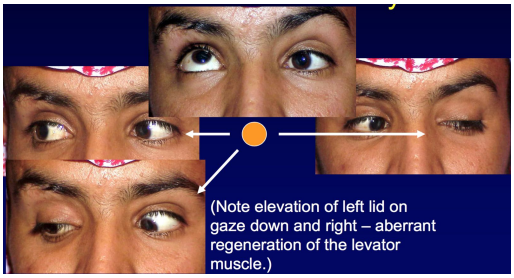
* The eye will deviate down and out
*Eyelid ptosis

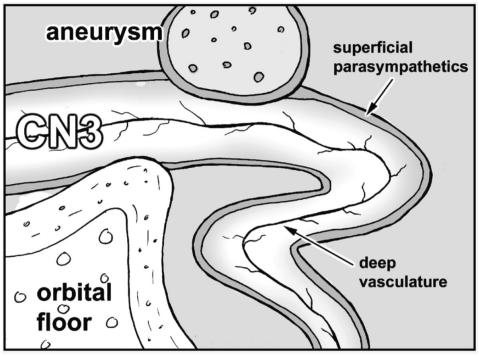
- *Causes:*

*Ischemic events at the nerve secondary to hypertension or diabetes.
*Compressive aneurysm pushing on the nerve commonly posterior communicating artery and the internal carotid artery.
-The most common microischemic events and 2nd common PCA aneurysm.

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 tumor.







Pupillary involvement because the parasympathetic nerves innervating the iris travel with the third nerve. Pupillary involvement is a crucial diagnostic sign‹ compressive lesions tend to involve the pupil, while vascular lesions might actually spare it! As you can see, the parasympathetic nerves course along the surface of the oculomotor nerve making them susceptible to compressive lesions from the outside while ischemic lesions occur deeper within the oculomotor nerve and thus spare the superficial parasympathetic fibers.

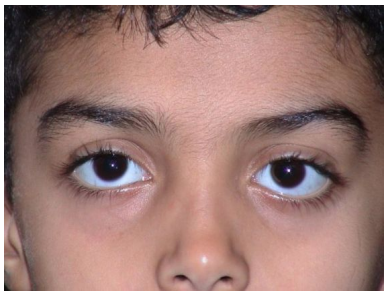
b. Trochlear nerve (CN4) Palsy

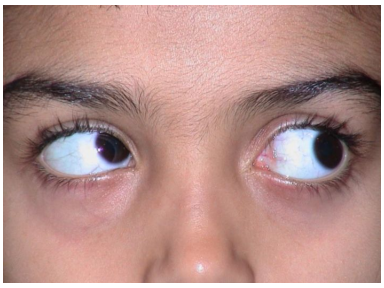
- *Clinical features:*

*Upward deviation.
*Cyclotorsion that makes them tilt their head away from the lesion.
*Vertical diplopia
*IO overaction

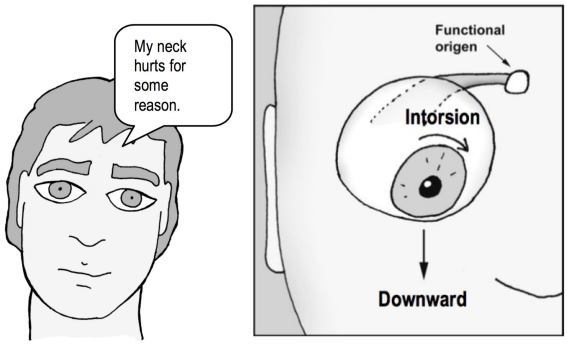
- *Causes:*

*Trauma
*Congenital
*Ischemic (diabetic)





*Tumor



Trochlear Muscle Action

c. Abducens nerve (CN6) Palsy

- *Clinical features:*

*Unable to abduct (turn out).

*Patients will go cross-eyed, so to compensate they may turn their head to avoid double vision (Horizontal binocular diplopia) worse at distance.

