ACUTE Visual loss (retinal occlusion)

-Increasing age may predispose to retinal vein occlusion at the 6th and 7th decade.
This may be due to atherosclerosis. The retinal vein and a
Artery enter through the optic canal with the same adventitial sheath. This sheath is not elastic. Once the artery is atherosclerotic, it becomes stiffer. It will compress on the vein resulting in occlusion and our pathology.

-Hypermetropia results in a crowded eye, these patients are at a higher risk of venous occlusion.

- What is the difference between branch and central retinal vein occlusion?
    - If we divide the retina to four quadrants. Each quadrant will get a major branch, superior temporal and superior nasal, and inferior temporal and inferior nasal. Each of these branches is a central artery. There are two trunks giving off superior and inferior branches. When we say occlusion, there will be occlusion of one of these branches. The main features of it is not total but partial visual loss.

-Branch retinal vein occlusion
- Signs: dilatation in the vein due to stasis and elasticity of the vein, increased venous pressure in that area, decreasing the drainage and elimination of the waste products. There will be accumulation of waste products and acid resulting in a local environment of increased CO2 and acidosis, further resulting in an area of vasoconstriction and ischemia. There will be damage to the venous wall and capillaries à leakage of the intravascular contents outside (RBCs, WBC, platelets, and plasma) à hemorrhage for the first contents, edema for plasma.
- Cotton wool spots, or soft exudate are present in areas of ischemia in the retina. Once we see hard exudates, this is a sign of leakage of lipoproteins and lipids, giving us an accumulation of yellow contents, irregular in shape, well demarcated, etc.

The way to differentiate between both is:
- § Soft: pale, white, fluffy ill-demarcated
- § Hard: multiple in size, irregular, well-demarcated, yellow.

- Complications:
    - § Macula is between the superior temporal arcade and inferior temporal arcade temporal (outer/lateral) to the optic disc. The diameter of the macula is 6 mm, or 4 disc diameters. The histologic definition of macula: increased amount of xanthophyll pigments.
    - § Retinal neovascularization: patients bleed easily. These new blood vessels are elevated above the retina not within the retina. They are associated with a fibrotic element. They are at a higher risk of hemorrhage, specifically vitreous hemorrhage. The fibrotic element will also cause traction of the retinal detachment.
        - ·Neovascularization is treated with laser photoregulation.

-Central retinal vein occlusion

- Fluorescein is a dye that reaches the retinal circulation and then we take photos with a special camera giving us an idea of the integrity of retinal circulation.

Seeing long white lines à good circulation, there are no areas of nonperfusion. The dark spots in the angiogram is hemorrhages, meaning that the fluorescein got to that area but was blocked by the hemorrhage. This is called blocked fluorescence.

§ Manifestation of a patient coming with the fluorescein angiogram picture in the slide

·Blurred vision, acute, hours to days. The venous process takes a long time. Hours à days. Arterial process would be near-instant.

§ In the examination for this patient, the retina is nonischemic so we will not get Relative Afferent Papillary Defect.

§ This patient is 6/12 – 6/60, but they won't come with severe visual loss (i.e. counting fingers).

§ Complication of this patient:

·Exudates à edema. This is a common fate of most occlusions.

·Can become ischemic from nonischemic (around 1/3 of patients in 2-3 years).

·If it becomes ischemic à retinal neovascularization.

·If no ischemia, good prognosis

- Ischemic CRVO

§ Fluorescein angiography

·It has lobular pattern, also called capillary dropout, seen as dark areas between the arterioles and venules. These dark areas are areas of nonperfusion. One of the features of fluorescein angiography in venous occlusion is delayed venous filling.

·What is the duration to see the vein filling after the artery?

- One second

· Fluorescein is in circulation for 30-40 seconds. Once the doctor injects, for the first drop of fluorescein to be seen at the fundus needs 12-20 seconds. We inject in the vein rather than the artery.

§ What are the signs to differentiate ischemic and non-ischemic?

·Sudden, dramatic vision loss, upon fluoroscopy we see hemorrhages. This is ischemic until proven otherwise.

§ Complications

·Angiogenesis

·Neovascularization in the anterior segment

- Growth of abnormal vessels on the iris and the angle. This will affect the drainage as well. There will be a sheath of fibrosis building on the whole angle, closing it, resulting in secondary angle closure. There will be *neovascular glaucoma*. It is one of the major complications of central retinal vein occlusion. This usually occurs at the third month post-onset. The patient will come with severe eye pain and injection. The pressure will be high (50-60) with abnormal neovascularization at the iris and the angle. This will tell us it is a neovascular glaucoma.

