Acute Visual Loss

[**Color index:** Important | Notes: **F1&F2** | Extra] **EDITING FILE**

Objectives:

- Properly screen and evaluate patients presenting with acute visual loss.
- Understand the pathophysiology and identify common causes of acute visual loss.
- Recognize situations requiring urgent ophthalmic care to prevent permanent visual loss.

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Resources: Slides (Prof. Saleh) + Notes + OphthoBook + 435 Team.

***Lecture was explained by F1: Prof. Abouammoh (who is in the examination committee) and F2: Prof. Saleh Alobeidan.**

***Prof. Abouammoh refused to handout his slides. However the secretory sent the slides done by Dr.Essam & Dr. Abdullah Almousa as instructed by him.**

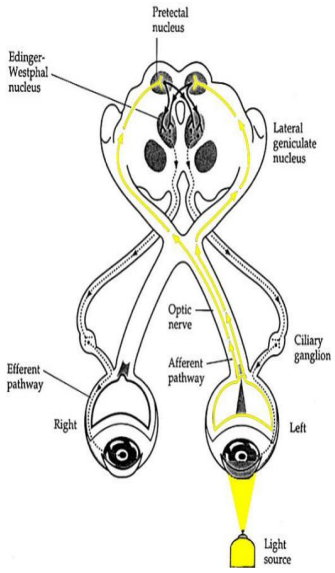
*** Special thanks to Shadn Alomran for her effort!**

Physiology

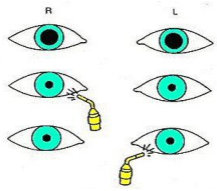
Extra from 435 team *the professor did not mention it but it is very helpful*

– Pupillary light reflex pathway

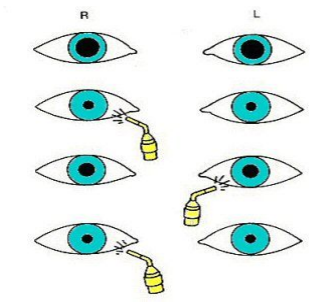
- It is very important to understand the pathway to locate the neurological pathology.
- Pupillary light reflex, which allows for the constriction of the pupil when exposed to bright light. This reflex serves to regulate the amount of light the retina receives under varying illuminations. The pupillary light reflex two main parts: an afferent limb and an efferent limb:

Afferent Pathway (yellow line)	Efferent Pathway (dotted black)	
1. Light enters the pupil and stimulates the retina. **2.** Retinal ganglion cells transmit the light signal to the optic nerve. **3.** The optic nerve enters the optic chiasm where the nasal retinal fibers cross to contralateral optic tract. while the temporal retinal fibers stay in the ipsilateral optic tract. **4.** Fibers from the optic tracts project and synapse in the pretectal nuclei in the dorsal midbrain in the collicular region. **5.** The pretectal nuclei project fibers to the ipsilateral Edinger-Westphal nuclei and also to the contralateral Edinger-Westphal nucleus via the posterior commissure.	**1.** The Edinger-Westphal nucleus projects preganglionic parasympathetic fibers, which exit the midbrain and travel along the oculomotor nerve (CN III) and then synapse on postganglionic parasympathetic fibers in the ciliary ganglion. **2.** Ciliary ganglion postganglionic parasympathetic fibers (short ciliary nerves) innervate the sphincter muscle of the pupils resulting in pupillary constriction.	

- The physiological result of the neuroanatomical pathways as described above is that light shined in one eye will result in pupillary constriction in both the ipsilateral pupil (direct pupillary light reflex) and the contralateral pupil (consensual pupillary light reflex).

Normal Pupillary Light Response (from top to bottom)	
Row 1: pupils in a dark room without light stimulation. **Row 2:** intact direct and consensual responses for right eye. Row 3: intact direct and consensual responses for left eye. From the above, we can conclude that both the afferent and efferent limbs of both eyes are intact in the above patient.	

- In the case of optic nerve dysfunction, such as in optic neuritis, as phenomenon called a relative afferent pupillary defect (RAPD) results. This is due to an impaired afferent limb of the pupillary reflex so that stimulation with light of the ipsilateral affected eye will not result in as much pupillary constriction as stimulation of the normal contralateral eye. A RAPD can be demonstrated by a test called the "swinging-flashlight test" as shown below:

Row 1: Unstimulated pupils in a dark room **Row 2:** Stimulation of the right eye produces bilateral pupillary constriction, indicating intact afferent right limb, and intact bilateral efferent limbs. **Row 3:** When moving the light source from the right to left eye, the left eye paradoxically dilates. This indicates a faulty left eye afferent limb, most likely from left optic nerve dysfunction. Note that the afferent limb of the left eye is not completely non-functional as the pupils are still more constricted then the pupils seen in the unstimulated row 1. **Row 4:** Demonstrates again that the right afferent pathway is functioning normally and that the problem is with the left eye's afferent pathway.	

Prologue

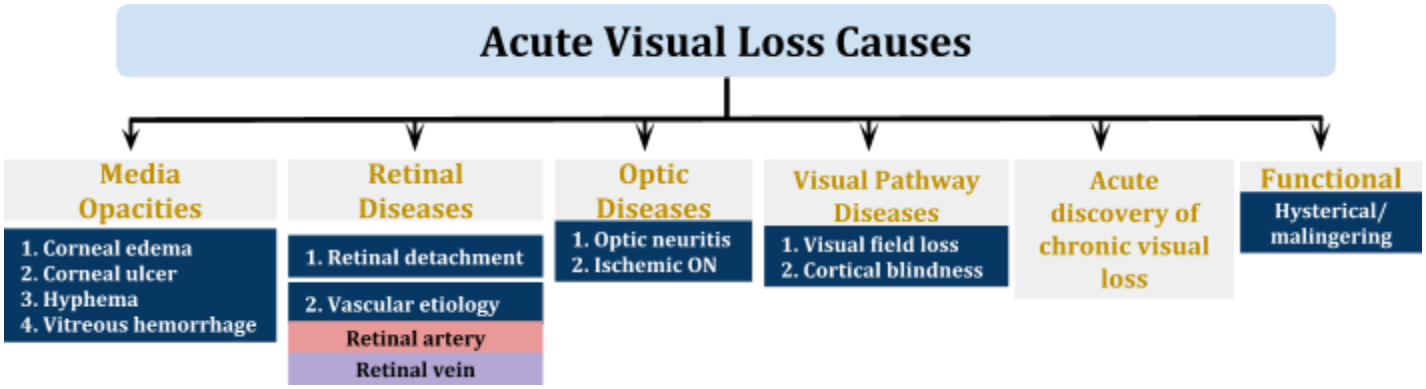
• What is acute visual loss (AVL)? (disastrous and requires urgent actions)

– Sudden onset of significant visual impairment or blindness. Loss of vision is usually considered acute if it develops within a few minutes to a couple of days.
– May affect one or both eyes | All OR part of visual field | Arise from pathology of any part of the visual pathway.

