Acute Visual Loss

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.**

[Color index : Important | Notes | Extra]

Resources: Slides + Doctor's notes
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Definition:

- 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.
  - It may affect one or both eyes.
  - All or part of the visual field.
  - Arise from pathology of any part of the visual pathway

A disaster and you should be able to evaluate such a patient and be able to recognize situations requiring an urgent action and know how to treat it.

Acute Visual Loss:

1. Media opacities (Something interferes with the passing of light. From cornea to vitreous, usually the lens don't cause acute visual loss unless if there was severe trauma).
2. Retinal disease (Improper absorption of light).
3. Optic nerve disease
4. Visual pathway or neurological disorders (optic nerve, optic chiasm, optic tract, radiation,...)
5. Functional disorders
6. Acute discovery of chronic visual loss[1]
7. vascular diseases

- All of the above may cause mild, moderate, severe visual loss or total blindness.

[1] Ex: 50 years old patient examined his eyes 5 years ago, after 5 years he discover that he has left eye blindness due to cataract.

History taking:

1. **Is the visual loss transient, persistent, or progressive?**
   - Transient (sec to min): Vascular (Ex: amaurosis fugax).
   - Persistent (continuous) such as Retinal detachment, hemorrhage, or optic neuritis.
   - Progressive: Not vascular, could be the progression of optic neuritis.
2. **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.
   - Binocular: Think about central causes and confirm it by pupillary reflex => it is 100% normal
3. **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.
4. **What is the patient's age and general medical condition?**
   - Young with no systemic disease:think about neurological problems like: Optic neuritis, or retinal detachment or trauma.
   - Old with chronic medical condition: Vascular cause.
5. **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

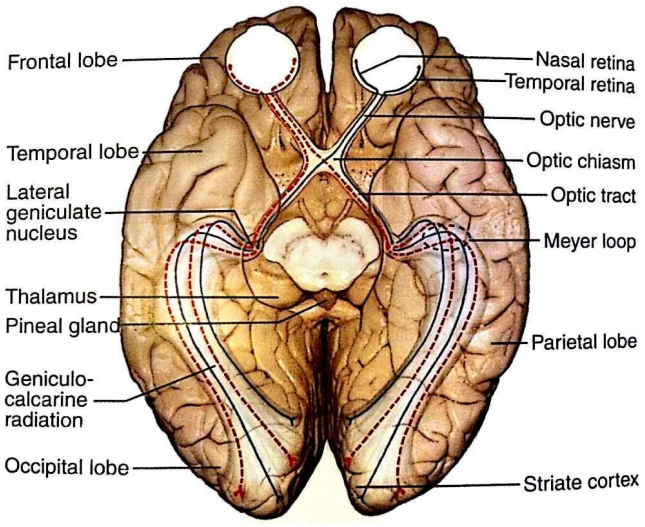
6. **Was pain associated with visual loss?**

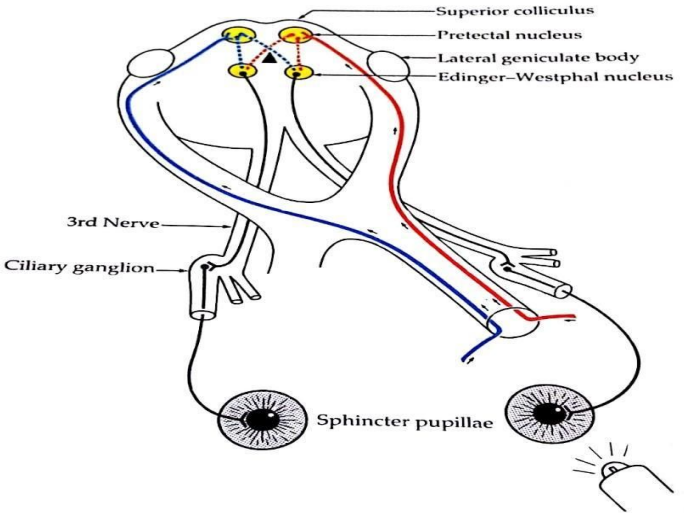
can separate most causes of avl to painful and painless, painful include Acute Angle Glaucoma and infections and trauma.

Examination

- Visual acuity testing(to see if the visual loss is mild, moderate, or severe).
- 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).
- Pupillary reactions(very important).
- Ophthalmoscopy exam (can exclude media opacity, we observe the red reflex, in normal people it is present and equal in both eyes).
  - External examination of the eye with a pen light(we will look at the eye in general to see if there's any trauma).
  - Biomicroscopic examination (Slit lamp examination)
- Tonometry to measure the intraocular pressure

Pupillary light reflex pathway (VERY IMPORTANT)1

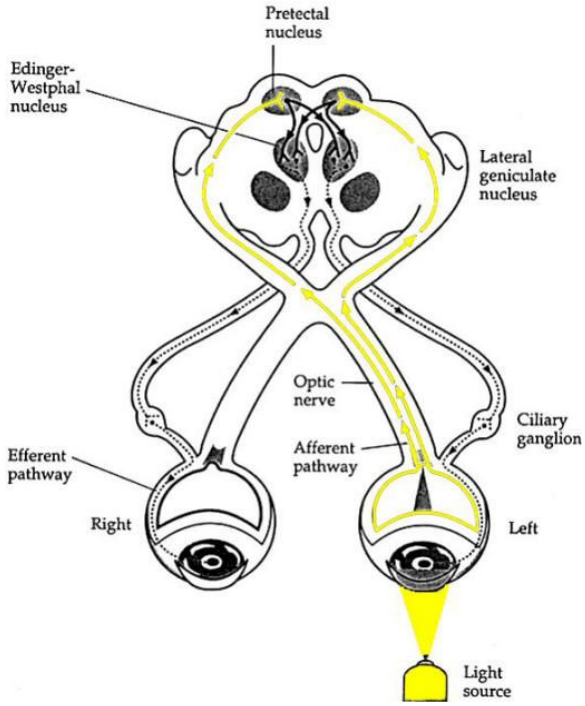




for better understanding here's the physiology of pupillary light reflex(it is very important to understand it cause it will help us locate the neurological pathology incase there was an abnormality).