- The earliest sign à twigs of capillaries on the iris. I will need to treat and prevent neovascular glaucoma. To treat, we will do panretinal laser photocoagulation for the whole retina except for the macula and optic disc (the central part). We will thereby kill the peripheral retina.blunt the effect of glaucoma.
- Hypoxia Ischemic Factor (HIF) à secretion of angiogenic factor from the brain in ischemic retina.
  - § Prognosis
    - · It depends largely on the presenting visual acuity.
    - ·We treat to lessen the pain the patient feels. It is not only to restore vision, but to
  - § There is a picture of twigs for neovascularization on gonioscopic appearance.
  - § Management
    - · Macular edema:
- First line is anti-VEGF (Vascular endothelial growth factor). VEGF is a potent permeability and angiogenic factor. The anti-VEGF are effective in managing macular edema secondary to ischemic CRVO.

PREPARE THE ANTI-VEGF DRUGS AVAILABLE

- Argon laser: second-line.

Arterial Occlusions

- Sudden, dramatic visual loss. The patient will say that he lost vision immediately like a curtain covering.
- Amaurosis fugax (check spelling) is a transient attack of arterial occlusion.
- RF for arterial occlusions
  - Atherosclerosis, smoking, diabetes, hypertension

-Causes: cholesterol, thrombi, calcified, vegetations

-Signs: RAPD, pale funduscopy.

-The macula is supplied by ciliary circulation. 20% of the population's macula are supplied by the cilioretinal artery

-Box scarring (check spelling) of the retina. <ADD INFO ON IT>. Cherry-red spot: the normal foveola. It appears cherry red because the area around it becomes pale. The fov eola was spared because it's supplied by <ADD SUPPLY> and not from the central artery.

-Management: Arterial occlusion is very critical. The time limit before irreversible damage is 90 minutes. Management is trying to dislodge the thrombus further. I will do vasodilatation and decrease the ocular pressure.

Retinal Detachment

-Separation of the neurosensory retina from the underlying retinal pigment epithelium.

-9/10 of the retinal layers are neurosensory and 1/10 (pigment epithelium – supportive cells) isn't. The detachment occurs at the level of pigment epithelium.

-**B-scan:** This will allow us to see the retinal detachment. eye ultrasound

- Mechanism*
  - The vitreous gel is present since birth. It's an organ (relatively speaking), with a gelly core and cortex. 99% of this gel is water. 1% is protein (collagen, proteoglycans, etc). When there's degeneration of the collagen, we will get water vacuoles that expand and coalesce. The water

vacuoles will breach the cortical vitreous and the fluids will come between the cortical vitreous and the retina. This is called posterior vitreous detachment, a normal aging process.

- If the vitreous continues to be pushed and the gel pulls, a tear will occur at the periphery of the retina. The tear will stay open due to traction of the vitreous gel and fluids will leak and come between vitreous and the vacuolar regions. The risk of detachment in normal population à 1 in 10000

- Features
  - Rhegmatogenous RD
    - § The pulling force will be interpreted as flashes of light indicating traction on the retina.
    - § If there's bleeding, we will get floaters. A tear will take part of the pigment epithelium with it.
    - § Flashes of light and vitreous floaters à vitreous detachment; peripheral visual loss and scotoma à retinal detachment.
  - Appearance: bolus in shape,convex, corrugated margin between the free and attached retina.

-Difference between rhegmatogenous RD and Tractional RD?

- There are white bands, also tractional with no convexity and retina is tethered and fixed by fibrous tissue.

-Management

- Photoreceptors will die upon detachment (there is no support from the retinal pigment cells). There is a critical period of one week maximally for RD treatment, since there is minimal chance for treatment afterwards.
- Where is the tear? What is the state of the macula? are the two most important questions to ask. If the macula is removed, the patient will never get 6/6 again. If the macula is attached, there is a chance for me to save it and thus it is more important. Macula on à 24-48 hours, Macula off à 1-week emergency.

The important determinants are the duration of the RD and the status of the macula

-Treatment

- Buckling: putting a silicone band around the eye
- Vitrectomy: take out all the vitreous, aspirate the fluid from the subretinal space and get the retina flat again.
- Retinopexy: I inject small volume of SF6 gas, this will seal the tear. The fluids that entered the retinal bed epithelium will absorb the fluids that leaked in. used in the superior tears only

-Risk factors

- Certain peripheral retinal degenerations
- Surgery
- Trauma
- Myopia (stretched eye)

Diabetic Eye Disease

- Duration and glycemic/metabolic control are the most important risk factors in diabetic neuropathy. Duration is a static risk factor, i.e. I can't change it. Glycemic control is modifiable or a

dynamic risk factor. If I manage the modifiable factors, I will delay any terrible symptoms for a long time.j