Etiology		
AVL classified by PAIN		AVL classified by STRUCTURE
Painful	Painless	1. Media opacities: something interferes with the passing of light from cornea to vitreous. usually the pathology is not within the lens as it only causes visual loss in cases of very severe trauma. 2. Retinal disease: improper absorption of light. 3. Optic nerve disease. 4. Visual pathway or neurological disorders: Stroke/tumor 5. Functional disorders 6. Acute discovery of chronic visual loss: usually **unilateral**
– **Acute (congestive) Glaucoma:** in the past they misdiagnosed it with MI due to pain severity, they presented with severe headache, drop of vision,severe eye pain,nausea and vomiting. – **Uveitis:** patient is always in pain. – **Keratitis**: infection or inflammation of cornea "very severe pain, more than uveitis". – **Hyphema (Traumatic):** can be asymptomatic unless if it's associated with other things.	– **Vitreous Hemorrhage:** it can be painful if it is traumatic. – **Retinal Detachment:** the patient may have it and not discover it until covering one eye – **Retinal vascular occlusions**:arteries/veins – **Optic neuritis:** sometimes eye movement may cause mild pain, but usually it is painless. – **Ischemic optic neuropathy** – **Cerebrovascular accident (CVA)** – **Functional**	**All of the above may cause mild, moderate, severe visual loss or total blindness.**

Clinical approach	
History (Hx)	Physical examination (P/E)
– Is the visual loss transient, persistent, or progressive? ⋆ **Transient**: Vascular, migraine (Ex: amaurosis fugax). ⋆ **Persistent** (continuous) such as Retinal detachment, hemorrhage, or optic neuritis. ○ Progressive: Not vascular, could be the progression of optic neuritis. **– Is the visual loss monocular or binocular?** ⋆ **Mononuclear** (before optic chiasm-decussation) such as optic neuritis. ⋆ **Binocular** (after optic chiasm-decussation) such as cortical blindness. Think about central causes and confirm it by pupillary reflex => it is 100% normal **– Did the visual loss occur suddenly or it developed over hours, days or weeks?** ⋆ **Sudden:** Vascular. (ischemic, central retinal artery occlusion) ○ **Hours:** Acute angle closure glaucoma. ⋆ **Days-Weeks:** Optic neuritis and Retinal detachment. **– What is the patient's age and general medical condition?** ○ Young with no systemic disease:think about neurological problems like: Optic neuritis, retinal detachment or trauma. ○ Old with chronic medical condition: Vascular cause. ⋆ Acute glaucoma vs corneal abrasion. **– Did the patient have normal vision in the past and when was vision last tested?** Some people will only realize loss of vision from one eye; when they cover the good eye **– Was pain associated with visual loss?** **– Contact lens use?** corneal ulcer	**1. Visual acuity testing:** to see if the visual loss is mild, moderate, or severe. **2. Confrontation visual fields test:** it is useful if there is a pathology in the distal part of visual pathway if it is suspected in the history, so it is useful in neurological deficit. **3. Pupillary reactions (very important).** **4. External examination of the eye with a pen light:** we look at the eye in general to see if there's any trauma. **5. Biomicroscopic examination (Slit lamp examination)** **6. Ophthalmoscopy exam:** can exclude media opacity, we observe the red reflex, in normal people it is present and equal in both eyes. **7. Tonometry to measure the intraocular pressure**

This lecture was brought to you by:
Badly stored contacts!
Alright, let's get to the real stuff. Onward.

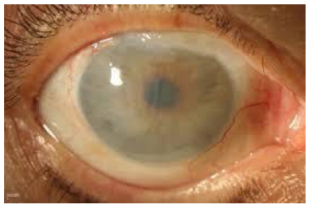
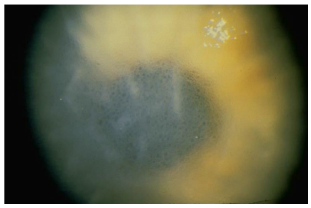


Media Opacities

Corneal opacity is either due to edema or infection (like uveitis) or trauma corneal ulcer.

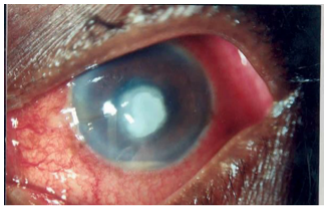
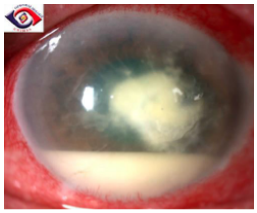
1. Corneal edema

– The cornea appears like **ground glass** rather than its normal clear appearance. (steamy cornea)
– The most common cause of corneal edema is **increased intraocular pressure** typically in **acute angle closure glaucoma** (this is almost always the presentation of corneal edema) so why does it cause edema? that is because high intraocular pressure interferes with the function of the endothelium which is bundling the aqueous humor from the stromal cells to detergent the cornea. This is true for abnormal ocular pressure of any cause!
– Other causes of corneal edema include severe ocular hypotony[1]
– Any acute infection of the cornea resulting in a corneal ulcer may **mimic** corneal edema.

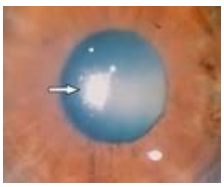
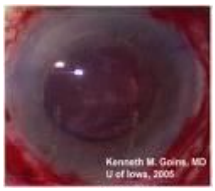
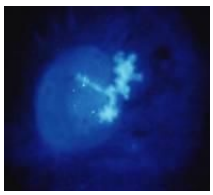
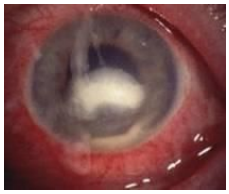
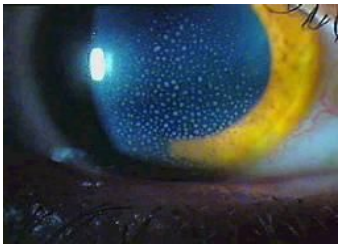
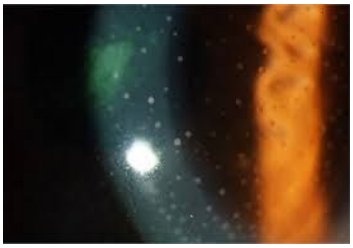
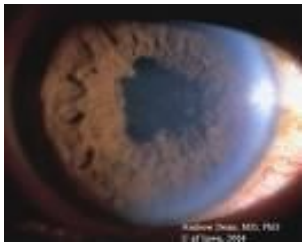
2. Corneal ulcer

– When there is a corneal opacity due to destruction of tissue by infiltration of microorganisms and WBCs.
– Could be viral, bacterial, fungal, protozoal or neurotrophic in etiology

[1] **Hypotony** is usually defined as an intraocular pressure (IOP) of 5 mm Hg or less.