(في شرحين: الأول من موقع كايسميد و الثاني الازرق شرح الدكتور, تختاروا اللي يريحكم)

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**.



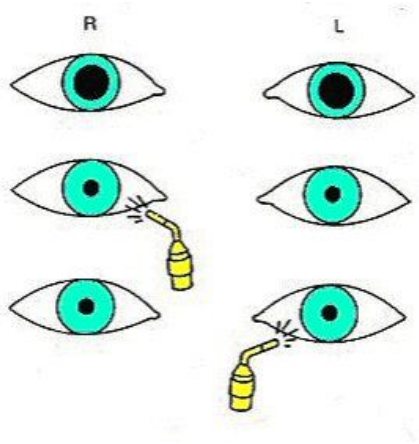
Afferent Pathway of Pupillary Light Reflex (**yellow line**):

- Light enters the **pupil** and stimulates the **retina**.
- Retinal ganglion cells transmit the light signal to the **optic nerve**
- 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
- Fibers from the optic tracts project and synapse in the **pretectal nuclei** in the dorsal midbrain in the collicular region
- The pretectal nuclei project fibers to the ipsilateral **Edinger-Westphal nuclei** and also to the contralateral Edinger-Westphal nucleus via the posterior commissure

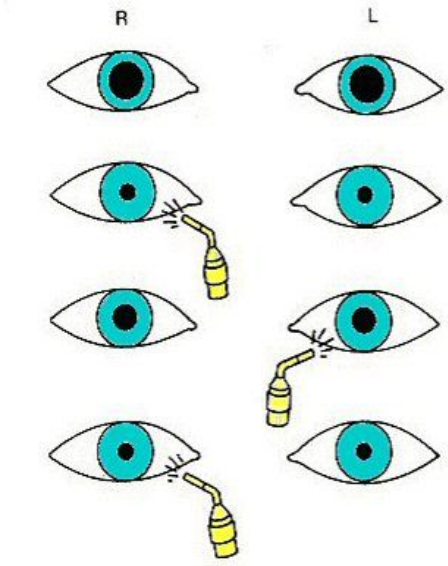
Efferent Pathway of Pupillary Light Reflex (black dotted):

- 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**
- 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.

The pupillary light reflex two main parts: an afferent limb and an efferent limb.

- **Afferent**:

1. Light stimulates the retina => ganglion cells => Optic nerve.
2. Optic nerve enters Optic chiasm (hemi-decussation occurs here to the optic tract).
3. Fibers from optic tract synapse in the pretectal nuclei in the dorsal midbrain.
4. The pretectal nuclei project fibers to the ipsilateral and the contralateral Edinger-Westphal nuclei (Bilateral innervation).

- **Efferent**:

1. The Edinger-Westphal nucleus projects fibers and synapse in the ciliary ganglion.
2. Ciliary ganglion postganglionic parasympathetic fibers (short ciliary nerves) innervate the sphincter muscle (Which constricts the eye)

So, light shined in one eye will result in **EQUAL pupillary constriction in both the ipsilateral pupil (direct pupillary light reflex) and the contralateral pupil (consensual pupillary light reflex).**

Based on this, abnormalities along the pathway could affect the reflex differently, causing relative afferent pupillary defect "RAPD".

- Optic nerve damage at either side or retinal damage (For example, complete transection of the left optic nerve):

1. **Loss of both direct and consensual reflexes** upon shining the light on the left eye (Because the afferent pathway is lost, so, no signal is passing).
2. **Normal and EQUAL direct and consensual reflexes** upon shining the light on the right eye.

The above example describes what happen if there is a complete cut of the optic nerve. But in diseases that affects the optic nerve or retina partly (Ex: early optic neuritis), here there is still an impulse transmission, but the constriction is weak.

In case of relative afferent pupillary defect, swinging light test is used. Here, the flashlight is swinged back and forth from eye to eye. Both pupils constrict when the light is shined in the good eye (Same as above**). However, both pupils dilate when the light is shined in the abnormal eye**. This sign is called Marcus gunn pupil.

Explanation: In a Marcus Gunn pupil, there is reduced afferent input and the pupils fail to constrict fully. Stimulation of the normal eye produces full constriction in both pupils. Immediate subsequent stimulus of the affected eye produces an apparent dilation in both pupils since the stimulus carried through that optic nerve is weaker**. SO, IT A WEAK CONSTRICTION COMPARED TO THE STRONG CONSTRICTION THAT HAPPENED BEFORE => THE EYES DILATE.**

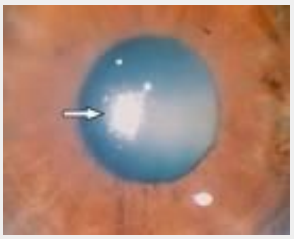
Optic tract damage at either side: **Normal EQUAL direct and consensual reflexes of both eyes** (the amount of fibers going to the nucleus are equal, which then innervate both nuclei). **So, the test has no value if the pathology is at the optic chiasm or beyond** (Unless in rare cases, where the pathology is in the chiasm but before the hemi-decussation occur).