-Clinical features

- Microaneurysms
  - § Clinically, they appear like tiny red dots (less than 100 microns). If it's larger, it's not a microaneurysm, rather a hemorrhage. Both indicate the same pathology however.
  - § Difference between aneurysm and hemorrhage on fluorescein is that hemorrhages are black/dark, and aneurysms are white.
- Hemorrhages
  - § The colour of the hemorrhage reflects its depth. The deeper it gets, the darker it gets.
- Exudates
  - § Clinically, they are yellow and usually occur around and within the macula. They are in a circinate (circular? Spelling) pattern. The exudates are deep in the retina and not above the retina.
  - § Cotton wool spots indicate ischemia
- Venous changes
  - § Loops and beading; the mechanism behind it is weakening of the venous wall due to ischemia leading to the kinking and beading of the vein.
- Neovascularization shows up as tree-like arteries and branches at different areas in the funduscopy.
- Classification of non-proliferative vs. proliferative depends on the hemorrhages, IRMAs, venous changes, aneurysms, etc.
- 4-2-1 rule
  - § Hemorrhages in all four quadrants
  - § Venous bleeding in 2 quadrants
  - § IRMAs in 1 quadrant
  - § A patient with one of these criteria is severe NPDR; a patient with two of these criteria is very severe NPDR.

- Diabetic maculopathy
  - Hyperfluorescence close to the hard exudates means that we have leakage in that area, and edema at that site.
  - OCT view: this one is in the center of the retina. There should be central depression at the foveola. The picture on the bottom à water vacuoles (cystic spaces), separation of epithelium, edema (central)

-Neovascular glaucoma, nonclearing vitreous hemorrhage and retinal detachment are what advances the patient to an incredibly severe/advanced case.

- Picture A: hemorrhage above the retina; B: hemorrhage with different pattern; C: detachment (seen due to fibrous fibers), D: NVI
- Following picture is vitreous hemorrhage.

- Treatment:
  - Nonproliferative stage: Conservative management. Control of risk factor and regular check-ups.
  - Proliferative stage: Panretinal photocoagulation is the treatment of choice

- Advanced stage: Surgery

Thyroid Eye Disease

-Conjunctival injection: Blood vessels are apparent with the naked eye.
-Scleral show during lid lag test = test positive.
-Thyroid eye disease is the more common cause of unilateral AND bilateral proptosis. “in adults “
-Hering's Law: There is reciprocal signals for two antagonistic muscles. There will be positive and negative impulse for opposite muscles. If this doesn’t happen, the muscle will move halfway and then the action will be opposed by the contract of the other muscle. A negative impulse on that other muscle will result in full movement. In restrictive myopathy, the nerve and muscles’ impulses are intact, but the muscle’s skeletal fibers become fibrotic fibers à restrictive movement. This fibrosis is irreversible.
- Clinical features:
  - Overall thyroid state (systemic manifestations), diplopia, pain and discomfort, visual loss, corneal ulceration, cosmetic

-Management
  - Stabilize the thyroid state of the patient
  - Lid retraction and proptosis may result in dryness of the cornea, we will have to do eyelid surgery to narrow the fissure and decrease the dryness.
  - Steroids, radiotherapy or surgical decompression for the optic nerve
    - § Surgical decompression – orbit has four walls. I can break one or all four of the walls to separate the orbit and allow the pressure to be released
  - Diplopia
    - § Surgery is one of the options; use other muscles to heal the fibrotic fibers of the restrictive myopathy-affected muscles.

-Prognostic factors
  - Level of thyroid hormones, pretibial myxedema, etc.
  - One of the most important factors is smoking. It will change the whole course of the treatment.

Eye Trauma

-The most important thing in chemical injury is copious irrigation.
  - Ringers, normal saline, etc.
  - We usually irrigate until the pH is neutralized or 15-30 minutes of irrigation.
  - Everting the lower lid is very important
- Grading severity
  - Corneal clarity, limbal ischemia are the most important factors

- Adhesion between bulbar and palpebral conjunctiva = symblepharon
- We have to evert the upper lid in these cases. It's very important.
- Hyphema: blood in the anterior chamber.

-Iridodialysis: separation of the iris from its iris root.

- Main cause of D-shaped pupil

-Elevated IOP leads into corneal blood staining in hyphema.

->35 for >7 days or persistent clot > 10 days.

-Rupture and laceration are both open globe injuries: rupture from a blunt object, laceration from a sharp object. (The eyewall has or doesn't have a full thickness wound.)

- Most important of this list is the open closed rupture and laceration.

-Penetrating injury is usually associated with a FB.

-Perforation and penetration are differentiated by the presence of an outlet or not. An outlet (exit wound is seen in perforating. There is no exit wound in penetrating.

