
team 435
Ophthalmology

Lectures

0. **Course Orientation & General Notes.**
1. **Orientation, History taking, Examination.**
2. **Basic Anatomy & Physiology.**
3. **Ocular Manifestation of Systemic Disease.**
4. **Ocular Emergencies & Red Eye.**
5. **Ocular Pharmacology & Toxicology.**
6. **Refractive Errors.**
7. **Chronic Visual Loss.**
8. **Neuro-ophthalmology.**
9. **Lids, Lacrimal & Orbit Disorders.**
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11. **Strabismus, Amblyopia & Leukocoria.**

Course Orientation & General Notes

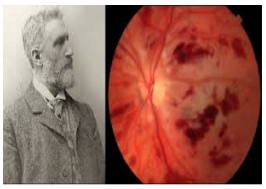
[**Color index:** Important | Notes: **F1**, **F2** | Extra] **EDITING FILE**

Objectives of the course:

- To know the basic ophthalmic anatomy and physiology.
- To recognize assessment and management of common ophthalmic diseases.
- To know how to handle common ophthalmic emergencies.
- To handle simple ophthalmic diagnostic instruments.
- To be aware of common ophthalmic operations.

Done by: Lamya Alsaghan, Munerah AlOmari
Resources: Slides + Notes

Course Orientation: General Notes




● Difference between *optometrist & ophthalmologist*

- Optometrist (دكتور بصريات): is the one who measures the vision refraction.
Opto- = vision | **-metrist** = measurement
- Ophthalmologist: is the one who diagnoses and treat eye diseases.

● Eye is the window for body, examples:

- You can detect systemic diseases by simply examining the eye!
- **Diabetes mellitus:** sometimes first discovered with eye examination, examples:
  - Diabetic retinopathy with fundoscopic examination.
  - Multiple infections in the eyelids, also known as *stye*.
- **Jaunidence;** looking at the color of the sclera. | **- Anemia;** pale conjunctiva
- **Brain Tumor**; manifests as optic disc edema. | **- Thyrotoxicosis.**

● What you need to learn in this course:

- Know the basic anatomy provided to you in lectures, you don't have to go into details!

- You have to have an idea about how to investigate, diagnose and treat **common** eye disorder.

- You'll not be asked about details, like: how to perform retinal detachment surgery or pars plana vitrectomy. You should only know the names for common ophthalmic operations! For example:
  - ***Vitrectomy:*** removal or cutting of vitreous. No one will ask you what is the treatment of vitreous hemorrhage (how to perform the procedure)
  - ***Phacoemulsification:*** cataract (phaco) surgery. suction of the nucleus نفتح فتحة صغيرة و نعمل
  - ***Trabeculectomy:*** glaucoma surgery.
  - ***Oculoplasty types:*** surgical repair of entropion and ectropion, nasolacrimal duct probing & syringing.

- Know how to handle **common** ophthalmic emergencies, like:
  - ***Central retinal artery occlusion -CRO-*** (جلطة في شريان العين): patient will have **sudden loss of vision**. If you don't manage the patient within **1 hour**, it will lead to permanent irreversible vision loss.
    Pathophysiology: thrombus from any part of the body occluding retinal artery just like heart attacks (example: coronary artery occlusion due to hyperlipidemia)

Important question: how to manage central retinal artery occlusion?
Your main goal is to dislodge the thrombus form the artery, how?

- **Dilate the artery:** one way is by making the patient breathe into a plastic bag (اخنقيه) → ↑ CO_2 → vasodilation → dislodgement of thrombus.
- **Decrease pressure in the eye:** by simply doing firm ocular massage. (apply pressure)

  - ***Chemical injury***: "flash" or alkaline in the eye, how would you manage the patient? Wash the eye (irrigation with normal saline)**. Question:** for**: (a)** 30 minutes **(b)**1 hour **(c)**10 minutes | **Answer:** a

- Know the most dangerous and important ophthalmic complications:
  - ***Endophthalmitis:*** infection inside the eye. Bear in mind that a lot of surgeries are performed daily.
  - ***Conjunctivitis:*** infection inside the eyelids. (not to be confused with endophthalmitis)

Side note: don't write loss of vision anywhere in the exam! you will get a ZERO! Most ophthalmic disorders end with loss of vision. You have to say what lead to vision loss (the reason)

- Exam questions are scenarios, example: patient did phaco surgery, he presented to the ER after surgery with pain, redness, and decrease vision what does he have?
(A) Blebitis (area of filtration of glaucoma surgery) ✘
(B) Infection ✓
(**Exam tip:** if you never heard of the term during the course do not choose it as an answer)

- **Lectures: total = 11**

1. Orientation, History Taking, Examination.

2. Basic Anatomy and Physiology of the Eye.

3. Lid, Lacrimal, and Orbit Disorders. SAQs

- Most pictures in the exam are of the retina, lids, orbit and lacrimal system. Examples:
  - **Exophthalmos:** with thyrotoxicosis (most common cause of **unilateral** & **bilateral** exophthalmos)
  - **Nasolacrimal duct obstruction**: babies are born with teary eyes because the lacrimal system is not developed. Normally tears go from the eye to the nose but in this case there is obstruction. The baby will have watering of the eye or *epiphora.*
    Nasolacrimal duct obstruction in children 1st year? Massage then probing & syringing

4. Ocular emergencies and red eye. MCQs

- Acute glaucoma: high IOP
- Endophthalmitis: infection.
- Trauma: ruptured globe or open globe injury.
Immediate **management** = **surgical repair**, Immediate **action** = **eye shield** (not patch not Antibiotic)
- Chemical injuries: acid or alkali **What's worse? Alkali** injuries
- Artery occlusion.
- Foreign body, metallic or nonmetallic, common in construction workers who don't wear protective eye goggles.
- Red eye: **Painful**: Acute glaucoma, uveitis, corneal ulcer. | **Non-painful:** conjunctivitis (bacterial, fungal, viral)

5. Strabismus, Amblyopia and Leukocoria.

- Strabismus, also known as squint حول.
  - **Types:** exotropia & esotropia
  - Anyone with squint/ strabismus should undergo **Cycloplegic refraction**.
  - **How does the Cycloplegic refraction work?** Applying cycloplegic drops → paralyzing the ciliary muscles → giving you the action of the nerve → you give them the full correction and patching the good eye.