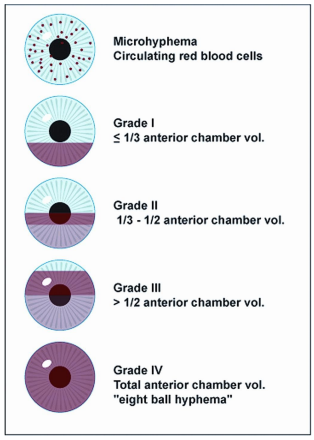
– Uveitis doesn't only cause visual impairment on the corneal side but also on the turbidity of the anterior chamber. In uveitis the inflammation leads to changes in aqueous humor contacts, usually there is a protein present in the anterior chamber and its concentration is 1% of that in the serum. In severe uveitis, the concentration is similar to the serum.
– Posterior synechiae[2] itself doesn't cause visual loss, but the sequences that happen after posterior synechiae.

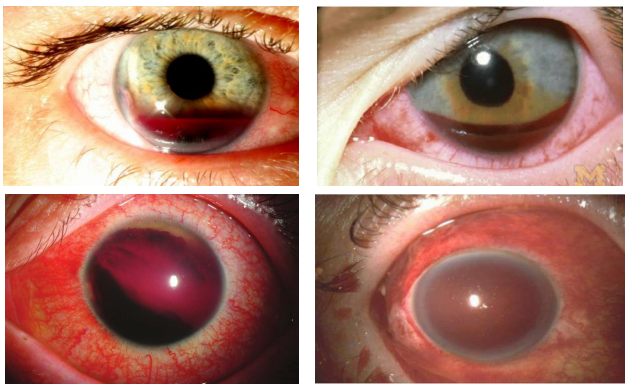
			
Abscess	**Edema**	**Viral herpetic herpes**	**1) Infection: abscess in the cornea with hypopyon[3].** 2) **Congested eye.**
			

Uveitis: Mutton fat keratic precipitates appearance due to accumulation of WBC "mainly macrophages" on the corneal endothelium, resembles edema

3. Hyphema

– Hyphema is blood in the anterior chamber.
– The hyphema is a direct consequence of blunt trauma to a normal eye.However, it can occur with tumors, diabetes, intraocular surgery and chronic inflammation which all cause neovascularization.
– The most common cause is trauma. In case of trauma, it usually resolves spontaneously within 3 days (Bed Rest and minimize the activity to avoid repleading).
– If it's not resolved and the pressure is high it may cause corneal blood staining, which would take years to clear. This will affect the vision dramatically.
– It may need evacuation in sickle cell patient, to avoid vascular accidents "There is high IOP and the deformed RBCs can't pass through the trabecular meshwork"





[2] **Synechiae** are adhesions that are formed between adjacent structures within the eye usually as a result of inflammation.
[3] A collection of inflammatory cells-puss- in the anterior chamber.

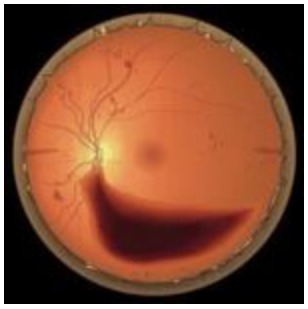
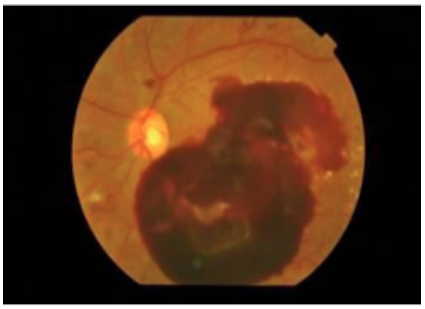
4. Vitreous hemorrhage (VH)

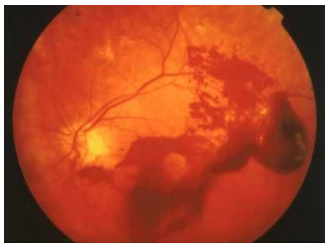
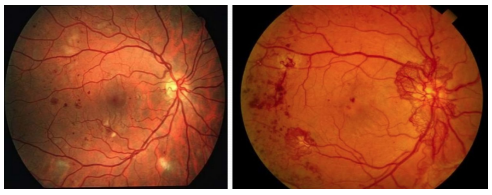
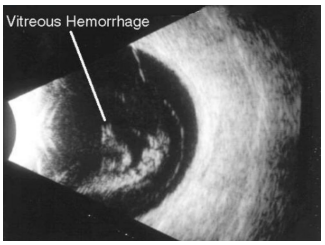
Mechanism

– Extravasation or leakage of blood into the areas in and around the vitreous humor of the eye.
– Any bleeding into vitreous cavity will reduce visual acuity. (because it's a media opacity: opacity in the cornea? Edema. Opacity in the vitreous? Blood. Opacity of lens? Cataract)
– **Can result from:** trauma, diabetic retinopathy (common cause in ksa), retinal vascular occlusion and acute posterior vitreous detachment and intraocular surgery. Many diseases can cause you VH, even TB can cause VH
– Rarely, can accompany subarachnoid hemorrhage. (Terson's syndrome)
In summary, mechanisms of Vitreous Hemorrhage:
A. Abnormal Vessels: DM (accounts for 30-50% of VH), Sickle cell retinopathy, etc
B. Rupture of normal vessels: Retinal tear, trauma, Terson's…
C. Blood from adjacent source: Macroaneurysm (0.6–7%) Age-related macular degeneration (0.6–4%)

Diagnosis

– **Not a diagnosis but rather a sign for many diseases.**
– **First step:** ophthalmoscope. If you cannot appreciate a RED REFLEX → **Second step:** B scan (ultrasound).
– If you cannot appreciate a red reflex with an **ophthalmoscope** and the lens appears clear, you should suspect VH!
– The diagnosis is confirmed with slit lamp examination through a dilated pupil. So you know the lens is definitely clear, further you stop seeing anything inside the vitreous cavity
– **B scan (ultrasound) is important.** (important to know the etiology, it tells you how thick the blood & distribution)

Nonproliferative Diabetic Retinopathy	Proliferative Diabetic	B scan
		

Treatment

Bed rest and treatment of the underlying cause.