-Lamellar laceration is a closed globe injury

-Blow-out fracture: Blunt trauma with an object that has a diameter larger than the diameter of the optic orbit. The blow will be transmitted to the orbital wall and we will get the fracture at the infraorbital canal <CHECK THIS>

from wiki:
When an external force is applied to the orbital cavity from an object whose diameter is larger than that of the orbit, the orbital contents are retropulsed and compressed. The consequent sudden rise in intraorbital pressure is transmitted to the walls of the orbit, which ultimately leads to fractures of the thin medial wall and/or orbital floor

- CT scan is the investigation of choice.
- There will be a teardrop sign. A herniation or protrusion from the orbits to the infraorbital area.
- Enophthalmos: recession of the globe inside the orbit. From above the patient, you will not be able to see the cornea.
- Infraorbital nerve anesthesia is very important as a sign of blow-out fracture.
- Periocular signs, enophthalmos, infraorbital nerve anesthesia, ocular damage, and diplopia.
- Subcutaneous emphysema should not blow their nose out because then they will increase the infection.

- Lateral canthotomy and cantholysis will decrease the pressure in the eye.

-Raised IOP and blunt trauma may lead to Boucher's ring? <CHECK THIS> i think it is Hyphema

-The importance of lid lacerations is that we should always think of the deep structures. If fat is coming out of the eye, the orbital septum is hit and when that happens à levator aponeurosis is also hit preventing the patient from looking upwards.

- If the lid laceration is vertically and involving the lid margin, the patient will need special suturing to get them close to each other. It will involve more than one layer. We try to return the lid margin to its original position.

-Levator aponeurosis, lid margin, and the lacrimal system are the three areas we have to be wary of when looking at lid lacerations.

- يا عفو الله
- Bani Melhem says: الطب علم"

Chronic Loss of Vision

-The patient will tell you that my vision is weakening over 2 years progressively.

-Questions to ask

- When have you gotten this symptoms (loss of vision) “from 1 to two years”
- Has it gotten worse? “Yes”
- Painful or painless?
  - § Pain: differentiate itching, discomfort, photophobia, or true pain

- Causes of progressive, painless vision loss
  - Cataract, glaucoma, corneal opacification, age-related

-Cataract (definition: white water): opacification in the lens (a biconvex structure, crystal clear, held in position within the zonules that are connected to the ciliary body or muscle). With accommodation, the zonules will have less tension, and they will become more convex and the person will see things clearer.

-Hypertensive retinopathy, by definition, is normal acuity.

- With age, the muscles and zonules will become stiffer and the accommodation will be deteriorated. Then, the patient will need glasses for reading (+) for the rays to be converged onto the eye. This isn’t called Hypermetropia.
  - Myopia: when the parallel rays of light (coming from infinity) refract and come to a point of focus in front of the retina in the absence of accommodation. Thus, since accommodation isn’t in this, it cannot be called Hypermetropia. Or patients who are already myopic cannot become normal with age because they will still have myopia; it’s only their accommodation (flexibility of the lens) that will be affected.

-The crystallized lens is divided into capsule and cortex. Inside the cortex is nucleus (very hard). Thus, it will be capsule – cortex – nucleus – cortex – capsule. For simplification, we have anterior and posterior capsule.

-Classification of cataract:

- Pediatric: 1/3 of the cases are congenital (1/3 autosomal dominant with <u>variable expressivity and incomplete penetrance</u>), 1/3 with syndromes (toxoplasma, rubella, François syndrome), 1/3 traumatic.

§ NF-I: short stature, café au lait spots, axillary freckles, and optic nerve gliomas.

·The full-blown picture of the disease has five criteria. You need to see 2 of five to determine the disease. Variable expressivity: The expression of the same disease is variable from one person to another. Incomplete penetrance: when the gene wants to penetrate the generations,

this would be an incomplete process, and it may skip a generation. I.e. the parent would have congenital cataracts, the son may not have it, and the grandson would have it.

·When we say penetrance rate is 80%, this means that it's very high. Or there is an (0.8*0.5=0.4) or 40 per cent chance of getting congenital cataracts (provided the mother doesn't have congenital cataracts).

- Pre-senile: Caused mostly by steroids and trauma, plus:
  - § Local (ocular diseases) such as uveitis, myopia, tumour, glaucoma, topical steroid use.
  - § Systemic: SLE, diabetes, radiation injury

- Senile: due to the normal aging process. The lens does not stop growing throughout life. It is continuous. There is always a buildup of proteins.

- Clinical symptoms:
  - The patient may be asymptomatic early on. If the cataracts go into the visual axis, then they start making problems. If the light hits the cataract, it will be massively scrambled in the eye. The patient will have a glare effect on the eye, especially at night when driving.
  - Progressive loss of vision
  - The patient will get pupillary constriction with accommodation resulting in decreased ability to see.
  - Refractive error: the proteins are usually stacked on top of each other. They will get nuclear sclerosis of the lens' nucleus. The refractive index would increase for the lens and the patient will suddenly get myopia (-1 at first, then -3, then even worse).