- Amblyopia, also known as lazy eye كسل العين: one eye is strong (vision 6/6) and the other is weak -amblyopia- (vision 6/12), if is defined by a decrease in 2 or more lines from the other eye.
You have to know the THREE types:

Deprivation amblyopia	Strabismus amblyopia	Anisometropic amblyopia:
"طفل اتولد وانجرح في عينه، سكروله عين patching the eye of a baby who is just delivered in one week can get لأن amblyopia the brain is not receiving images from the eye فيهملها"	"طفل تاني اتولد بحول squint، عين straight وعين برا أو جوه، يبتدي يركز بالعين الـ straight، فتقوم العين التانيه يحصلها amblyopia"	Errors of refraction in one eye differ in the other eye. "العين اللي فيها higher refraction يحصلها amblyopia، خاصة إذا الطفل محدش عملو نظارة من مرحلة الولادة إلى 6 سنين، إذا ما سوا النظارة ولبسها و correct the vision فـ most likely he/she will develop poor vision in the high refractive error eye"

- Leukocoria: when you shine light on the eye, the pupil normally looks black. If you examine the eye with an ophthalmoscope it will be red (red reflex), if it looks white we call it **leukocoria.** You'll learn all **5** causes of leukocoria in the lecture.
  - The **most common** cause is **congenital cataract** which might lead to amblyopia.
  - The **most dangerous & mortal** is **retinoblastoma**: malignant tumor in the eye (photoreceptors)
  - **Premature with Leukocoria? retinopathy of prematurity**

" فلزلك تاخدو بالكو من الـ common محدش حيسألكو عن الPHPV فيه حاقه اسمها persistent hyperplastic primary vitreous هزي one
of the causes و Coats' disease و vitreous hemorrhage من الـ causes إنما أنا يهمني الحاقات الـ dangerous
to be excluded زي الـ retinoblastoma ونخاف منها لأنها might kill the baby أو يفقد النظر "

OSCE: when you examine the simulated patient (SP) -ophthalmoscopic examination- and you find out he/she has white pupil and the SP is 30 years old.
DO NOT say white pupil or leukocoria! It is only used with **pediatric patients.**

6. Acute Visual Loss. FOUR + 1 MCQs

" أربع حقات لازم تعرفونهم صم وتحفزوهم وحنزود عليهم وحده you should know it لأن ما فيه exam يخلى عنها very important حاقه اسمها "neovascular glaucoma

1. **Acute glaucoma (closed-angle glaucoma)**
2. **Central retinal artery OR vein occlusion.**
3. **Retinal detachment.**
4. **Optic neuritis.**

\+ **Neovascular glaucoma: very important topic.**

" topic بين الـ retina و الـ glaucoma فأنتم ما بتخدهوش في الـ details بالمحاضرات بس لازم تعرفوه، يعني ايه neovascular glaucoma يعني glaucoma سببها new vessels كيف؟ because or retinal ischemia مريض عنده diabetic retinopathy و has retinal ischemia محدش عمله laser فـ it can present with acute glaucoma بـ "what is called neovascular glaucoma

7. Chronic Visual Loss. FOUR + 1 MCQs

" أربع حقات لازم تعرفونهم، وحنزود عليها وحده مش حتلاقوها في المحاضرات، افتكروا دايما أربعة ومعهم وحدة"

1. **Diabetic retinopathy.**
2. **Cataract: commonest**. 70% of surgical procedures done in the hospital are cataract surgeries.

Glaucoma unit is mainly responsible for cataract surgery (anterior segment) + oculoplastic unit.

3. **Chronic glaucoma. (Open-angle glaucoma)**
4. **Age-related macular degeneration:**

"تآكل في مقولة العين تدريجيًا في السن الكبير، وهذا ما يحصل المرض إلا فوق الـ **65 سنة"**

MCQs: Question 1: 40 years old patient and he has chronic visual loss, diagnosis?

لا يمكن الإجابة تكون age-related macular degeneration، يعني this is how you answer the exam يكون by exclusion من تشوفين الـ40 سنة تشيليها من مخك. إذا قلت hypertensive patient شيلي الـ diabetic retinopathy يبقى فاضل حاقتين اللي هما: cataract & chronic glaucoma أجي أقولك أن ال IOP is normal يبقى غالبًا أنها مهيش chronic glaucoma يبقى الـ diagnosis is cataract.
السيناريو في الاختبار يجي زي كده بالزبط!

Question 2: 70 years old patient with chronic visual loss, diagnosis?

أول حاقه أفكر فيها age-related macular degeneration إنما ممكن تكون diabetic retinopathy بس احنا بنعمل exclusion.

Question 3: 70 years old diabetic patient complaints of gradual decrease in vision. On examination, there dot and blot hemorrhage in the retina, diagnosis?

يبقى diabetic retinopathy مع أنه 70 بس مش "age-related macular degeneration

Question 4: 65 years old, complaining of gradual vision loss fundus examination is normal no afferent pupillary defect? CATARACT

"جاء سؤال للأولاد و لا عرفوا لما يقول الـ fundus طبيعي فيعني ما أقدر أقول glaucoma و لا age-related macular degeneration السيناريو ما قال انه مريض سكري عشان أقول diabetic retinopathy فالجواب هو الـ common و cataract!"