- **Media opacity: Light reflex is normal.**

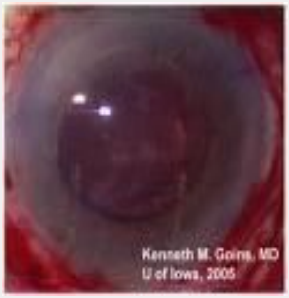
Causes of Acute Visual Loss:	
Painful	**Painless**
• **Acute(congestive)Glaucoma** In the past they misdiagnosed it with MI due to pain severity, they presented with severe headache, drop of vision,severe eye pain,nausea and vomiting. • **Uveitis:** The patient is always in pain. • **Keratitis** infection 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** • **CVA** • **Functional**

1.Media opacities:

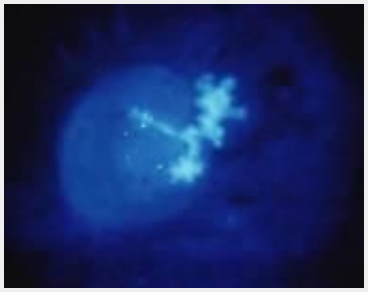
- Corneal opacity either edema or infection(like uveitis) or trauma.
- **Edema:**
  - When the cornea appears like ground glass rather than its normal clear appearance. (steamy cornea)
  - The most common cause of corneal edema is increased intraocular pressure typically in acute angle closure glaucoma(this 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.
  - other causes of corneal edema include severe ocular hypotony[2]
- **Infection:**
  - Any acute infection of the cornea resulting in a corneal ulcer may mimic corneal edema
  - 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, but in severe uveitis the protein concentration becomes similar to that of the serum.
  - posterior synechiae[3] itself doesn't cause visual loss, but the sequences that happen after the posterior synechiae.



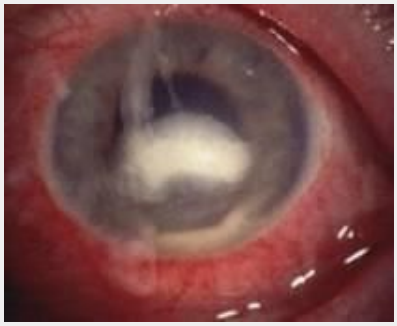
abscess



edema



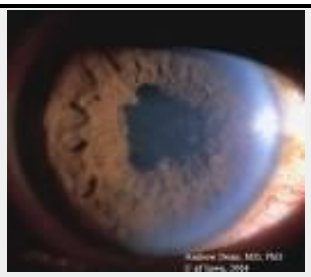
Viral herpetic herpes

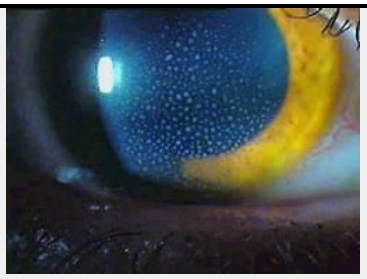


- Infection.
- Abscess in the cornea with hypopyon (which is a collection of inflammatory cells-puss- in the anterior chamber), congested eye.

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

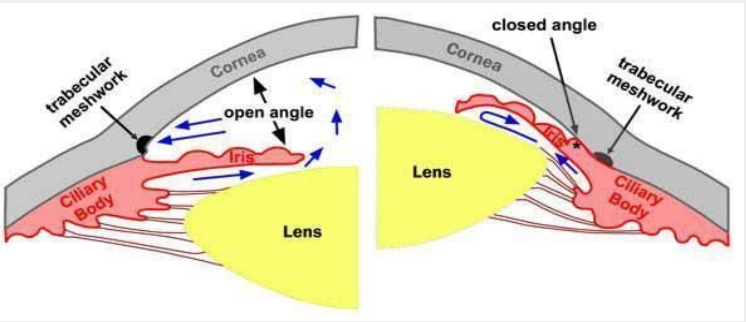
[3] Synechiae are adhesions that are formed between adjacent structures within the eye usually as a result of inflammation.

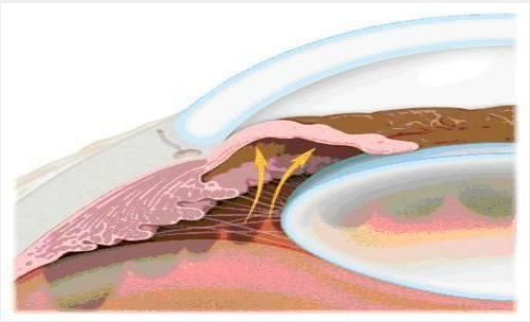



"Uveitis". Precipitate on the back of cornea that resembles corneal edema.

I. Acute angle closure glaucoma



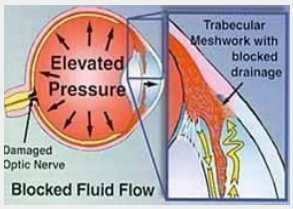


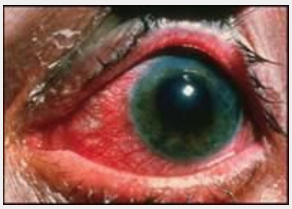
Those patients who are prone to develop acute angle closure glaucoma have unique features:

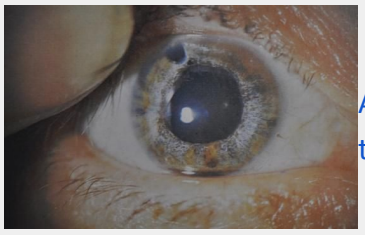
- Shorter eyes "Axial length", although it's within the normal range.
- Hyperopic vision.
- Large lens.

With ageing, 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.





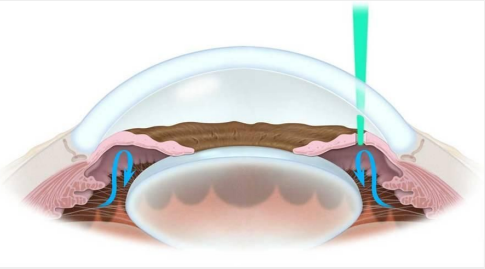
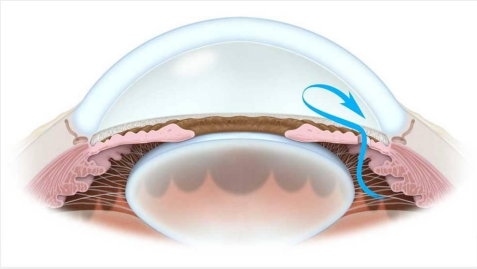
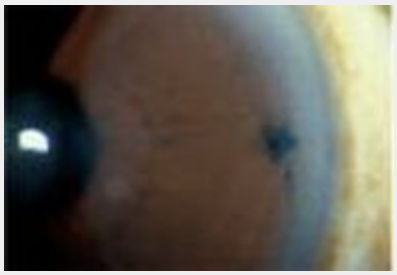
Atrophic Iris because the changes are irreversible, even after treatment. However, this depends on the severity of the disease.