-Treatment:
  - Depends on lifestyle of the patient and how much the symptoms are affecting the patient (if it's a driver, you have to do surgery).
  - Location and size of the cataract
  - General medical condition of the patient

- Surgeries
  - Intracapsular cataract extraction (ICCE) (with or without IOL)
    - § Contraindications for IOL
      - · Absolute: Patient refusal
    - § Phakic before surgery
    - § After surgery, the patient becomes aphakia (technically blind)
    - § Since the patient becomes blind after the surgery, the doctor would usually wait until the cataracts are fully matured.
    - § We put anterior chamber IOL for these patients since they don't technically have a posterior chamber any more.
    - § Indications: very severe glaucoma with hypertension
  - Extracapsular cataract extraction (ECCE+IOL) (WITH IOL)
    - § We do a cut from 10 o'clock to 2 o'clock, we open the lens in the anterior surface of the capsule, get the nucleus out and the cortex as one piece then clean up.
  - Phacoemulsification (WITH IOL)

§ Small opening (2.8-3 mm) with 1mm cuts on the circumference, then we do capsulorhexis (cutting across the whole circle). There will be a piezoelectric crystal at the end of the probe that moves at 15 MHz and ultrasonic waves, which grinds the nucleus. This coupled with fluid and emulsification will make the hard nucleus into a smooth one and then you will suck up the cortex. This will leave an empty lens. The lens will be folded into a tube, gets injected then it will unfold and expand and become the new lens. This will be a self-sealing wound (especially since it's only 2.8 mm). The healing will start immediately. There will be no accommodation for these patients.

- Now we use topical anesthesia. The whole operation may only take 10 minutes. Local anesthesia used is an injection (retro- or peribulbar) behind the globe to anesthetize the ocular nerves. There will then be akinesia and anesthesia. The perfect anesthetic would be one that does akinesia, anesthesia, and amnesia.

- Complications:
  - Pre-operative: Anxiety (acute chest pain à MI), local/ocular (underlying ocular disease such as ophthalmitis – the infection will spread à MOST fearful complication Endophthalmitis
    - § Endophthalmitis treatment: removal of the whole eye
  - Intra-operative: Anesthesia complication and surgical complications.
    - § General Anesthesia: poor induction, difficult recovery, laryngeal spasm, atelectasis, ileus, rigid spasm
    - § Local Anesthesia: injury to important structures, i.e. nerve and globe injury, incomplete anesthesia, retrobulbar hemorrhage (bleeding behind the eye, especially in patients with platelet inhibitors à warfarin, aspirin)
    - § Topical anesthesia: It needs an excellent, fast surgeon because it will only stay for 15 minutes. This doesn't have akinesia so the patient may be moving his eyes. Sudden movement of the patient's eye will lead to a risky surgery. Lacrimal, Frontal, and Trochlear nerves go through the superior orbital fissure.
      - ·MEMORIZE THE STRUCTURE THAT PASS THROUGH THE SUPERIOR ORBITAL FISSURE WITHIN THE MUSCLE AND OUT OF THE MUSCLE. EXAM QUESTION.
    - § Surgical complications:
      - ·Remember the steps of the surgery and apply that to the complications.
      - · Incomplete opening of the capsule
      - ·Dropped nucleus with rupture of posterior capsule.
      - ·Dropped IOL
      - ·Hemorrhage
  - Post-operative:
    - § Early: not all of these need management
      - · Wound leak
      - ·Transient corneal edema
      - ·Dropped IOL
  - Due to possible rupture of the posterior capsule
    - · Retinal detachment
    - ·Endophthalmitis

- The vitreous becomes pus; absent red reflex, ocular pain, eyelid swelling, severe ecchymosis, and severe injection/redness
    - § Late:
        - ·Persistent corneal edema
        - ·Posterior capsule opacification (PCO)
- Hit it with laser and the patient will be able to see
    - ·Refractive errors
- Pre-existing astigmatism
- Wrong IOL
- Sutures (too tight, too lax) – we usually take them off 6-8 weeks

-Progressive**:**

- Phaco is very fast healing
- ECCE is slow healing
- Same prognosis

Glaucoma

- A group of disease characterized by “characteristic” and progressive optic nerve damage and “characteristic” and progressive optic field changes with or without raised IOP.
- Low-tension glaucoma: the blood circulation of these patients is already compromised in which they don’t tolerate slight changes in the IOP.

-Ocular hypertension: these patients have increased IOP but without the glaucomatous changes. This is also called “glaucoma-suspect”.