*Face turn in the direction of the paralyzed muscle

Causes:

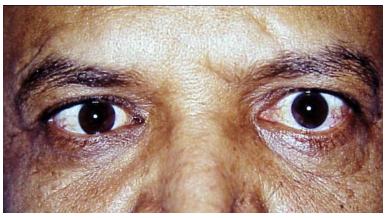
*Intracranial tumors

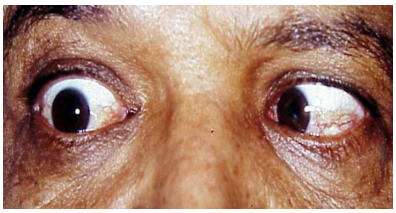
*Trauma (most common cause because it's long nerve)

*Microvascular diseases (mostly DM)

*High ICP from pseudotumor cerebri

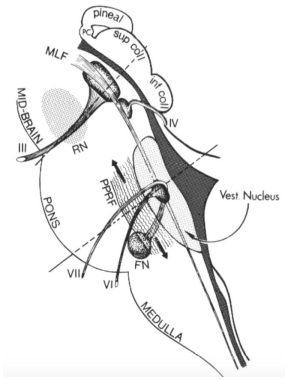
If patient is more than 55 and have chronic disease it's simple but if patient is young with no explained pathology you need to do an MRI to rule out serious pathologies.





4. Multiple cranial nerves

If patients present with symptoms indicating multiple cranial nerves involvement you need to do imaging to rule out cavernous sinus or brainstem lesions, as it might be a lesion compressing the cranial nerves at the sight of their origin in brainstem.



5. Intraparenchymal problem

a. Internuclear ophthalmoplegia (INO)

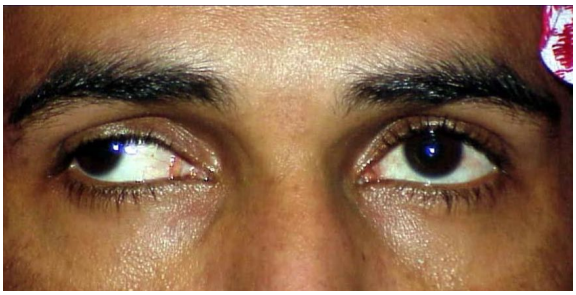
- *Clinical features:*

*One eye will have limited adduction
*The other eye will have abducting nystagmus

- Causes:

-Lesion in the medial longitudinal fasciculus; Most common cause:

- Young > Multiple sclerosis
- Elderly > Strokes

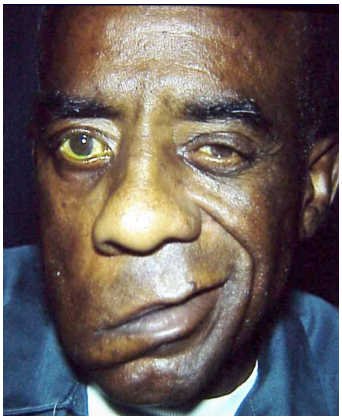


b. Facial nerve palsy:

We all know that :) i'll just mention what the doctor focused on:

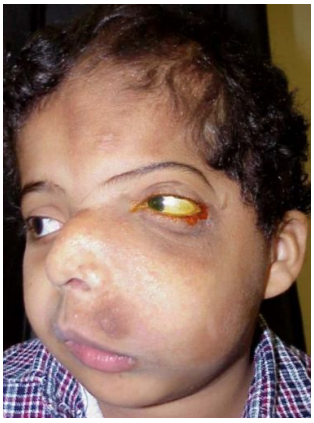
- *Common ophthalmological* Complications:

*Corneal dryness > ulceration > melting > perforation.

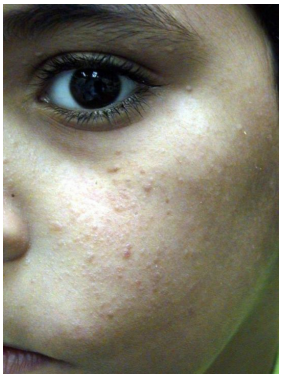


★Others:

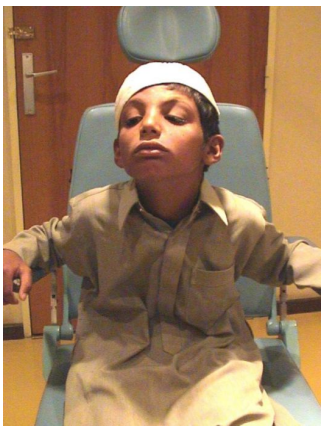
1. Unusual faces
   - Neurofibromatosis: can affect face, orbit, or optic nerve (ON glioma)



2. Unusual skin lesions
   - Adenoma spicia (tuberous sclerosis) can cause brain astrocytoma



3. Unusual postures
   - obtaining head posture > chin-left to compensate ptosis
   - If you leave this child he will have spinal deformities

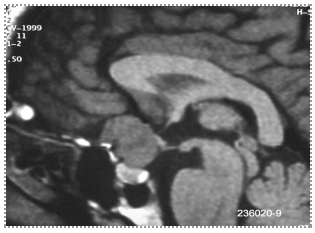


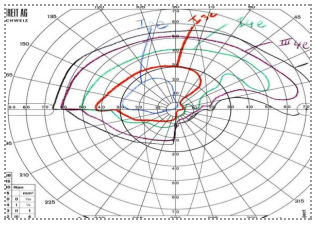
and further more, so neuro-ophthalmology is not only about the globe it's about the whole body.

★Cases:

1. 35 year old woman, reported 3 months of gradual visual loss OU , 7 months pregnant, rapid visual loss OU over 1 week before, N-O evaluation: VA at presentation OD 20/40 and OS NLP.

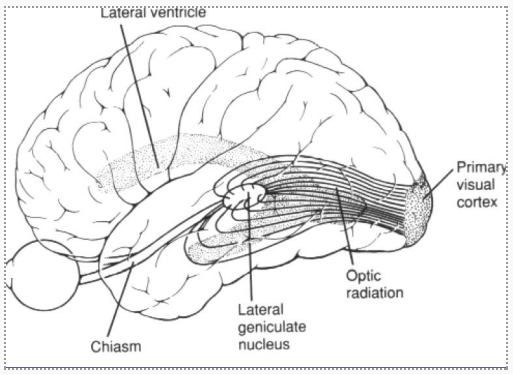
VA=Visual acuity
VF=Visual field
OU=Oculus uterque (both eyes)
OS=Oculus sinister (left eye).
OD=Oculus dexter (right eye)
NLP=No light perception

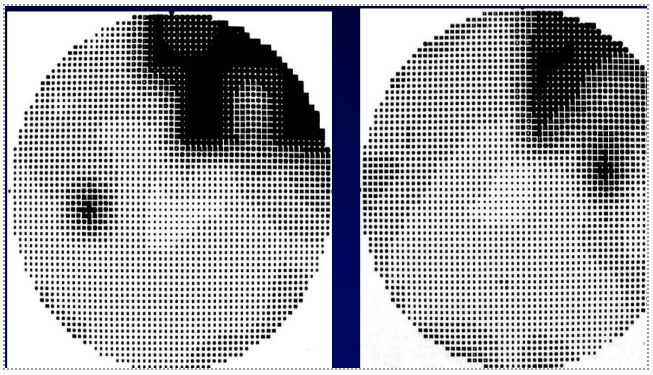




post-op:

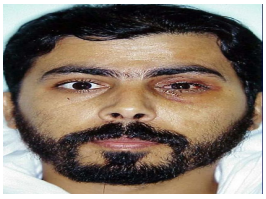
Before temporal lobectomy After





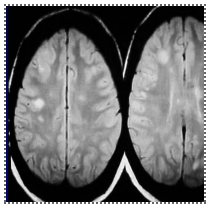
2. 25 year old man, Firecracker exploded, near left eye ,NLP OS. ON ablation

*The commonest cause of trauma in children is door handle



3. 27 year old woman, Developed blurred vision OD and mild right periorbital pain, VA 20/50 , MRI abnormal