\+ **Retinitis pigmentosa.**

"ده موجود بالسعودية كتير جدًا اللي هو العشى الليلي، many patients they are not seeing at night و المشكلة مش في انهم ما يشوفوا بالليل، ولكن gradually، ما تيجوا تقولوا ما نعرفهوش وما درسنهوش، by age they lose vision completely المشكلة أنهم you should know it"

Keywords		
Diabetic retinopathy	**Chronic glaucoma**	**Age-related macular degeneration**
k/c of diabetes , Dot & blot hemorrhage.	High intraocular pressure.	More than 65 years.

MCQs
"نص الـ MCQs هو acute & chronic visual loss ما فيها كلام، مع شوية amblyopia على شوية squint على الـ optic neuritis يعني خلاص إذا عرفتوا هذه المحاضرة you'll know how to answer the exam كل lecture بيجي منها 1 question at least إنما كلهم بيجتمعوا في الآخر على ال causes of acute & chronic visual loss"

"الكلمات مهمة، if you don't know the term you'll not answer the question in the exam"

8. Refractive Errors. "محاضرة بسيطة وظريفة"

- **Myopia** قصر نظر light rays so they focus in front of the retina, treatment? Concave lens
- **Hyperopia** طول أو بعد نظر incoming light rays so they focus Behind the retina, treatment? Convex lens
- **Astigmatism**
- **Presbyopia:** 40 عدم القدرة على الرؤية في القريب للكبير في السن، يعتبرون الكبير في السن

(حَتَّىٰ إِذَا بَلَغَ أَشُدَّهُ وَبَلَغَ أَرْبَعِينَ سَنَةً قَالَ رَبِّ أَوْزِعْنِي أَنْ أَشْكُرَ نِعْمَتَكَ الَّتِي أَنْعَمْتَ عَلَيَّ) (سورة الأحقاف - آية 15)

What happens at age 40:

1. Loss of elasticity of lens capsule (in other words increase density).
2. Loss of accommodation.
3. Increase the refractive index of the lens.

"يعني ايه elastic lens؟ الـ lens بتاعتنا ربي خلقها بتكبر وتصغر، لما أجي أبص بعيد الـ lens automatically بتبقى thin، لما أجي أبص قريب الـ lens بتبقى bulging، زي كأنها تعملت نظارة، كأني لابسها. هذا في عمر أقل من أربعين سنة، بعد الأربعين سنة يحصل الـ **physiological** presbyopia"

You need to know the types of treatment: Lens, **lasik surgery when do we do it?** Age of 21

MCQs: Example in the exam: 7 years old patient has difficulty with reading.
One of the options is presbyopia ✘ (not the answer)

9. Ocular manifestations of systemic diseases. MCQs & SAQs "انتبهوا للصور"

- Chronic uveitis.

- **Tuberculosis (TB) of the eye:** infection in the eye → chronic uveitis.
- **Sarcoidosis:** infection in the eye → chronic uveitis.
- **Behcet's disease:** skin/genital/mouth ulcers + uveitis (inflammation of the uveal tissue) + sometimes loss of vision + sometimes they even get hypopyon of the eye.
- **Vogt-koyanagi-harada syndrome.**

- Diabetes mellitus.

- Hypertension: hypertensive retinopathy.

SAQs: *picture from the same lecture* child with deafness, has undergone cataract surgery in both eyes and presbyopia "لابس نظارة بتاعت الـ دي"

Q: What is the diagnosis? Ocular manifestation of **Rubella syndrome**

د. عصام: "لما جا مره بالـ batches اللي قبل ما عرفوا يجاوبوه! موجود في المحاضرة والصورة في المحاضرة، ويكتبوا شكوى،[هازا ما تدرّس في الـ curriculum] يا ابن الحلال، هازا في المحاضرة! لما يجينا حاقه زي كده i never answer them، لأن أخليه يروح يشتكي وحيسقطوه أساسًا في الامتحان! طبعًا الـ exam will not come from lecture لكن if you take the lecture carefully أنا واسق أن كلكم حتجيبوا A+ إزا حتى ما قريتي from the lecture"

10. Neuro-ophthalmology.

Very nice lecture. It will talk about cranial nerve palsy (especially **CN III, IV**) you have to know how it manifests!

11. Ocular Pharmacology and Toxicology. MCQs 3 Qs "بتهملوها، محدش بيقرأها وفي الغالب بتغلطوا فيها"

- Eye drops & systemic medications.
- Mostly treatment of -which is most prescribed-:
  - **Glaucoma**: anti-glaucoma medication. Patient takes it for life.
  - **Allergies** (temporary): antihistamine or steroids (only prescribed in serious situations and for a short period because of the serious side effects).
- Know the complications (side effects), especially **steroids**: **cataract & glaucoma**

● Clinic حتى لو الدكتور ما كان معاكم بالكلينك يشرح أنتم شوفوا بنفسكم

- You will have rotations in the clinic once or twice during the cycle.
- Sometimes there will be no clinic and you will have to go the emergency. You'll be lucky because you'll get to see common ophthalmic disorders and procedures.
- Most of the time it is busy. Try to ask. Try to engage yourself. خليك غلز (غلظ) شويه
- You should look at the slit lamp. There is an extra tube whether the doctor asked you or not. You're the priority.
- **In Clinics make sure you cover:** Tonometry, Visual field exam, Optical coherence tomography (OCT), Slit lamp, Direct & Indirect ophthalmoscope, 90 lens and Bi… something موو اضح بالركوردنق و لا هو موجود في قوقل

● Clinical skills sessions:

- Topics:

- External Ocular Examination, Ocular motility and Alignment.
- Visual acuity and Ophthalmoscopy.
- Visual field, Tonometry, **Pupil Examination.**

- They are tutorials (like PBL sessions) you have to prepare the topic & discuss it with the group.
- Attend & prepare well. That is all you need.