-Pathology:

- Optic nerve atrophy and disc cupping
    - § The disc is the nerve fiber layers which converge and make up the optic nerve. The disc is reddish/pink, called neuroretinal rim. The cup is an empty area, where the central retinal artery and vein pass through. The neuroretinal rim will atrophy and the cup will increase in size in the cases of glaucoma. Cup Disc Ratio is 0.1 – 0.3. Above that is abnormal. The number has to be taken in its own context. The glaucoma, while bilateral, has asymmetric progression. Thus, we need to look at the CDR in context. i.e. if you find one is 0.3, another is 0.1. This means that the normal number is 0.1 and 0.3 is a pathology.

Pediatric

-Different pediatric age groups have different ways to test their visual acuity. We depend on the estimation of visual acuity and the symmetry between the two eyes. At birth, children can see. Their visual acuity is to blink when met with a bright light. The visual acuity in children is low

- Eye is immature.
- The fovea is covered by a thick layer of ganglionic cells. As the patient ages, there will be peripheral migration of the ganglionic cells.

-Fixation behavior starts to develop in children at 6 weeks and fully establishes at 3 months. i.e. looking and following things.

- Children focus on heterogeneous objects more than homogenous objects.

-For infants, those with poor visual behavior, we ask for their age first. Ask for other things:

- Smiling to the mother's face?
- Notices when a toy is thrown in his way of walking
- Does the baby follow her movement?
- These are all indirect cues that show they are in a good position.

- Blind infant/behavior is divided into two broad categories
  - Blindness with nystagmus
    - § This has to be bilateral with significant anterior visual pathway problem appeared and not corrected during the first three months of life.
      - ·These three are the critical period of development for patients.
    - § Since they are anterior, they can be further classified into:
      - ·Diseases that affect the anatomical eye
  - Corneal layer: bilateral corneal opacity
  - Lens: Bilateral congenital cataract
  - Vitreous: Bilateral vitreous hemorrhage coupled with retinal detachment.

ROP – Retinopathy of Prematurity (occurs in 10% of children in gestational age less than 32 weeks). The more developed a country, the higher the rate of ROP, since they actually test for it.

- § Major RF:
  - ·Gestational age less than 32 weeks (less than 7.5 months).
  - · Less than 1500 grams at birth
- § Minor:
  - ·Hyper-oxygenation
  - ·Necrotizing enterocolitis
  - ·Neonatal sepsis
  - ·intraventricular hemorrhage
  - ·long exposure to O2
- § ROP stages:
  - ·0: No ROP <ADD THIS>
  - ·1: follow-up on patient
  - ·2-3: laser treatment to the avascular areas
  - ·4: inoperative with RD; incomplete RD
  - ·5: Complete RD
- RD in this disease specially is inoperative!
- · We keep screening for ROP until the infant gets to 44 weeks. Follow-up every 2 weeks.

- Retina: Foveal hypoplasia
  - § This occurs with two main diseases; aniridia and oculocutaneous albinism (autosomal recessive)
    - ·in aniridia: WAGR
- Wilms tumour
- Aniridia

- Genitourinary
- Retardation of Growth
- Optic nerve: Bilateral hypoplasia (variable vision loss) and coloboma. Usually associated with short stature, mental retardation, convulsion disorders, and dysmorphic facial features.
    - § Coloboma: ineffective/incomplete growth
- Children with hereditary syndromes (i.e. Down's)
    - § At higher risk for certain diseases and child abuse
        - ·Diseases that affect the functional eye
        - ·Leber's congenital amaurosis: total damage of photoreceptors; rods and cones. The ERG will be extinguished ERG since it's composed of A and B. A for rod, B for cones à flat-line. The three below are variable effect on rods and cones.
        - ·Cone dystrophy
        - ·Retinitis pigmentosa
        - ·Achromatopsia?????

from wiki:
·Achromatopsia: *refers to an autosomal recessive congenital color vision condition, the inability to perceive color and to achieve satisfactory visual acuity at high light levels (typically exterior daylight). The syndrome is also present in an incomplete form which is more properly defined as dyschromatopsia. It is estimated to affect 1 in 40,000 live births worldwide*

§ Any patient that presents to the clinic with poor visual behavior and nystagmus with normal eye exam, they must receive electrophysiological investigation or testing. The most imp ortant two tests

·ERG (ElectroRetinoGram)

- Tests the function of the photoreceptors (rods and cones)
    - ·Visual evoked potential (VEP)
- Tests the function of the optic nerve
- Blindness without nystagmus
    - § The problem is posterior to the chiasm and central (in the brain). Eye examination is normal but the processing of the image in the brain is defective.
    - § Always exclude high refractive errors.
    - § Always do fundus examination and refraction examination for these patients.
    - § Sometimes, we will have to wait and see at 2-months or more for these patients.
    - § Some patients have blind visual behavior, ERG VEP normal, MRI normal but the fixation and behavior is practically blind. These patients, you will have to wait for them. They might be DVM. Delayed Visual Maturation. By definition, DVM is a disease of exclusion and retrospection. The patient must improve to normal or near-normal levels by the end of the first year of life. After the patient gets better, then you can put the diagnosis, i.e. "The patient got better. He HAD DVM". Otherwise, the patient has CVI à Central Visual Impairment. The problems might be on the micro level and not present on the MRI.