Sign and symptoms:

- Mid-dilated fixed non-reacting pupil “Ischemia of the sphincter”.
- Severe pain. due to high pressure (compression on nerve endings especially those of cornea)(that is the main issue for them to seek medical help!)
- Drop of vision “Blurred vision due to edema”.
- Headache.
- Nausea and vomiting, and it might even proceed to epigastric pain.

Aims of ACG management:

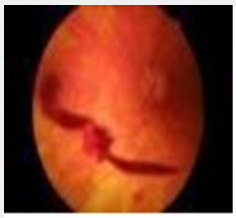
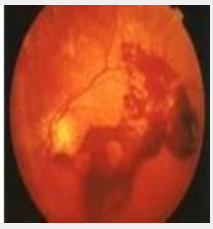
- Decrease IOP
- Prevent future attacks in OU[4]. “Prophylactic laser to the other eye, VERY IMPORTANT!”
- Needs emergent treatment.
  the management is by decreasing the pressure by medications** and then do laser iridotomy “in the outpatient clinic”. This will deflate the iris and open an alternative pathway for the aqueous. If it’s not treated, it will cause fibrosis and the laser doesn’t help anymore.

**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.

II .Vitreous hemorrhage Not a diagnosis rather than a sign of many diseases

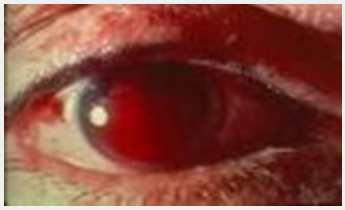
- Any bleeding into vitreous cavity will reduce visual acuity.
- Trauma, seen in diabetics, retinal vein occlusion and acute posterior vitreous detachment and intraocular surgery.
- Rarely, can accompany subarachnoid hemorrhage.
- **If you cannot appreciate a red reflex with an ophthalmoscope and the lens appears clear, you should suspect of vitreous hemorrhage.**
- **The diagnosis is confirmed with slit lamp examination through a dilated pupil.**
- B scan(ultrasound) is important.

[4] Abbreviation for Latin oculus uterque, meaning each eye or both eyes.

III. 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.Retinal Diseases:

I. Retinal Detachment:

- It is retinal splitting, and it happens between 2 layers, the Neurosensory retina and retinal pigmented epithelium.
- 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.

Symptoms:

Prodromal symptoms:

A. flashes برق

B. floaters: 1. VF loss- curtain-like

2. sudden, painless loss of vision

-There is an 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.

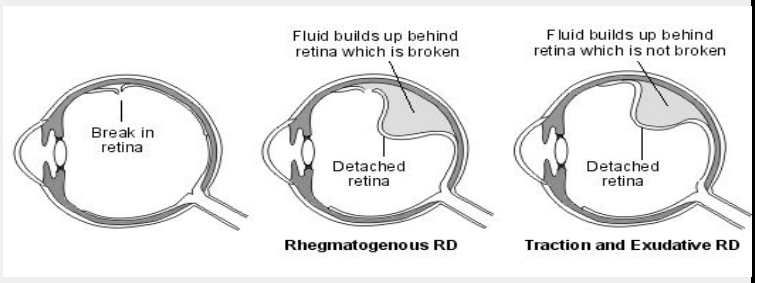
-usually old age.

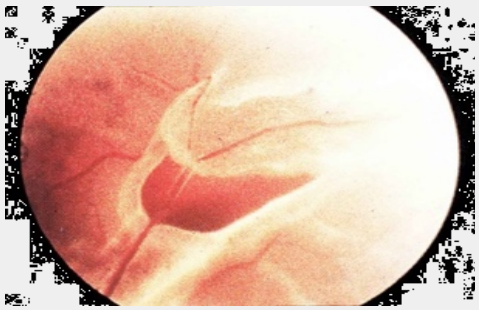
Types:

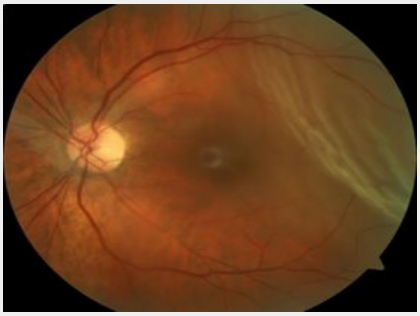
1.Rhegmatogenous RD[5] Due to a tear in the retina "most common".

2.Traction RD[6] in Diabetes

3.Exudative RD[7] due to inflammation, and if we treat the underlying pathology the problem will be solved.







[5] If a tear occurs in the retina, allowing liquified vitreous to gain entry to the subretinal space and causing a progressive detachment.

[6] If it is pulled off by contracting fibrous tissue on the retinal surface.

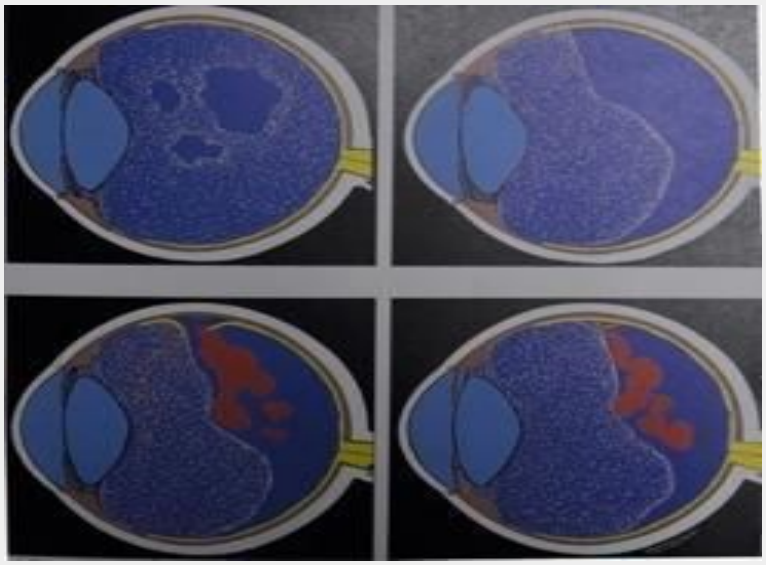
[7] Fluid accumulates in the subretinal space as a result of an exudative process.