-" المفروض تاخدوها كاملة، إنما للأسف بيتاخد فيها 3 على الـ commitment يعني لو الدكتور بدأ الساعة تمنية ودخلتي تمنية وربع بتنقصي تلات درجات، حرام! عيب! فتكونو حريصين قدًا قدًا أنه قبل ما يجي تكونون أنتم موجودين كلكم، تاني حاقه بقية النمر بقه على الـ discussion يعني بتعملي الـ test ويشوف how you are doing قاريه و لا مش قاريه ويديك بقية الدرجة. طبعًا صوابعنا مش زي بعضها. تيجي أي small mistake وبعض ال attending يشيل على طول ما تفرقش معاه وبعضهم they sympathise with you بتقولوله مسلًا تأخرت المواصلات… الكلام ده، فأنتو تكونوا حريصين في الـ teaching أنك تيجي على الـ time وتحضر الـ test قبل ما تيجوا "

- **Recommended source:** OphthoBook **www.OphthoBook.com**
- **Side note:** APD (afferent pupillary defect) → occurs with optic neuritis & multiple sclerosis.

● Mark distribution

	Assessment	Questions / stations / sessions	Marks
Same **Day!**	**SAQs**	20 (1.5 minute for every question)	**40**
	MCQs	30 (45 minutes)	**30**
لجنة واحدة	**OSCE**	2 **a)** Fixed: ophthalmoscope. | **b)** Variable: hx or clinical sessions	**20**
	Clinical skills	2	**10**

- Do the evaluation before the exam (2 evaluation forms) so you can get your results early.
- Ophthalmoscopic examination. There is a mannequin in the exam center (at KKUH)

"بنسحبوا قبل الامتحان بأسبوعين، في نص يناير كده، فيه سلايدز وما بتتغيرش، أنا مديهالك:
Artery occlusion, diabetic retinopathy, papilledema إشوفوها قبل الامتحان!
"i'm testing how you're doing the exam ظ we are not testing the diagnosis فيه حاقه قديده عملناها السنة دي، صور مطبوعة

OSCE: ophthalmoscope. You'll get two pictures: retina or squint. (esotropia) "فيه فرق بين اللي يحل أي كلام والعارف"

- **Pupil Examination:** start by saying i'll dim the light, watch red reflex, direct & indirect test or (consensual)... etc [You'll find it in OphthoBook]

SAQ: Chalazion, Entropion or Ectropion, Old trachoma scar "جاء للأولاد بس 2 جاوبوه" & treatment = lubricant, Foreign body.

● References

- Clinical Ophthalmology: A Systematic Approach by:Jack T. **Kanski** جميل جدا تشوفوا منه الصور تلقونه في المكتبة
- **SAQs:** google images is your best friend (look for **chalazion and stye**)

- **Required Text(s): a.** Lecture notes Ophthalmology (latest edition) By: Bruce James (published by Blackwell Science) **b.** Basic Ophthalmology (latest edition) By: Cynthia A. Bradford (latest edition) (published by American Academy of Ophthalmology) **c.** Practical Ophthalmology: A manual for Beginning Residents (latest edition) By: Fred M. Wilson (published by American Academy of Ophthalmology
- Vaughan and Asbury's general Ophthalmology By: Paul Riordan-Eva (published by LANGE)

- **Retinitis pigmentosa**
  Genetic disorder of the eyes that causes loss of vision. Symptoms include trouble seeing at night and decreased peripheral vision (side vision). Onset of symptoms is generally gradual. As peripheral vision worsens, people may experience "tunnel vision". Complete blindness is uncommon.

- **Keratoconus**
  Painless disorder, usually sporadic but sometimes inherited, in which progressive central corneal thinning leads to an ectatic conical shape and marked myopia .With time,stromal corneal scarring occurs,which may further reduce vision.Onset is in youth,with atypical history of myopia which, with the development of irregular astigmatism, becomes uncorrectable by spectacle lenses.

- **Endophthalmitis**
  Intraocular infection may occasionally occur within days following intraocular surgery. It causes a marked generalized increasing conjunctival inflammation. The eye is painful (unusual after routine intraocular surgery) and the vision reduced. A history of recent surgery is the clue treatment: **intravitreal antibiotic injection.**

- **Ocular TB**
  The term “ocular TB” describes an infection by the M. tuberculosis species that can affect any part of the eye (intraocular, superficial, or surrounding the eye), with or without systemic involvement. “Secondary ocular TB” is defined as ocular involvement as a result of seeding by hematogenous spread from a distant site or direct invasion by contiguous spread from adjacent structures, like the sinus or cranial cavity.

Orientation, History Taking, Examination

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

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- To know how to handle common ophthalmic emergencies.
- To handle simple ophthalmic diagnostic instruments.
- To be aware of common ophthalmic operations.

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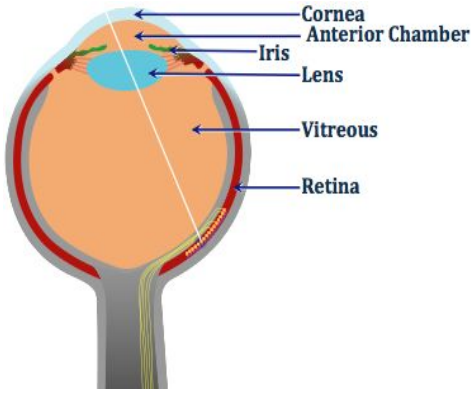
Resources: Slides + Notes + 434 / 435 Team + Lecture Notes of Ophthalmology.