Acute angle-closure glaucoma (AACG)

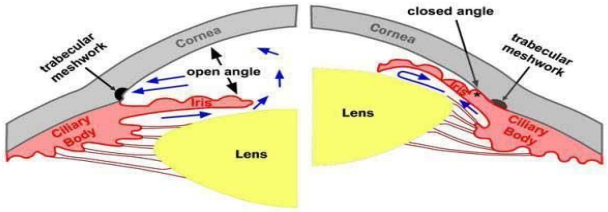
Features: patients who are **prone** to develop acute angle closure glaucoma have unique features:

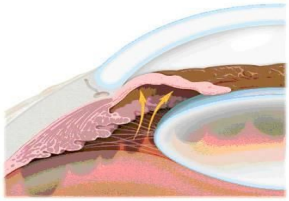
Shorter eyes "Axial length" (within the normal range). | Hyperopic vision. | Large lens.

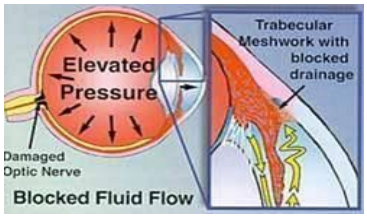
Mechanism

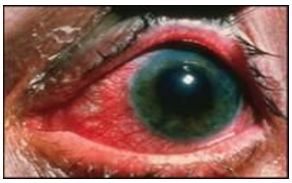
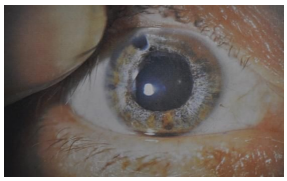
The most common mechanism is **pupillary block:** This occurs when the lens plasters up against the back of the iris, blocking aqueous flow through the pupil. **This resistance produces a pressure gradient IOP goes up** (this is the keyword) across the iris that forces the iris and lens to move anteriorly and in turn the **irido-corneal angle closes (angle block)**, blocking the trabecular meshwork. Without an exit pathway, aqueous fluid builds up, eye pressure increases rapidly, and the optic nerve is damaged from stretching and decreased blood supply. This sequence of events can occur for many reasons, commonly in people with naturally shallow anterior chambers such as hyperopes (far-sighted people with small eyes) When the iris dilates, the iris muscle gets thicker and the irido-corneal angle becomes smaller, making it more likely to spontaneously close. Along those lines, medications that dilate the eye, such as over-the-counter antihistamines and cold medications, also predispose angle closure.

In other words, With aging, the space between the iris and the lens become narrower, until it reaches the point where the aqueous fluid becomes trapped in the posterior chamber. The fluid push the iris anteriorly and closes the trabecular meshwork "the angle". The iris sphincter muscle will be ischemic, causing a mid-dilated fixed non-reacting pupil.







Accumulation of aqueous fluid behind the cornea	Mid-dilated fixed pupil, inflammation, and corneal edema	Atrophic Iris because the changes are irreversible, even after treatment. However, this depends on the severity of the disease.
		

Presentation

Severe pain and redness (high pressure compresses nerve ends in cornea), Sluggish, non-reactive mid-dilated pupil (sphincter ischemia), increased IOP (often 60 mm Hg or higher) Blurry vision (due to corneal edema). patients also often describe seeing halos around lights, Headache, Nausea and vomiting.

Management "medical emergency"

– **Aim:** Decrease IOP, Prevent future attacks in OU[4]. "Prophylactic laser to the other eye, **VERY IMPORTANT!** because the other eye is likely to also have AACG so we don't wait, high IOP can damage the optic nerve within hours.

– Decrease the pressure by medications[5] > laser iridotomy "in the outpatient clinic". This will deflate the iris and open an alternative pathway for the aqueous. If it's not treated, it will cause fibrosis and the laser doesn't help anymore.

[4] **Oculus uterque** (OU) Latin term for "each eye," used in vision correction prescriptions to indicate both eyes.

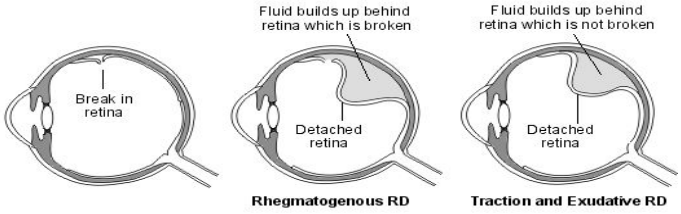
[5] Acetazolamide is administered intravenously and subsequently orally together with topical pilocarpine and beta-blockers. Pilocarpine constricts the pupil and draws the peripheral iris out of the angle; the acetazolamide and beta-blocker reduce aqueous secretion and the pressure across the iris. These measures usually break the attack and lower intraocular pressure.

Retinal Diseases

1. Retinal detachment (RD)

– An abnormal separation between the sensory retina and the underlying retinal pigmented epithelium (RPE) and choroid plexus. the outer third (the part furthest from the inner vitreous) of the retina gets its nourishment primarily from the underlying choroidal vascular bed. With a detachment, the photoreceptor layer separates from the choroid, and without this blood supply becomes ischemic.
– In normal retina, there is no actual connection or junction between them. It is a potential space, firm, and adherent.
– When the retina breaks, fluid come between the 2 layers and separates them.
– Retinal detachment is one of the painless causes of acute visual loss, and it is not an ocular emergency.
– It will cause sudden or acute visual loss if it was in the macula, but macular involvement takes time, so the pathophysiology is chronic but the visual loss will be acute.

Types

1. Rhegmatogenous RD	2. Traction RD	3. Exudative RD
– Most common – Rhegma = break. Tear in the retina allowing liquified vitreous to gain entry to the subretinal space and causing a progressive detachment.	– In Diabetes, SCA. – Here the the retina is detached but continuous with no tear. If it is pulled off by contracting fibrous tissue on the retinal surface.	– Due to inflammation, if we treat the underlying pathology the problem will be solved. (forget it. We treat systematically)
		– Picture: when a break happens, subretinal space allowed vitreous fluid to travel into the break and detach the retina (as a result of an exudative process)

Risk factors

– Posterior Vitreous Detachment (PVD) the vitreous is attached to the eye at the optic head and ora serrata. Due to trauma, surgery, or spontaneous liquefaction "aging", the vitreous detaches and pull the retina and break it.
– Peripheral retinal degenerations. e.g. lattice degeneration, retinal tufts... etc.
– **High myopia.**
– Aphakia: (no lens. In the past they used to deal with cataract aggressively, traumatic surgeries). (bc its a sort of trauma, its abnormal, there should be a lens inside: pseudophakia > less risk of RD)
– History of: trauma, retinal detachment, also previous history of detachment in the other eye, Keratoconus.
– Family history,
– Exudative RD can happen in: renal biliary pts, Vogt–Koyanagi–Harada syndrome, abnormal liver functions, etc
– usually old age.