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.[8]
- Trauma, History of retinal detachment.



Management:

- RD does not need urgent management, unless if the macula is 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).

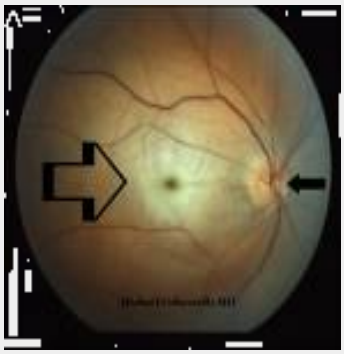
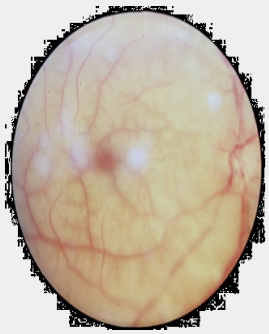
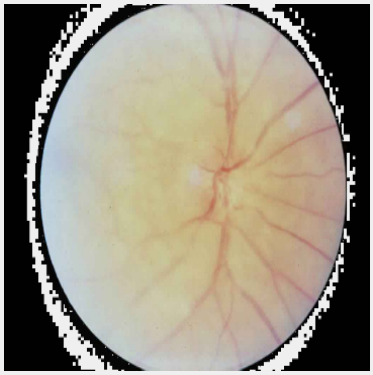
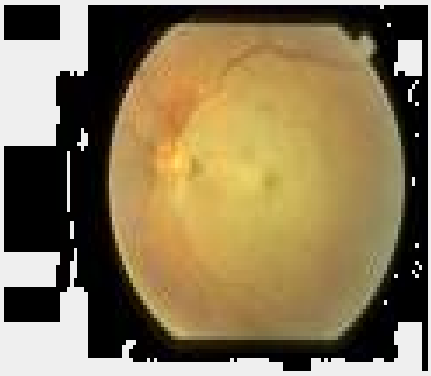
- You don’t need to know the treatment details below.
- Scleral buckle, cryotherapy, SRF drainage.
- Vitrectomy, AFX, endolasser, long acting tamponade (Gas, silicone oil)

[8] no lens. In the past they used to deal with cataract aggressively (traumatic surgeries).

II. Retinal vascular occlusions:

Central Retinal artery occlusion

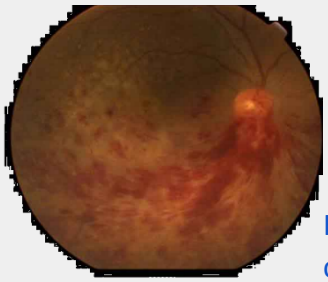
- A sudden, painless and often complete visual loss may indicate central retinal artery occlusion.
- Several hours after a central retinal artery occlusion, the inner layer of the retina becomes opalescent.
- A cherry red spot is seen due to the pallor of the perifoveal retina in contrast to the normal color of the fovea.
- A chronic cherry red spot is also a feature of the storage diseases such as Tay-Sachs disease and Niemann-Pick disease.

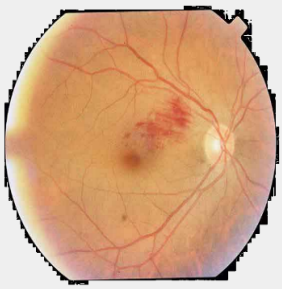
Central Retinal vein occlusion:

- Ophthalmoscopes picture of disc swelling, venous engorgement, cotton wool spots and diffuse retinal hemorrhages like blood and thunder.
- Loss of vision may be severe.
- It has two types: Ischemic and non-ischemic.
- Non-ischemic 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.
- Ischemic: It is a disaster that will lead to Permanent visual loss.
- In ischemic type, it will cause neovascularization which leads to “90-days glaucoma”.
- the way to differentiate between the two types is by visual acuity, and pupillary reflex, in ischemic type there must be an afferent defect.
- 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.
- **Non-ischemic => Intact pupillary reflex / Ischemic: RAPD.**

- There is no generally accepted management. Central retinal vein occlusion is not true ophthalmic emergency.
- Treatment should be directed at reducing associated macular edema by injecting anti-vascular endothelial growth factor agents “Anti-VEGF”.
-



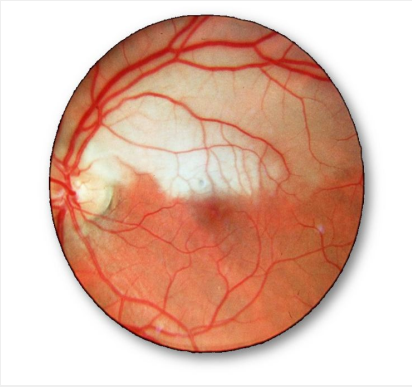
Hemiretinal vein occlusion/engorged veins/ cotton wool spots/disc edema



Branch vein occlusion

<u>Branch</u> Retinal Artery Occlusion:

- When only a branch of the central retinal artery is occluded, vision is only partially lost.
- 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



3.Optic Nerve Disease:

I. Optic neuritis (most common cause)

- Optic Neuritis is inflammation of the optic nerve and It is usually idiopathic but may associated with multiple sclerosis in a significant number of cases.
- Visual acuity is markedly reduced **and an afferent pupillary defect is present.**
- 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.
- 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: differentiating between papillitis, retrobulbar neuritis, and PAPILLEDEMA