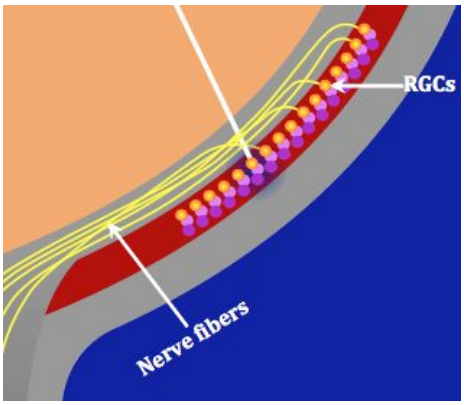
The Visual Pathway & Importance of the Eye

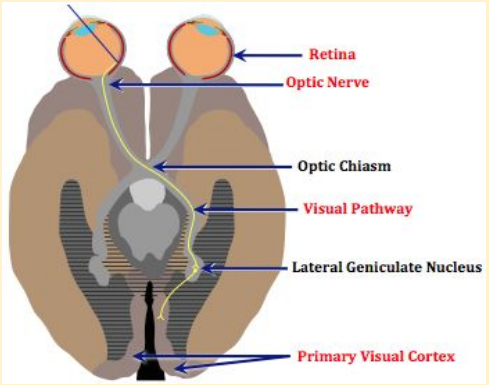
• Visual Pathway:

- Brief idea of the mechanism of vision: Light → cornea → anterior chamber → pupil → lens → vitreous → retina: stimulation of photoreceptors → bipolar cell **(1st order neuron)** → **retinal ganglion cells (2nd order neuron)** → decussation of fibers in the chiasm → lateral geniculate body **(3rd order neuron nucleus)** → perception of light in visual cortex **(3rd order neuron axons)**

1	2	3
- Light enters the eye via the refractive media, namely the cornea, anterior chamber, lens, and vitreous, and stimulates the retina posteriorly. - The **start** of the visual pathway: **light** striking the eye. - You should have a **clear media** to have a good image. What is meant by "clear"? For example some patients have corneal scars "سحابة على القرنية" or vitreous hemorrhage he will not see.	Light stimulates the photoreceptors, ie., the rods and cones. Through a series of other retinal nerve cells, the end result is that the RGC is stimulated. The RGC sends its axon, or fiber, in the nerve fiber layer to the optic disc and then down the optic nerve. ***Phototransduction:** by photoreceptors (rods and cones) ***Image processing:** by horizontal, bipolar, amacrine and RGCs ***Output to optic nerve:** via RGCs and nerve fiber layer.	From the optic nerve, about half of the fibers cross over at the chiasm to the opposite optic tract, and the other half remain on the same side. The fibers in the optic tract synapse in the lateral geniculate nucleus of the thalamus. Neurons in the lateral geniculate nucleus then project to the occipital lobe, to the primary visual cortex. From there, there is further processing with projections to other cells in the visual cortex and elsewhere, resulting in conscious visual perception. Now that we know how visual information is normally transmitted to the brain, what happens with a disease like glaucoma.







SAQ: you need to know the structures!
Q1: What is this? Primary visual cortex
Q2: What is this? Lateral geniculate body

• Importance of eyes:

- Diagnostically and functionally, it is the most important square inch of the body surface.
- The eye is so intimately connected with the rest of the body that it reveals enormous amount of general information: vascular and neurological connections.
- Eye is the only part of the body where blood vessels and central nervous system tissues can be viewed directly.
- 90% of our information reaches our brain via sight.
- Unfortunately, of all the parts of the body, the eye is the most vulnerable to minor injury.

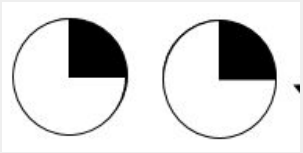
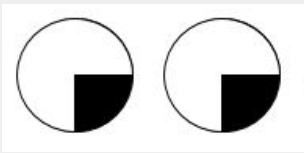
Extra from 435 team
- In order to see, you need **clear media**, but if it's not clear it called **media opacity** (so in **OSCE** the absence of red reflex in ophthalmoscopic exam is called **media opacity**) **Media opacity** caused by (hyphema , scar , cataract , vitreous haemorrhage , retinal detachment).

Pathology of the Eye

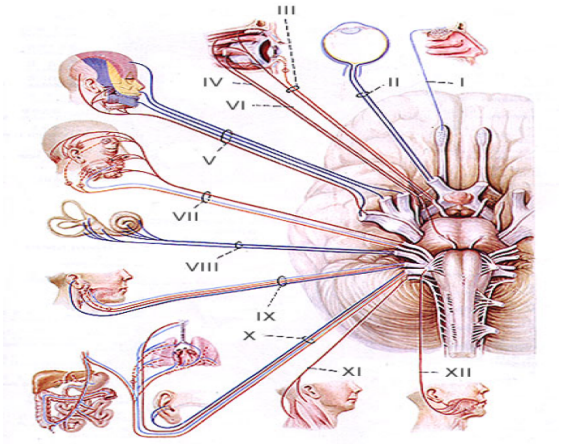
• Neurological connections:

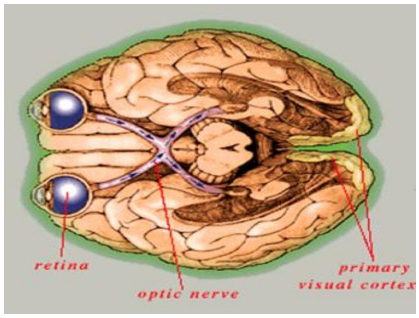
- The 12 **cranial nerves** provide us with a large part of our information about the brain. Of these, the eye examination evaluates CN II, III, IV, V, VI, VII, VIII **(7 CNs)**. you'll have a lecture about it!
- Provides information about the autonomic pathways. (Sympathetic/parasympathetic).
- **The best known connection between the brain and the eye is the optic nerve.**
- Visual pathways, which extends from front to back across the brain can be studied easily and safely using perimeter[1]. It can differentiate accurately between lesions of the **temporal**, **parietal**, and **occipital** lobes.