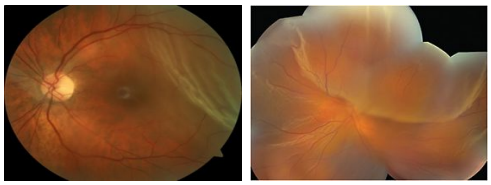
Signs & Symptoms

1. Prodromal symptoms: **flashes + floater**s (if you catch the pt here you can prevent RD. but patient almost never come at this point)
2. **Visual field loss- curtain-like** (from below: upper retina, and vice versa)
3. Sudden, painless loss of vision
4. **Afferent pupillary defect.**

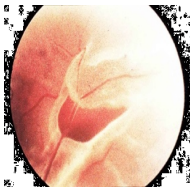
– The diagnosis is confirmed by ophthalmoscopy through a dilated pupil, and retina appears elevated with folds and the choroid background behind the retina is indistinct.

Management

– RD does not need urgent management, unless if its **macula ON.**
– In management, there are two types based on the status of the macula, macula on and off.
  - **Macula on: the macula is still attached, and the intervention is required within 24 hours** (Because central visual acuity is still preserved).
  - **Macula off: The macula is detached, and intervention is less critical (within 10 days).**
– Scleral buckle, cryotherapy, SRF drainage, Vitrectomy, AFX, endolaser, long acting tamponade (Gas, silicone oil)
(You don't need to know the treatment details)



Traction:
There is flat retina and the bulging part is detached (if the macula is not involved you can treat this surgically)

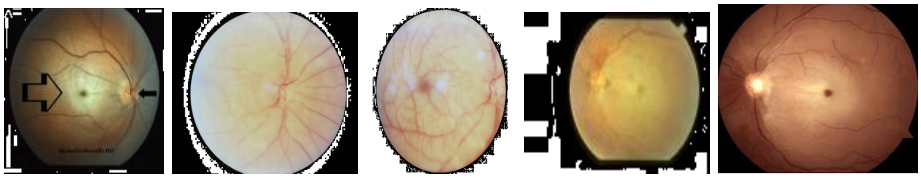


Horseshoe break:
f the break gets pulled more (by the vitreous) → can rupture a vessel → RD & hemorrhage

2. Retinal vascular occlusions

Artery

Central Retinal Artery occlusion

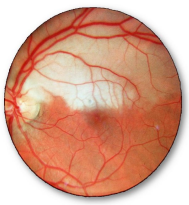


– A sudden, **painless** and often complete visual loss may indicate central retinal artery occlusion.
– Several hours after a central retinal artery occlusion, **the inner layer of the retina becomes opalescent (opaque, why? Bc of edema).**
– A cherry red spot is seen due to the pallor of the perifoveal retina in contrast to the normal color of the fovea. (in picture 1, there is an opaque retina with an edematous disc (small arrow) and then the big arrow is pointing at the macula, the dark area is a cherry red spot of the fovea in the center of the macula. Which means: central retinal artery occlusion)
– A **chronic cherry red spot (big arrow)** is also a feature of the storage diseases such as Tay-Sachs disease and Niemann-Pick disease.
– There is no generally accepted acute management.

Causes of cherry red spot on retina: *mnemonic: **C**herry **T**ree **N**ever **G**row **T**all*
Central retinal artery occlusion | **T**ay-Sachs disease | **N**iemann-Pick disease | **G**aucher's disease | **T**rauma (Berlin edema)

Branch Artery occlusion

– If only a branch of the central retinal artery is occluded, vision is only partially lost. (refer for VF)
– This is more likely to be the result of an emboli and the source of the emboli should be sought.
– If the visual acuity is affected, attempts should be made to dislodge the emboli by ocular massage
Basically for both branch and central occlusion you try to cause vasodilation, you can try to make the pt rebreathe through a bag so CO2 > vasodilation, but it does not always work > no treatment

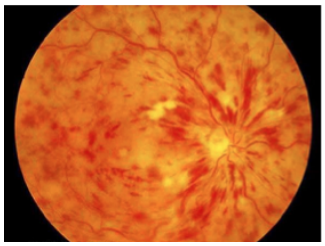


***Picture:** notice how white the affected retina (above) is compared to the normal retina. The ischemia stopped just short of the macula so this patient most likely didn't have his vision completely affected (but his lower visual field is gone)
Upper retina: lower. Lower retina: Upper Temporal: nasal. Nasal: temporal

Vein

Cental retinal vein occlusion

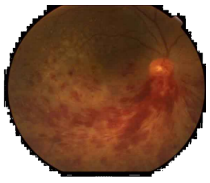
– Ophthalmoscopes picture of disc swelling, venous engorgement and tortuosity, **cotton wool spots (these are more white than exudates, and they are an infarctions of the nerve fiber layer) and diffuse retinal hemorrhages** like blood and thunder (flame shaped).
– Loss of vision may be severe. Bc it causes macular edema.
– Treatment should be directed at reducing associated macular edema by injecting anti-vascular endothelial growth factor agents "Anti-VEGF". CRVO is not true ophthalmic emergency.
– Visual prognosis depend on degree of associated retinal ischemia.



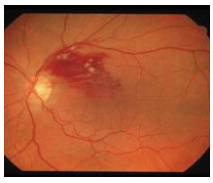
Types

Ischemic	Non ischemic
– It is a disaster that will lead to Permanent visual loss. – It will cause neovascularization which leads to "90-days glaucoma". Explanation: Ischemia causes reduction of oxygen supply => leading to VEGF production " which promotes new blood vessels formation" => Ultimately leads to formation of fibrovascular membranes => The fibrovascular membranes accompany neovascularization and block the trabecular meshwork => Causing glaucoma "Neovascular glaucoma", typically named 90-days glaucoma because it usually takes around 90 days to occur after the onset.	May resolve fully (benign). However, in 50% of the cases it may turn to ischemic. Non-ischemic if there is no hemorrhage the patient will be unaware of it.
Relative afferent pupillary defect **(RAPD +)**	**Intact pupillary reflex**

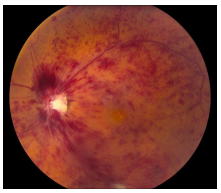
The way to differentiate between the two types is by visual acuity, and pupillary reflex, in ischemic type there must be an afferent defect.



Hemiretinal vein occlusion
/engorged veins/ cotton wool spots/ disc edema



Branch vein occlusion
Veins appear thicker than arteries.