	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
Unilateral/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

II. Ischemic optic neuropathy

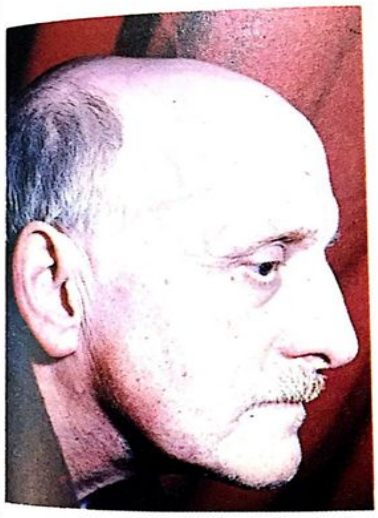
- Anterior ischemic optic neuropathy [AION] is a relatively common cause of severe visual loss.
- The basic lesion is a segmental or generalized infarction of the anterior part of the optic nerve caused by occlusion of the short posterior ciliary arteries.
- Irreversible painless visual loss.
- It has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

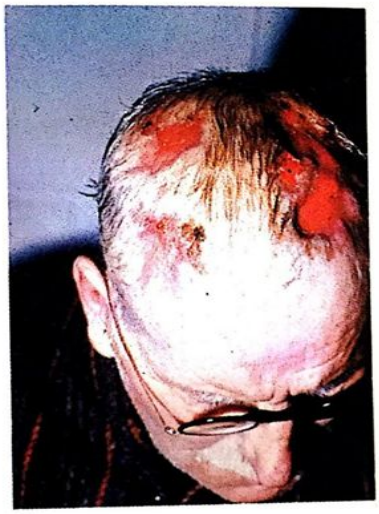
Arteric:

- 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.

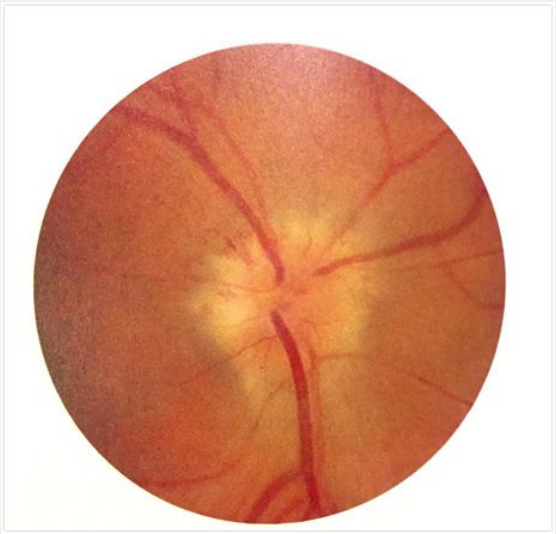
Non arteritic:

- Due to non-inflammatory disease of the small blood vessels.
- Common cause is atherosclerosis.
- There is no treatment.





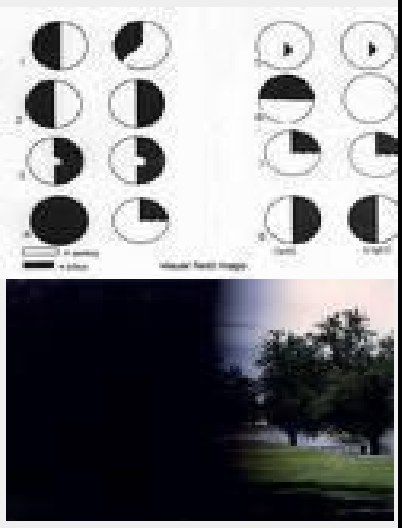
Arteritic AION



4. Visual Pathway Disorders:

I. Homonymous hemianopia

- loss of vision on one side of both visual fields
- may result from occlusion of one of the 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.
- Any patient with a hemianopia needs at CT or MRI to localize and identify the cause.
- behind the optic chiasm.



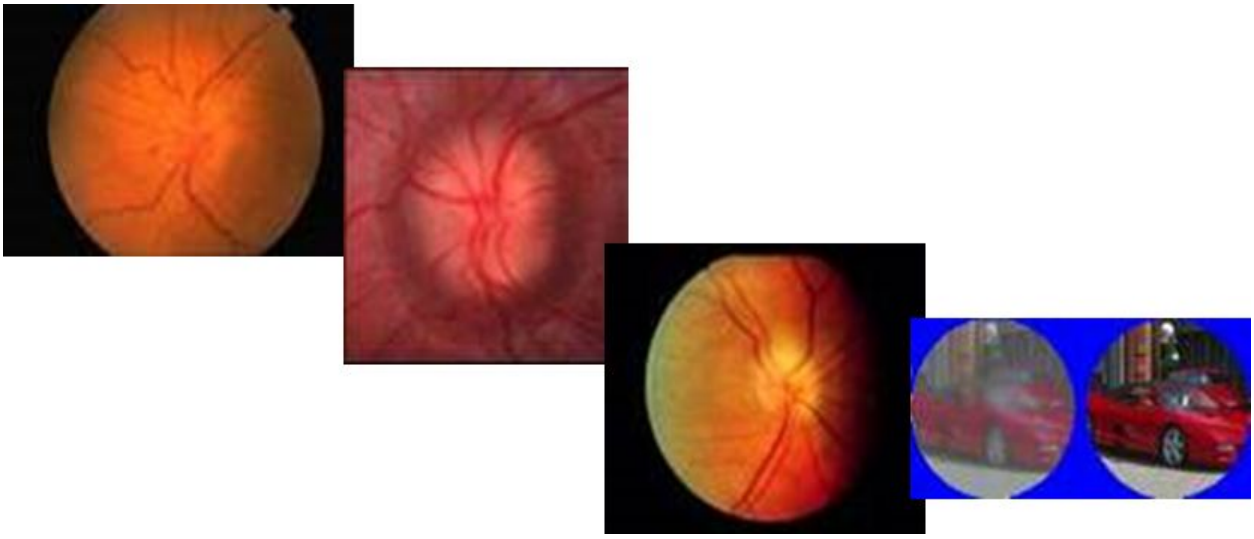
II. Cortical Blindness:

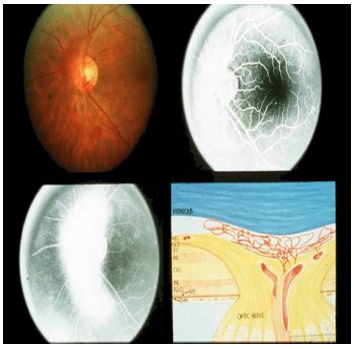
- A rare bilateral extensive damage to the cortical visual pathways results in complete loss of Vision.
- This condition is referred to as cortical, central or cerebral blindness.
- As the pathways serving the pupillary light reflex separate from those carrying visual information at the level of the 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.

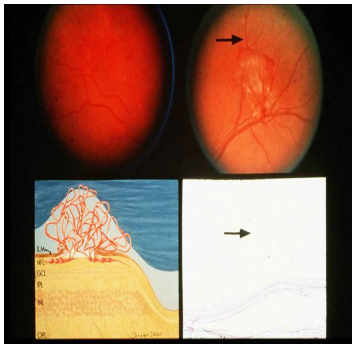
5. Functional visual loss:

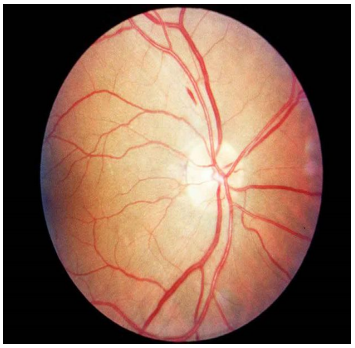
- A functional disorder is used in preference to hysterical or malingering to describe visual loss without organic basis.
- A patient may report complete blindness in one eye and normal vision in the other eye, and no relative afferent pupillary defect

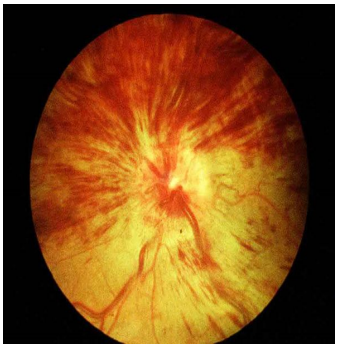
The following pictures are listed in the slides at the end, but the doctor didn't explain any of them.