- **Squint:**
  - It's misalignment of the eye, usually noticed by the mother at the age of 9 months. always binocular.

There is comitant strabismus and concomitant

- § Comitant: equal angle of deviation in different angle of gaze
- § Incomitant: unequal angle of deviation in different angle of gaze
  - · Happens in nerve palsy. In 6th nerve palsy, you will only see it in abduction of the right eye.
  - · Happens in restrictive myopathy à myasthenia gravis etc.

- According to the direction
  - § Eso: inward; exo: outward; hyper: upward; hypo: downward
- `Tropia`: obvious (manifest) deviation
- `Phoria`: hidden (tendency or latent) manifestation
- Treatment:
  - § Treat the underlying cause
  - § Correct the refractive errors if any (give the patient eyeglasses with the correct adjustment for myopia or emmetropia)
  - § Treat amblyopia if possible
  - § Surgically treat the residual angle of deviation (if a patient's eyes become straight with the glasses, there's no need for surgery since there's no residual angle of deviation).
  - § Myopia doesn't create squint. Hypermetropia creates squint.
- Case: A patient comes with +6. Without glasses, he has +40 esotropia, with glasses he has +15. We do surgery to correct for the extra +15 angle. When he takes off the glasses, he will still have squint.

- Amblyopia: Poor vision in one eye creating a difference in visual acuity in both eyes in the absence of organic lesion in that particular eye.
  - Classification
    - § I: Sensory deprivation amblyopia; something preventing the light from going into the eye. Treated up to 5 years of age.
    - § II: Strabismic; the abnormal image from the deviated eye will be suppressed by the brain. The eye will grow anatomically but not functionally. Up to 8 years of age.
    - § III: Anisometropic; this is due to anisometropia (difference in refractive errors in two eyes). Up to 11 years of age. After that age, it becomes irreversible. This latter idea applies to all of the above classifications.

anti-VEGF

1-Avastin(bevacizumab)
2-Lucentis(ranibizumab)
3-Eylea(aflibercept)

1-Avastin(bevacizumab)

monoclonal antibody
MOA:inhibits VEGF by trapping the molecules and prevent it from binding to receptors (FLT1,KDR)on endothelial cell surface
How is it given? intravitreal injections
side effects:infection ,↑IOP ,retinal detachment, vitreous floaters
dose:1.25mg/0.05ml

2-Lucentis(ranibizumab)
Fab fragment
for macular edema, wet AMD ,DM retinopathy
MOA:same as avastin
How is it given? intravitreal injections
side effects:hemorrhages,↑IOP,headache, nausea, dry itchy eye, floaters, redness
dose:0.5mg/0.05ml every 6 wks

3-Eylea(aflibercept)
soluble decoy receptor (placental derived GF)
MOA:same as avastin
How is it given? intravitreal injections
side effects:pain, redness,sudden vision problems,floaters,flashes,↑ sensitivity
dose:2 mg/0.05ml every 8 wks

Dr. Abu Ain Review:

-In high IOP, we're afraid of vascular injury and CRVO. If the pressure is above 50, we might get trabecular artery occlusion. Thus, this is an emergency and you need to interfere soon to relieve the pressure on the eye.

-Funduscopy:
- We sometimes dilate the patient
- We need a dark room
- Ask the patient to look at a fixed distant point.
- Neer triad: Conversion, accommodation and miosis.
- The optic nerve leaves the eye inferonasally.
- We see the red reflex of both eyes.
- Dark spots: might be due to cataracts, vitreous floaters/hemorrhage.
- Dull reflex: dense cataract, RD, any lump in the retina obliterating the reflection, retinoblastoma in pediatrics.

-Optic disc

- Contour
- Cup: central wide depressed area of the optic disc. We look at the cup of the neuroretinal rim. The colour in the slides is normal in both.
- Examples:

1: indistinct margins, pink, and loss of cupping. There is hyperemia. Dilated and engorged blood vessels indicate optic nerve head swelling/compression.

2: indistinct margins, loss of cupping, there is presence of exudates. The cotton wool spots cover the underlying area of the retina. There is presence of flame hemorrhage. This is swollen optic nerve head. If bilateral, central cause. If unilateral, papillitis, optic nerve compression, infiltration, malignancy, etc.