Central vein occlusion
(flame shaped hemorrhage in all quadrants) the disc is not swollen here

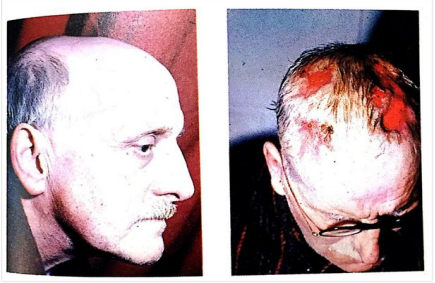
Optic Nerve Diseases

1. Optic neuritis:

– Optic neuritis is an inflammation of the optic nerve and It is usually idiopathic but may associated with **multiple sclerosis** (as first clinical manifestation.) in a number of cases.
– Visual acuity is markedly reduced **and an afferent pupillary defect is present. (+)**
– Associated with pain on extraocular muscle movement in 90% of patients
– The optic disc initially appears hyperemic and swollen.
– The visual acuity usually recovers. However, repeated episodes of optic neuritis may lead to permanent loss of vision. (so the goal of management is to prevent recurrence)
– It has three types: Optic papillitis (Optic nerve head is involved), retrobulbar neuritis (the posterior part of the nerve is involved), or neuroretinitis (Optic nerve head with contagious retinal inflammation).
– Most common type is retrobulbar neuritis. Here, the fundus looks normal but the vision is severely affected with central visual fields defect (most common presentation).
– Most of the time It is reversible with return of normal vision within 4-6 weeks (self-limiting).
– But if one eye only is affected you may use steroids to enhance the recovery(speed it up)

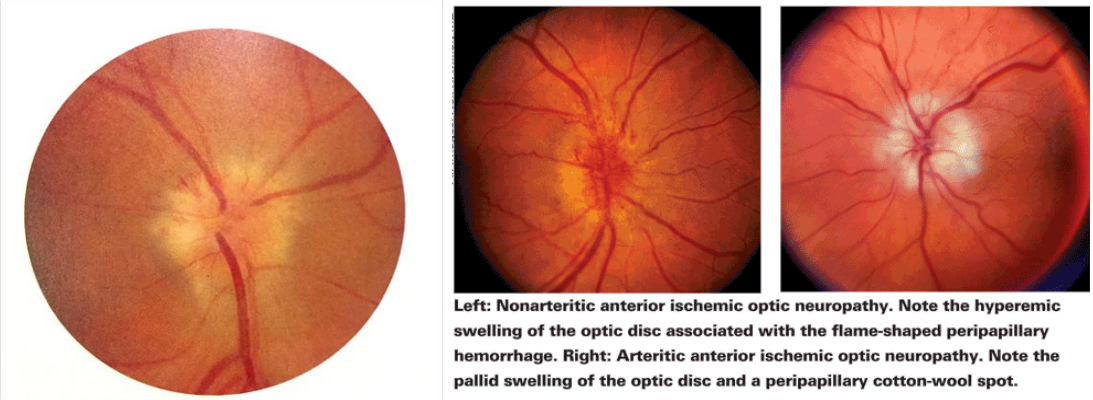
	Extra information: difference between		
	Papilledema	**Papillitis**	**Retrobulbar neuritis**
Definition	Swelling of optic nerve head due to increased ICP	Inflammation or infarction of optic nerve head	Inflammation of orbital portion of optic nerve
Uni/bilateral	Bilateral	Unilateral	Unilateral
Vision impairment	Enlarged blind spot	Central/paracentral scotoma to complete blindness	Central/paracentral scotoma to complete blindness
Fundus appearance	Hyperemic disk	Hyperemic disk	Normal
Vessel appearance	Engorged, tortuous veins	Engorged vessels	Normal
Hemorrhages?	Around disk, not periphery	Hemorrhages near or on optic head	Normal
Pupillary light reflex	Not affected	Depressed	Depressed
Treatment	Normalize ICP	Corticosteroids if cause known	Corticosteroids with caution

2. Ischemic optic neuropathy



– Anterior ischemic optic neuropathy [AION] is a relatively common cause of severe visual loss.
– The basic lesion is a segmental or generalized infarction of the anterior part of the optic nerve caused by occlusion of the short posterior ciliary arteries.
– Irreversible **painless** visual loss.
– It has two types: Arteritic and non arteritic.

Arteric	Non arteritic
– The loss of vision is due to inflammation of the arteries. – Caused by Giant-cell arteritis "Temporal arteritis". – Causes headache and gangrene of the scalp. On physical examination there is tenderness over the temporal area. – Investigation: ESR and C-reactive protein "if both are elevated => highly suggestive". The gold standard is biopsy. – Treatment is possible if you catch the patient early => Give steroids.	– Due to non-inflammatory disease of the small blood vessels. – Common cause is atherosclerosis. – There is no treatment.



Left: Nonarteritic anterior ischemic optic neuropathy. Note the hyperemic swelling of the optic disc associated with the flame-shaped peripapillary hemorrhage. Right: Arteritic anterior ischemic optic neuropathy. Note the pallid swelling of the optic disc and a peripapillary cotton-wool spot.

Visual Pathway Disorders

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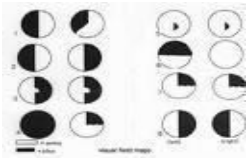
1. Homonymous hemianopia



– Loss of vision on one side of both visual fields
– May result from occlusion of one of **posterior cerebral arteries** with infarction of the **occipital lobe.**
– Other vascular abnormalities occurring in the middle cerebral artery distribution may produce a hemianopia, but usually other neurological signs are prominent. (like in stroke)
– **Any patient with hemianopia needs a CT or MRI to localize & identify the cause.** Refer to neurology
– Behind the optic chiasm.

2. Cortical Blindness

– A rare bilateral extensive damage to the cortical visual pathways results in **complete loss of Vision**.
– This condition is referred to as cortical, central or cerebral blindness.
– As the pathways serving the pupillary light reflex separate from those carrying visual information at the level of the optic tracts, a patient who is cortically blind has normal pupillary reactions. Thus a patient with normal fundus examination along with normal pupillary reactions, most likely has cortical blindness.
– Poor vision, loss depends on which part of the cortex was affected.

Functional Visual Loss (FVL)

– Describes vision loss due to hysterical or malingering reasons. ie: not explained by organic basis.
– A patient may report complete blindness in one eye and normal vision in the other eye, and no relative afferent pupillary defect (RAPD)
– Various techniques exist to confirm functional visual loss.

Diabetic Retinopathy

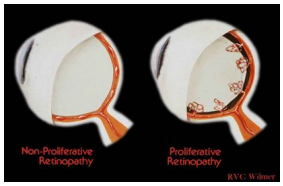
The doctor explained this briefly but said it's important, I tried to simplify it to the best of my abilities

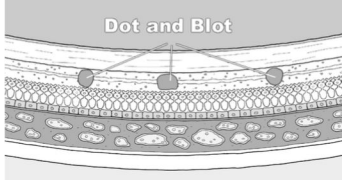
– Diabetic retinopathy is the term used to describe the retinal damage causing this visual loss. Diabetics have a high prevalence of retinopathy, and one out of every five patients with newly diagnosed diabetes will also show signs of retinopathy on exam.