*Diagnosis multiple sclerosis

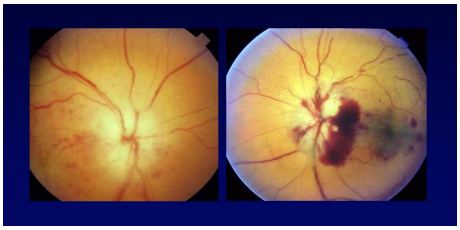


4. 28 year old woman, Developed modest, left periorbital pain 3 weeks ago, Some blurring of vision OS,

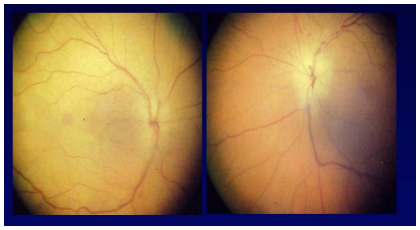
B-scan showed, posterior scleritis

5. Giant cell arteritis:

Day 2 post Rx



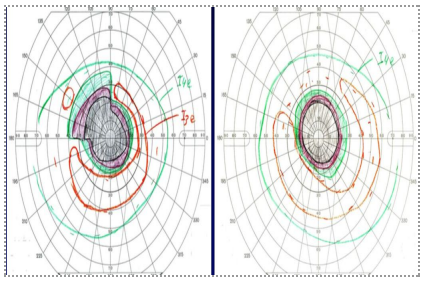
Day 4 post Rx



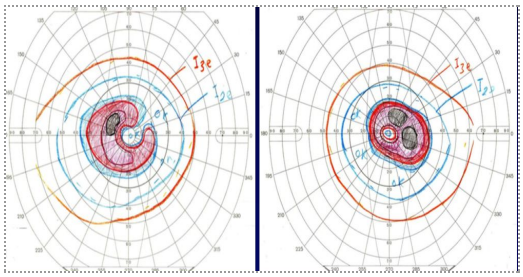
6. 14 yo girl, Vision OS began to decline gradually without pain, First visit with VA OD 20/40 and OS CF at 3, Pale, flat optic disks OU, VA 1 month later CF at 3' OU, VA 10 months later 20/20 OU

Diagnosis LHON-like optic neuropathy

VF 1 mo after



VF 10 mo after



Notes:

- Papilledema is bilateral disc edema caused by high ICP
- Bilateral disc edema is caused by paplitits ON inflammation.(without increase in ICP)
- The most common pathology in young females 20-40+ headache+N/V+transient visual obscurations+pulsatile tinnitus=> (Idiopathic intracranial hypertension, old names pseudotumor cerebri and benign Idiopathic intracranial hypertension).
- Pale ON=> Atrophy=>ON damage=> do MRI to determine the cause.

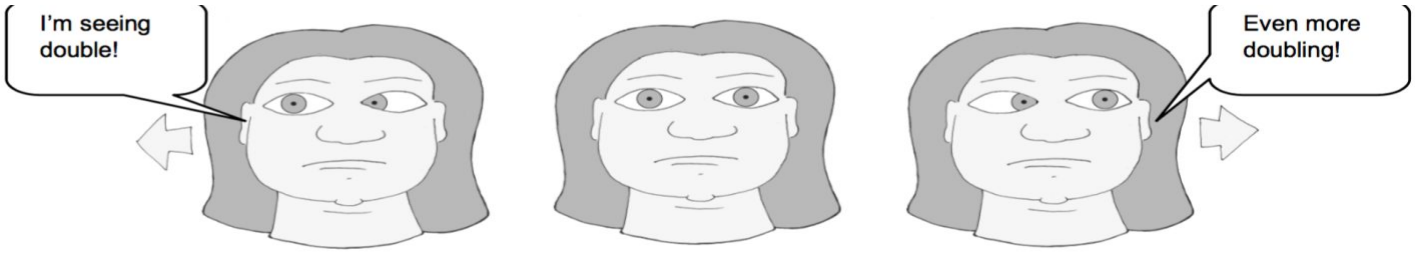
Warning!! My dear, please do not panic, the following questions are EXTRA, just to further expand and test your knowledge in neuro-ophthalmology.
If you're not interested; skip'em ;)

★Questions: (Ophthobook)

1. 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.