3: This is pallor and not cupping. The way we know is bending of the vessels at the disc. Optic nerve atrophy/compression Pale optic disc indicating optic nerve atrophy

4: commonest cause of extensive optic disc cupping is glaucoma. There is glaucomatous, ischemic and compressive optic nerve atrophy. There is loss of tissue at the glaucomatous one.

5: slightly ill-defined margins, loss of cupping, slight pallor. One differential diagnosis is Hypermetropia and small-eyes. There is optic disc druisins as well, where there will be calcium deposits within the optic disc. CT and U/S will help us determine the calcium deposits.

*Peri-papillary atrophy is the green area signified by the black arrow on the slides.

*SVP: Spontaneous Venous Pulsating is the first sign of optic disc swelling. It is absent in 20% of the population seen on funduscopy.

6: This is a fundus photograph and not just a disc photograph.

·There is a central macular lesion. This lesion is 2-disc diameters. Distinct borders. Hypopigmented compared to the area around it. Most likely macular atrophy. This is most likely dry AMD.

-Folder: Iris coloboma, large disc, granular, hydrops, cd ratio, map dot, lattice, vogts stria, Munson’s, microcornea, dendritic ulcer, iridodialysis, keratoconus, seidel's test

-Dendritic ulcer: There is a fluorescein stain with cobalt blue light, showing uptake of the fluorescein by the cornea which are dendritic in shape. Rose Bengal stain is another stain we use

-The thickest part of the cornea is the stroma. When the cornea is thin, the shape of the cornea will change. This picture is keratoconus. Treatment is cross-linking. If advanced, we can do intracorneal rings or rigid contact lenses. If the patient looks down, we can see a triangle for the patient, called Munson’s sign.

-This is a fluorescein stain with cobalt blue light, showing leakage and dilution of the stain with aqueous humour indicating injury or perforation. This is called seidel’s test or concentrated fluorescein test.

-OCT: This is a cross-section of the central macula. There is a central depression devoid of ganglion cells. We can see the internal limiting membrane. The retinal pigmented epithelium will show high reflectivity showing bright red. The higher the reflection, the brighter, the lower the darker it is.

- Maculopathy is the most common cause of blurred vision in diabetic patients. Fundus Fluorescein angiogram helps us see the fundus and vascularization. There is an area of Hyperfluorescence showing leakage, indicating macular edema. Recently, the OCT is able to show loss of foveal depression, with dark shadows intraretinal. There is also subretinal fluid. This indicates cystoid maculopathy. The common cause in working age group is diabetes. For > 70 age, it's wet AMD.
- There is a macular hole in the retina (OCT photograph), there is slight fluid depositions. There is a detached part of the retina. It detached because of traction of vitreous. This is Posterior Vitreous Detachment. This is RD between the neurosensory retina and RP.
- OCT: This is subretinal fluid, usually to malfunctioning RPE.
-This is a fundus photograph with evidence of dark spots showing scattered hemorrhages. It is a panoramic picture.
-There is looping and beading
-This is a fundus photograph of the left eye showing Panretinal photocoagulation. The commonest indication is a diabetic patient. We have to tell this patient that he will lose peripheral vision. The patient won't be able to drive as well.
-Eye picture: There is chemosis, and conjunctival hyperemia, hypopyon (pus within the anterior chamber), there is corneal ulcer (infiltration). This is an active inflammation (keratitis). The commonest cause is contact lens in adult age group …pseudomonas aeruginosa. The pupil in this picture is mid-dilated.
- Another eye picture: This is a dendritic ulcer.
- Another eye picture: superior subluxation of the normal or crystalline lens with presence of zonules on the inferior aspect. It can be seen in Marfan's and homocystinuria.
-This is a picture of a RD: U-shaped tear. There is a hyperemic tear on the peripheral aspect of the retina causing fluid to go through the tear. The vitreous will be liquefied. Myopic patients are more likely to develop RD.
-This is a fundus photograph of the peripheral retina. There is a retinal tear. The central white area is a flap. The choroid is under it. You will do argon laser Retinopexy. The fluid will thus not extend beyond that area.
    - What are the prerequisites?
        * Dilate the pupils, examine the peripheral retina, get the consent of the patient, etc.

-Keratoconus picture, endothelium will keep the cornea dry. There will be a tear in the endothelium and the cornea will collect fluids. It is self-resolving. It is called corneal hydrops.
-This is a fluorescein stain of the left eye, with 360-stitches there is uptake of the fluorescein from the upper two thirds or one half of the cornea. When there is epithelial damage, it will take up the stain. This is a PKB, a corneal graft. Pain, redness, cloudy vision, photophobia are signs of rejection of corneal graft. HALA YA SEEDDDEEE!!!!