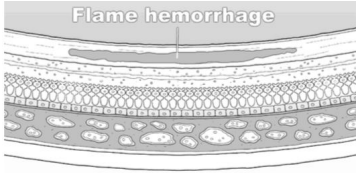
– **Mechanism of Vessel Breakdown**

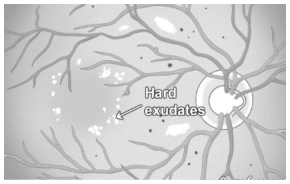
- Diabetes is a disease of blood vessels. With large amounts of glucose coursing through the circulatory system, a glycosylation reaction occurs between sugar and the proteins that make up blood vessel walls. Over time, this reaction promotes denatures the collagen protein within the walls, creating capillary thickening and eventually, wall breakdown.
- While this process occurs throughout the entire body, the microvasculature of certain organs, such as the kidneys and eyes, are more susceptible to damage. Along these lines, a good predictor of microvascular damage in the diabetic eye is prior evidence of renal microvascular disease as measured by proteinuria, elevated BUN, and creatinine.
- Because vessel damage accumulates over time, the most accurate predictor of retinopathy is duration of diabetes. After 10 years, more than half of patients will show signs of retinopathy, and after 15 years this number increases to nearly 90%. The relative control of glucose during this time is also important, and studies have shown that patients who maintain lower hemoglobin A1C levels have delayed onset and slower progression of eye disease.

Types	
Nonproliferative diabetic retinopathy (NPDR)	**Proliferative diabetic retinopathy (PDR)**
– Most patients (95%) have NPDR. This is the earliest stage of retinopathy and it progresses slowly. Because so many diabetic patients have NPDR, this stage is commonly described as "background retinopathy." The earliest signs of retinal damage arise from capillary wall breakdown, seen on the fundus exam as vessel microaneurysms. Injured capillaries can leak fluid into the retina and the aneurysms themselves can burst, forming **"dot-and-blot hemorrhages."** – Dot-blot hemorrhages look small and round because they occur in the deep, longitudinally-oriented cell layers of the retina. This contrasts with the **"flame hemorrhages"** of hypertension that occur within the superficial ganglion nerve layer, and thus spread horizontally. – **Recall the vascular section of this lecture:** With worsening retinopathy and vessel damage, the retina begins to show early signs of ischemia. **Cotton-wool spots**, indicate ischemia/infarction of the superficial retinal nerve fibers. As vessel damage progresses, you can also see beading of the larger retinal veins and other vascular anomalies.	– With ongoing injury to the retinal vasculature, eventually the vessels occlude entirely, shutting down all blood supply to areas of the retina. In response, the ischemic retina sends out chemicals that stimulate growth of new vessels (fragile and easy to bleed). This new vessel growth is called neovascularization, and is the defining characteristic of proliferative retinopathy. Far fewer patients have proliferative retinopathy, which is fortunate as this stage can advance rapidly with half of these patients going blind within five years if left untreated. – The most common cause of blindness in diabetic patients is from macular edema. Macular edema occurs in about 10% of patients with diabetic retinopathy and is more common with severe retinopathy. On exam the macula looks mildly elevated, and you can see past evidence of edema in the form of yellow- colored "hard exudates." These exudates are fatty lipids that are left behind after past macular swelling subsides.







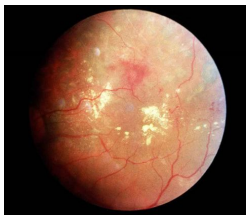


– **Retinal detachment in DM: i**n the areas where the neovascularization happens, fibroproliferation ensues and pulls on the retina.

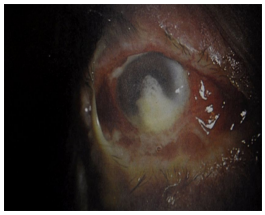
– **Treatment of DR (diabetic retinopathy)**

- Preventative medicine with tighter control of glucose is the ideal treatment, but for worsening symptoms, surgical treatment may be necessary.
- The two main surgeries are laser treatment and vitrectomy. DON'T laser the macula tho! (duh) Just laser them and the neovascularizations will involute and go away.

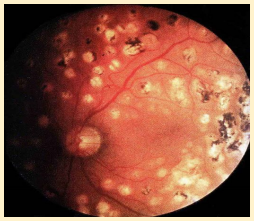
– **Complications:** visual field loss (tunnel vision)



Hard exudates



Fibrous tissue (Fibrous component comes with neovascularization)



SAQ:
Laser scars following diabetic retinopathy treatment

Questions

What are some common causes of acute visual loss based on patient's age ? (from Dr. Almousa slides)

1. Wet age-related macular degeneration.
2. Commotio retinae[6]
3. Rupture globe.
4. Orbital cellulitis.

MCQ: A 69-year-old woman presents with acute onset of ocular pain, decreased vision, and halos around lights in the right eye associated with nausea and vomiting. The most likely diagnosis is:

a. Primary open-angle glaucoma.
b. Lens induced glaucoma.
c. Pigmentary glaucoma.
d. Acute primary angle-closure glaucoma.

Answer: d

MCQ: A 30 -year-old woman presents with sudden vision loss of the right eye and mild pain on upgaze movement. Examination reveals that vision is 20/50 on the right and 20/20 on the left. There is a +RAPD on the right and a Visual field testing showed an inferior altitudinal defect on the same side. The left side is normal. Optic discs and fundi are normal in both eyes. What is the most likely diagnosis?

a. Branch retinal vein occlusion.
b. Anterior ischemic optic neuropathy.
c. Retrobulbar optic neuritis.
d. Compressive optic neuropathy.