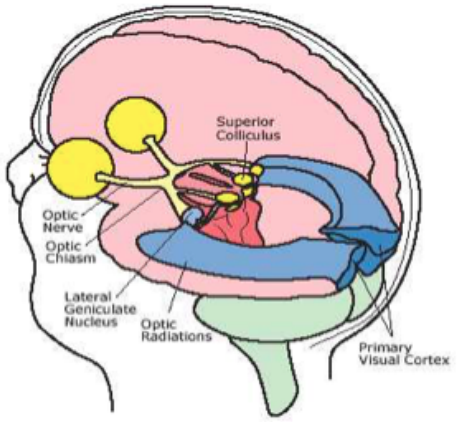
SAQ/OSCE: lesions in:

Temporal lobe: Pie in the sky (upper quadrantic hemianopia)	**Parietal lobe:** Pie on the floor (lower quadrantic hemianopia)
Lesion at **temporal** **lower** optic radiation: Contralateral homonymous **superior quadrantanopia** 	Lesion at **parietal** **upper** optic radiation: Contralateral homonymous **inferior quadrantanopia** 

- **Optic nerve** has important clinical relationships to the pituitary gland, the middle ventricles, the venous sinuses, the meningeal and Bony structures of base of the skull.

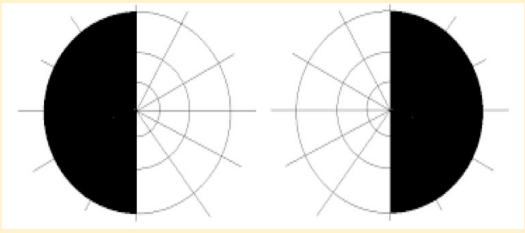




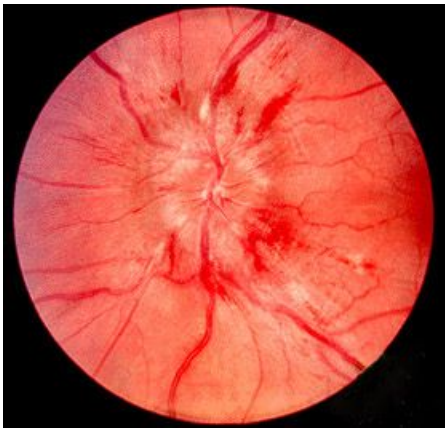
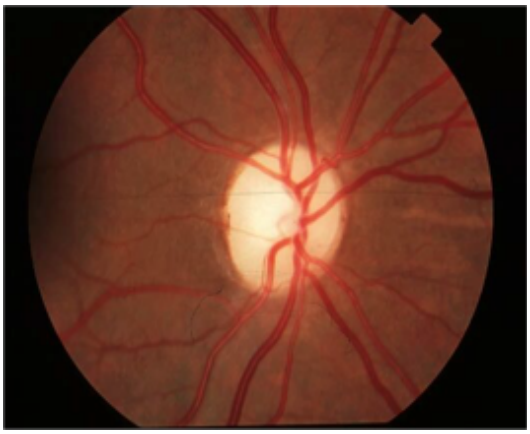


SAQ/OSCE:

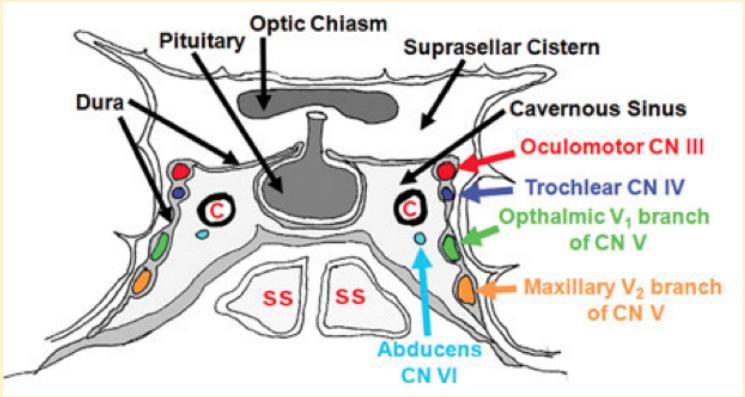
Q1: What is this? Visual field defect, **bitemporal hemianopia**
Q2: What is the reason, give one example? pituitary adenoma
Explanation:"Nasal fibers decussate at the optic chiasm while temporal fibers stay on the same side. When this discussion is touched by the pituitary gland due to an enlargement because of a tumor it will lead to bitemporal hemianopia"

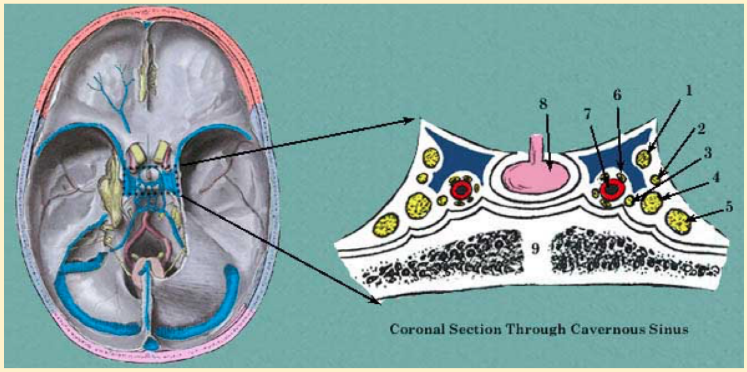


[1] **Perimetry** is the systematic measurement of visual field function.

Optic disc edema	Optic atrophy
Optic nerve has the diagnostically useful capability of congested veins + disc **swelling** and enlargement away from the retina with ↑ ICP (papilledema)	Optic nerve could be visibly **pale** بهتان when its nerve fibers damaged at any point from Retina → LGB.
You have to differentiate between the two pictures!	
	

- Study of **CN III, IV, V, VI** can evaluates the brain stem, cavernous sinus, apex of orbit.





SAQ "هذي الصور لازم تحفظوها صم بكل التفاصيل"

Question: What are the structures in the cavernous sinus?