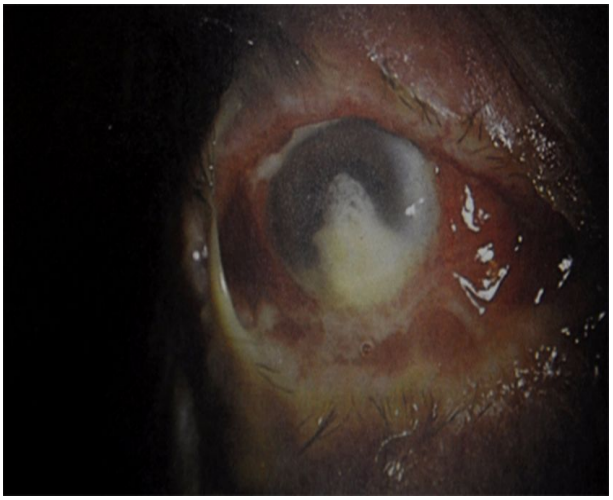


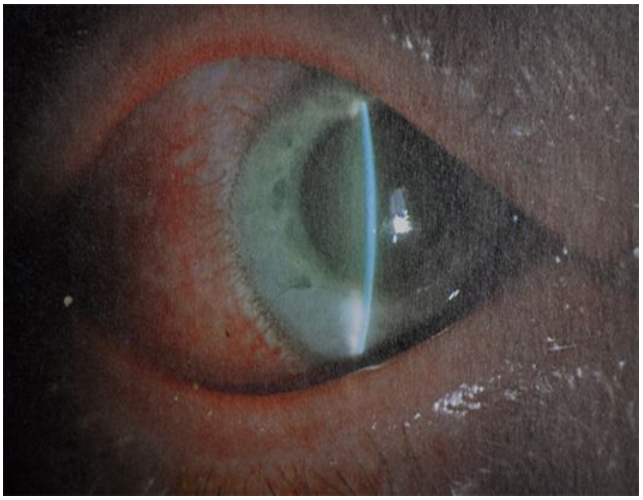


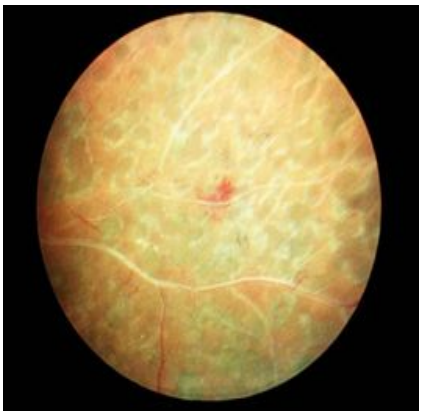


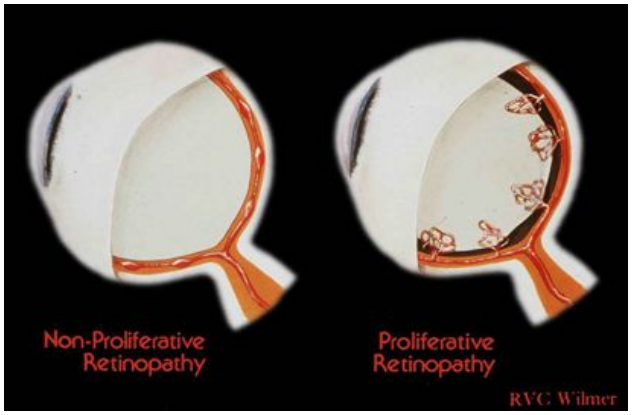


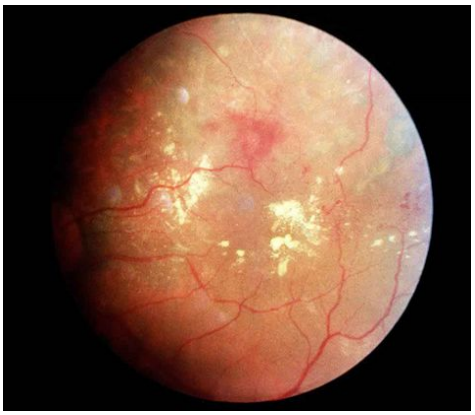


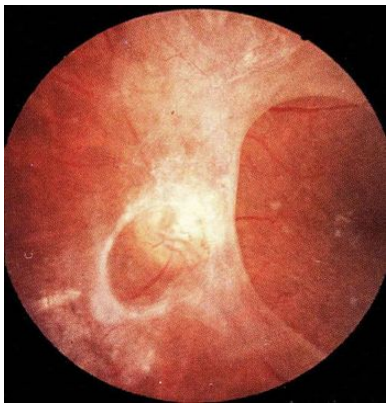


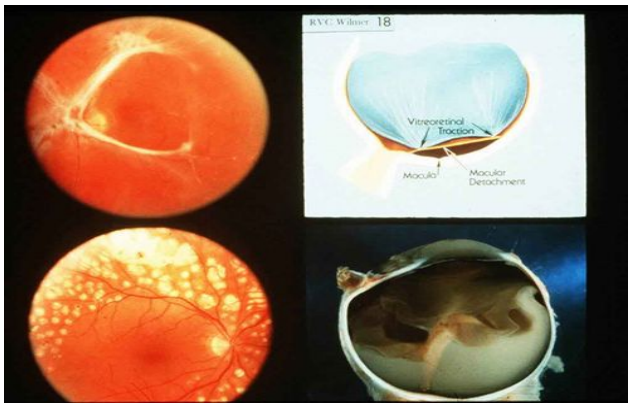


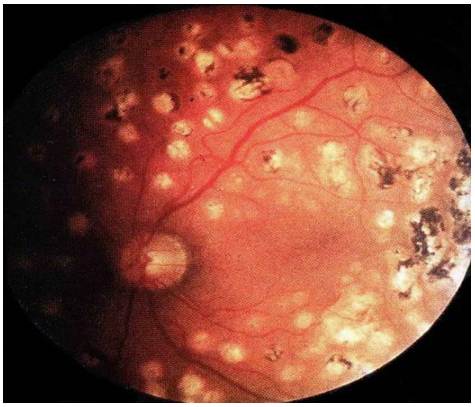


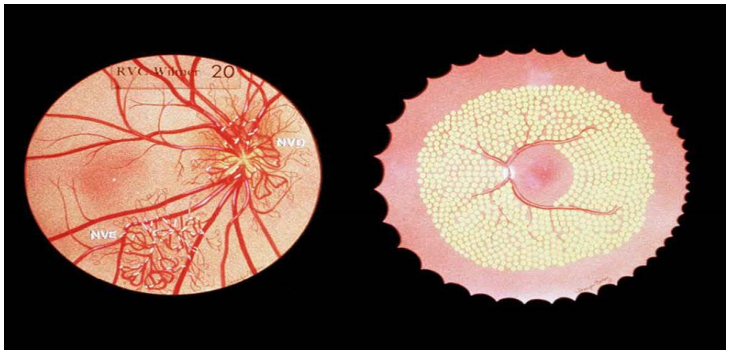

Non-Proliferative
Retinopathy
Proliferative
Retinopathy
RVC Wilmer

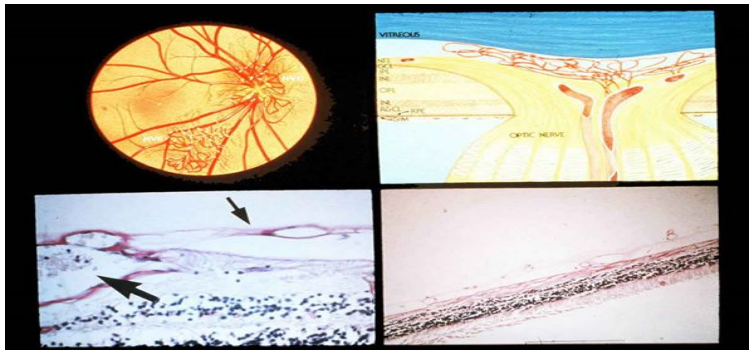


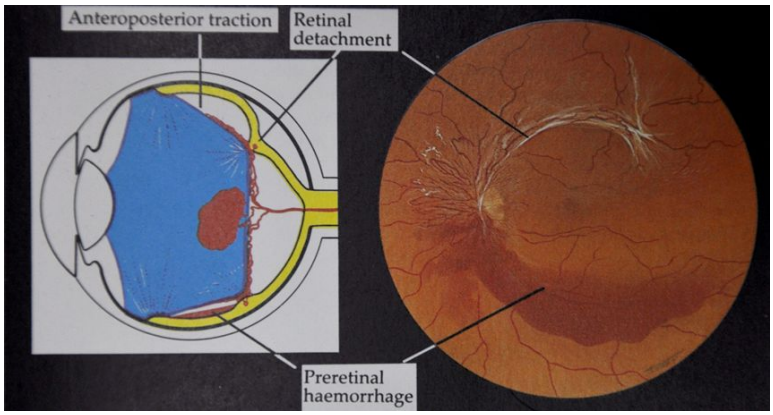



RVC Wilmer 18
Vitreoretinal
Traction
Macula
Macular
Detachment




RVC Wilmer 20
NVE
NVE


VITREOUS
OPTIC NERVE


Anteroposterior traction
Retinal
detachment
Preretinal
haemorrhage