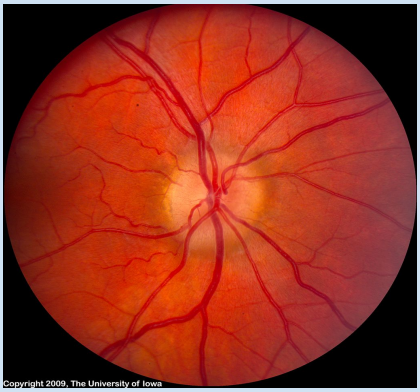
Answer: c

[6]The term describes the damage to the outer retinal layers caused by shock waves that traverse the eye from the site of impact following blunt trauma

Identify Pictures

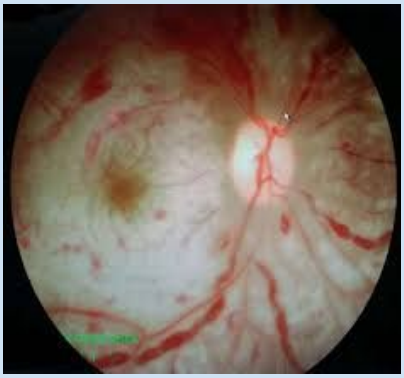
– **What is this?**
Leukocoria in a child

– **What could it be?**
– **DDx for peds leukocoria can be summed up in PREDICT**
Persistent hyperplastic primary vitreous
Retinoblastoma / **R**etinopathy of prematurity
Endophthalmitis
Dysplasia of the retina
Inflammatory cyclitic membrane
Congenital cataract / **C**oat's disease
Toxocariasis

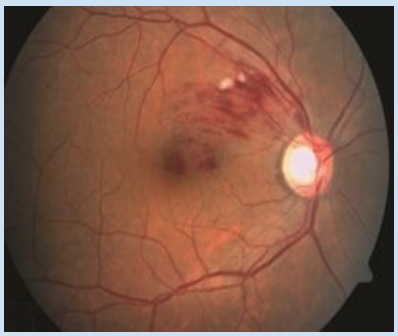


– **What is this?**
Disc edema

– **If it's bilateral it's called?**
Papilledema

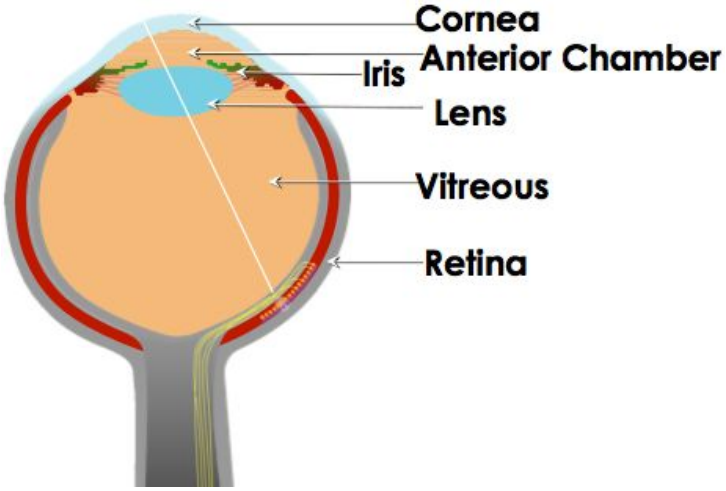


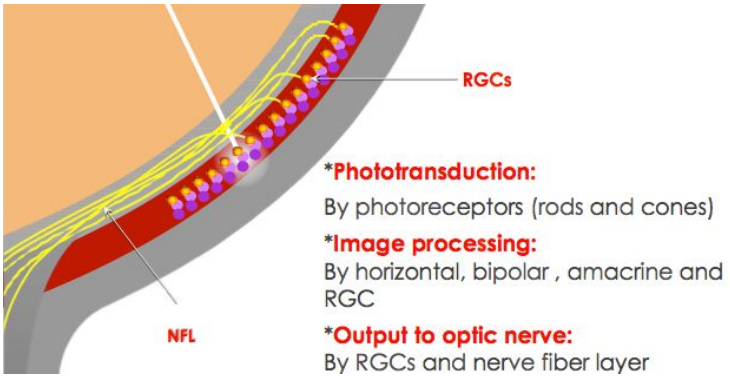
– Concurrent Central Retinal Artery and Vein Occlusion
– You can see a pale retina, a cherry red spot (artery)
– Cotton wool spots, flame shaped hemorrhage (vein) and if you follow the artery you can see points of occlusion

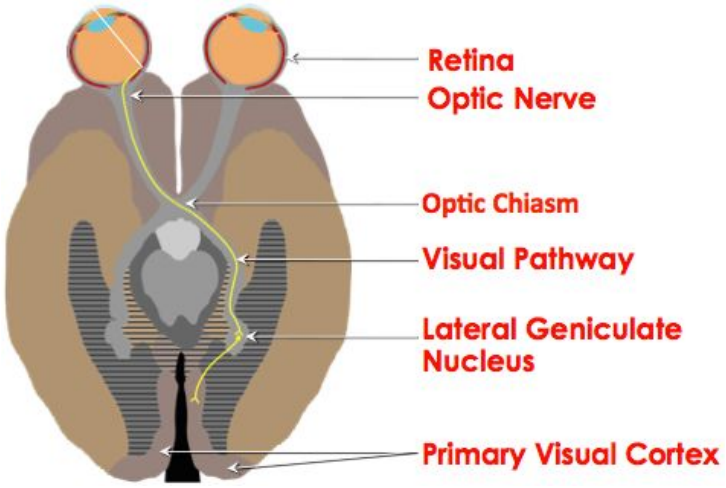


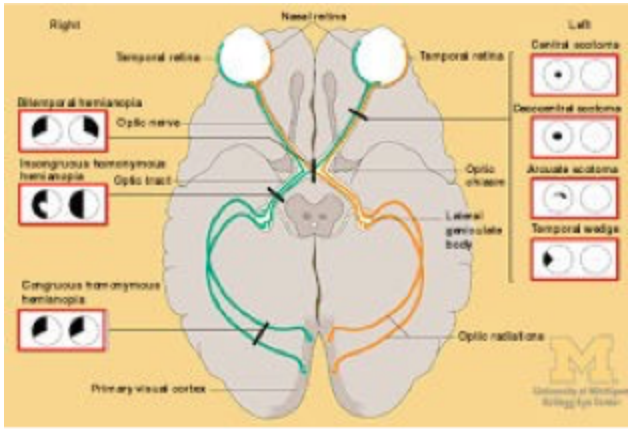
– Branch retinal vein occlusion (the whitish spots are cotton wool spots)

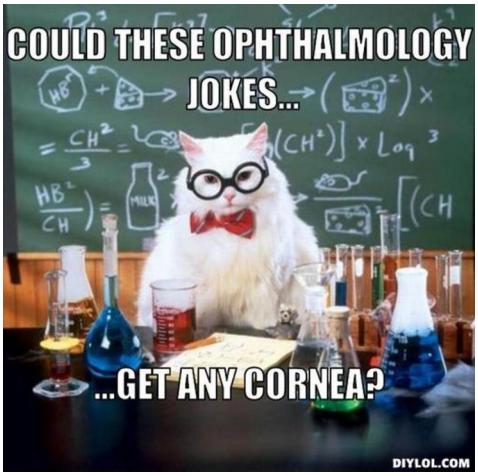
Extra from Dr. Almousa slides *the professor did not mention it but it is very helpful*











Aaaand we're done. Pat yourself on the back..