- In the wall: THREE nerves

- Superior & inferior divisions of CN III **(Oculomotor nerve)**
- CN IV **(Trochlear nerve)**
- Ophthalmic and maxillary divisions of CN V **(Trigeminal nerve)**

- Inside the cavernous sinus: ONE artery + ONE nerve

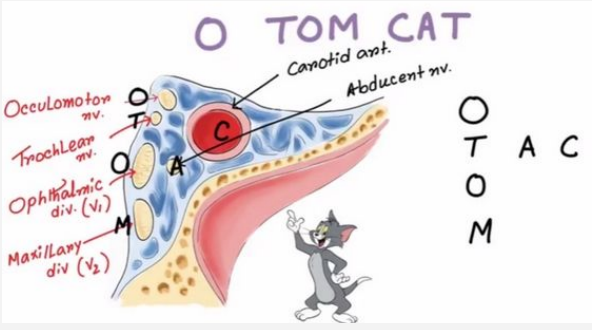
- Carotid artery.
- CN VI **(Abducens nerve)**

"هذي أهميتها لما يجيلك سؤال عن cavernous sinus thrombosis"

Cavernous Sinus Components

Mnemonic:

O TOM CAT

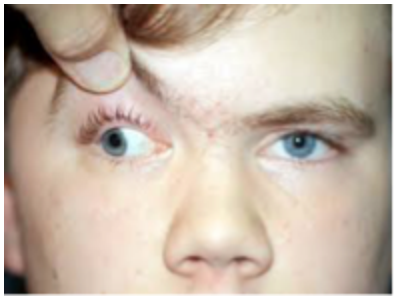


- Unilateral dilated pupil after head injury can occur due to pressure on pupil constrictor fibers of CN III.
- CN VI involved in mastoid infection (petrous ridge)
- CN VII involved in Parotid gland, Inner ear disease
- CN VIII involved in nystagmus.

Very important: third cranial nerve, **oculomotor nerve**:

Fibers	Innervation	Function
Somatic motor (general somatic efferent)	Supplies four of the six extraocular muscles of the eye **and** the levator palpebrae superioris muscle of the upper eyelid.	controlling the muscles responsible for the precise movement of the eyes for visual tracking or fixation on an object.
Visceral motor (general visceral efferent)	Parasympathetic innervation of the constrictor pupillae and ciliary muscles.	Involved in the pupillary light and accommodation reflexes.

- Supplies all the muscles of the eye **EXCEPT** lateral rectus and superior oblique.
- **Paralysis** will lead to: ptosis and squint (eye deviated out and little down). Pupil might be affected.
- **It has two fibers**.
  1) Inside the nerve: deep fibers responsible for the muscles.
  2) Surface of the nerve (periphery): superficial parasympathetic fibers.
- **Muscles only paralysis** is always due to medical reasons. Example: If CN III is affected due to ischemia because of DM or HTN, the fibers supplying the muscles will be affected.
- If there is a tumor compressing the periphery it will affect peripheral and central part of the nerve. There will be **muscles paralysis & pupil dilation** (loss of parasympathetic activity → unopposed sympathetic). It is an emergency situation. (surgical)
- **The difference between medical & surgical third nerve palsy is pupil affection.**



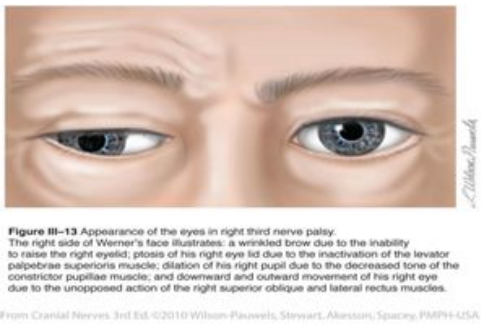
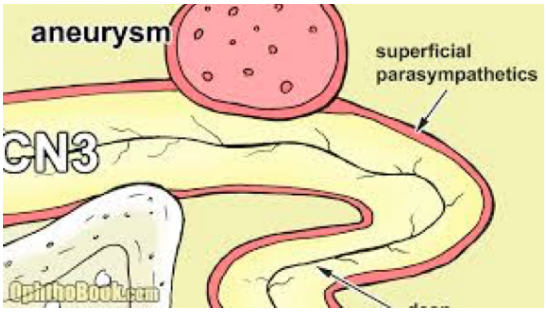


Figure III–13 Appearance of the eyes in right third nerve palsy. The right side of Werner's face illustrates: a wrinkled brow due to the inability to raise the right eyelid; ptosis of his right eye lid due to the inactivation of the levator palpebrae superioris muscle; dilation of his right pupil due to the decreased tone of the constrictor pupillae muscle; and downward and outward movement of his right eye due to the unopposed action of the right superior oblique and lateral rectus muscles.

From Cranial Nerves, 3rd Ed. ©2018 Wilson-Pauwels, Stewart, Akesson, Spacey, PMPH-USA



- Focal brain lesion like: vascular occlusions, hemorrhage, neoplasm.
- Diffuse brain lesion like: Infections, demyelinating disorders (nerve damage).

Extra from 435 team

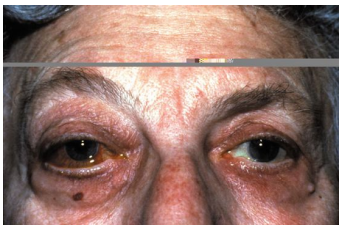
- Cranial nerve: CN II(Optic): Visual Acuity, CN III(Oculomotor): Eye Movement + Pupil Examination, CN IV(Trochlear): Superior oblique Muscle, CN V(Trigeminal): Sensation, CN VI(Abducent): Lateral Rectus muscle, CN VII(Facial): Facial Nerve paralysis (they can not close their eyes), CN VIII(Vestibulo-cochlear): Nystagmus.
- Remember: sympathetic: dilatation of the pupil/Parasympathetic: constrict the pupil.
- Binasal hemianopia happens if there is compression in the carotid due to pressure on the temporal fiber.
- Optic disc edema (**unclear disc margin** best answer will be disc edema in OSCE, rather than Papilledema) to detect increase in ICP help to diagnose **brain tumor**.
- Brain Herniation, Hemorrhage, Aneurysm of posterior communicating artery can compress CN III.
- Mandibular nerve or third division of trigeminal nerve pass through foramen ovale.
- Phacomorphic glaucoma (cataract induced glaucoma by increase lens size that lead to increased IOP) > so severe pain.
- Commonest cause of abducens nerve palsy? TRAUMA. Why? because it has a very long course and each course is perpendicular to each other
- **QUESTION:** investigation if you have a patient with 3rd nerve palsy with dilated pupil? Order a CT **ANGIO** or MR**A (angio)**
- How do we know if CN IV is involved in addition to CN III ? Patient will not be able to **look down** (damaged CN III) and Eyes will **not intort** (damaged CN IV).