2. Why do diabetic patients with oculomotor paralysis have "sparing of their pupil"?
The pupil is typically spared with ischemic third nerve palsies caused by vascular problems. This is because the parasympathetic pupillary fibers run along the surface of the nerve, making them susceptible to aneurysm/tumor compression but resistant to deeper infarction.

3. This 32 year old overweight woman complains of several months of headaches, nausea, and now double vision. What cranial nerve lesion do you see in this drawing. What other findings might you expect on fundus exam and what other tests might you get?



This looks like a bilateral abducens palsy as the patient can't move either eye laterally. While the majority of abducens palsies occur secondary to ischemic events in diabetics and hypertensives, this etiology seems unlikely in a young patient with bilateral involvement. Her symptoms sound suspicious for pseudotumor cerebri (obese, headaches). You should look for papilledema of the optic nerve, get imaging, and possibly send her to neurology for a lumbar puncture with opening pressure.

4. A patient is sent to your neurology clinic with a complaint of double vision. Other than trace cataract changes, the exam seems remarkably normal with good extraocular muscle movement. On covering the left eye with your hand, the doubling remains in the right eye. What do you think is causing this diplopia? The first question you must answer with any case of diplopia is whether the doubling is monocular or binocular. This patient has a monocular diplopia. After grumbling to yourself about this inappropriate neurology referral, you should look for refractive problems in the tear film, cornea, lens, etc.

5. A patient complains of intermittent double vision that seems to be worse in the evenings. On exam you find a confusing diplopia that doesn't seem to map out to any particular nerve palsy. What else is on your differential as a cause, and what tests might you perform in the office? Myasthenia gravis and thyroid orbitopathy are both great masqueraders that cause diplopia. Graves patients often have lid retraction and reduced upgaze from inferior rectus muscle restriction. The double vision in myasthenia patients can look like an isolated nerve palsy, a mixture of nerve involvement, or may not fall into any specific nerve combination - a changing palsy is more indicative of a process like MG. You can check for fatiguable ptosis by prolonged upgaze (hold your arm up and see who gets tired first). In addition, you can perform a cold-pack rest test or even a Tensilon test.

6. 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.

7. A patient develops optic neuritis. Should you treat with steroids? Would you start with IV or oral steroids? Will the MRI findings of numerous demyelinating lesions change your management? Do you tell the patient that she will develop MS? The ONTT study has shown that steroids can speed recovery from optic neuritis, but have little effect on long-term visual outcome. Surprisingly, the study also showed that oral prednisone may actually increase reoccurrence of optic neuritis. Therefore, you give IV Solu-Medrol and don't give oral prednisone!

The presence of optic neuritis does not necessarily mean the patient will develop multiple sclerosis, especially in the setting of a negative MRI. The patient's long-term risk for developing multiple sclerosis depends upon the number of CNS lesions found on presentation. If there are no CNS lesions, then the future risk is only about 15%. This jumps up to 50% or more with 3+ lesions. In these higher-risk patients, you should get neurology involved to discuss more aggressive treatment with Avonex.

8. 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 vision- threatening causes like GCA (giant cell arteritis).

9. The previous patient admits to "not feeling good" and "it hurts my head to brush my hair on the right side" for the past week, but denies all other symptoms. Should you order any labs? Start any medications? If you have any suspicion for GCA, you pretty much have to order a ESR and CRP. Start oral prednisone (about 1mg/kg/day) immediately and set up for temporal artery biopsy within a week or so. Steroids won't help much with his lost vision in these cases, but decreases the risk to the other eye, which can be affected within hours to days.

10. A young man complains of complete vision loss (no light perception) in one eye, however, he has no afferent pupil defect. Is this possible? How might you check whether this patient is "faking it?" Assuming the rest of the eye exam is normal (i.e., the eye isn't filled with blood or other media opacity) this patient should have an afferent pupil defect if he can't see light. There are many tests to check for malingering and factitious disorders: you can try eliciting a reflexive blink by moving your fingers near the eye. One of my favorite techniques is to hold a mirror in front of the eye. A seeing eye will fixate on an object in the mirror. Gentle rocking movements of the mirror will result in a synchronous ocular movement as the eye unconsciously tracks the object in the mirror.