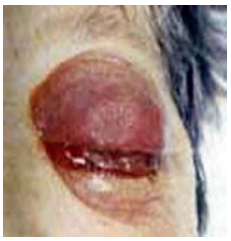
• Vascular connections:

- Venous flow disorder:

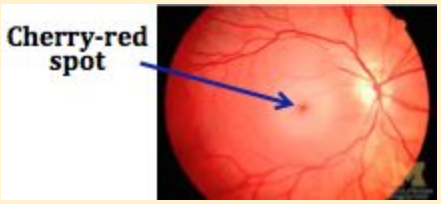
- Cavernous sinus thrombosis. (occlusion). Cavernous sinus is the venous drainage of the eye. The eye will be bulging, injected, congested sometimes with paralysis because the nerves are in the cavernous sinus
- Carotid – cavernous fistula (orbital congestion) eye will be bulging, injected, congested, same as cavernous sinus thrombosis.The difference is that the **eye is pulsating (bruit)**
- Arterial emboli can reach the retina from carotid artery, heart valves, subacute endocarditis and traumatic bone fracture.
- Neoplasm.

Cavernous sinus thrombosis (CST) **OR** Carotid-cavernous fistula (CCF)
Difference: pulsation of the eye with CCF





SAQ



Q: Diagnosis? Central Retinal Artery Occlusion
Q: What do you see? Pale retina in the center + cherry-red spot
"the fovea should not be red like this"

- Specific disease of the vessels like:

- Polyarteritis nodosa (PAN)
- Hypertension
- **Temporal arteritis,** "very important and you have to know it in details"
  - Also known as **Giant cell arteritis (GCA):** it affects the ophthalmic artery not retinal.
  - Autoimmune vasculitis that occurs in old age (>60 years, other sources: >50), common in females.
  - **Signs & symptoms:**
    * **Visual symptoms:** irreversible and usually painless[2] sudden loss of vision, diplopia.
    * **Polymyalgia rheumatica symptoms:** headache, pain in the shoulders and hips,malaise
    * **Others:** jaw and tongue claudications (pain on chewing), ~~pulsating~~ pulseless superficial temporal arteries, scalp/temporal tenderness (eg. on combing),
    * **Fever and constitutional symptoms.**
  - **Diagnosis:** elevated erythrocyte sedimentation rate (ESR) & C-reactive protein (CRP), temporal artery biopsy. If you miss the diagnosis the patient will have loss of vision in the other eye.
  - **Management**: IV Steroids to protect the other eye. Steroids will not reverse the visual loss but can prevent the fellow eye being affected. Unfortunately, there is no treatment for GCA.

Extra from 435 team

- Carotid-cavernous fistula (orbital congestion) (it has the same clinical presentation (proptotic, retinal edema) of cavernous sinus except that it has increased IOP, and Thrill+pulsating **eyes (Bruit)** orbital congestion.
- Bilateral carotid artery aneurysm Binasal Hemianopia.
- **Came in the exam**: central retinal artery occlusion/ history: of multiple bone fraction , what is the diagnosis? Retinal central artery fat embolism.
- Case: a 60 year old pt w/ heart disease and using penicillin injections (meaning that he has bacterial endocarditis and might have a possibility of embolism manifestations leading to central retinal artery occlusion)
- Best initial investigation in temporal arteritis is ESR, followed by C-reactive protein, then Biopsy (a negative biopsy does not rule out the dx due to the focal and segmental nature of the infiltrates).
- When a patient complain of any changes in vision, rule out DM first!

[2] It is certainly **painless** but some references might say **painful** because it might be associated with headache and jaw claudications.

• Hematological disorders

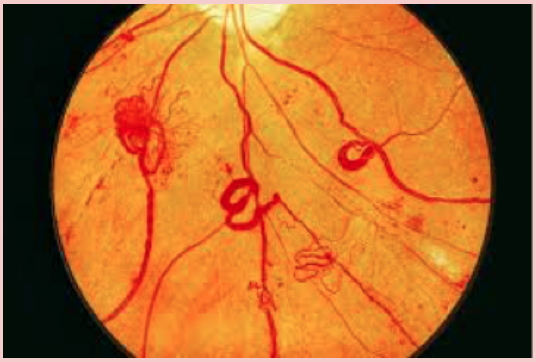
- All types can manifest in the fundus.

Extra from 435 team
- Anemia,leukemia,AIDS(diagnosed by cotton wool spots in the retina, ischemia of the nerve fiber in the retina)

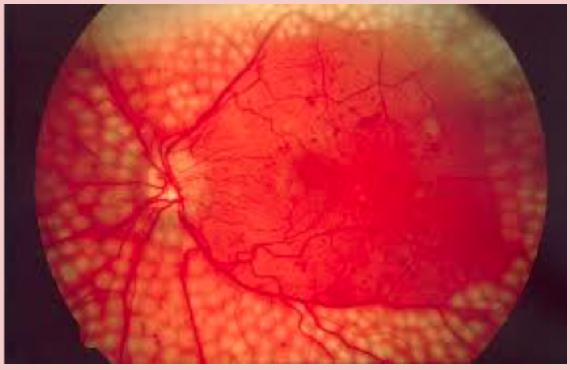
• Metabolic disorders

- Almost all metabolic disorders can affects the eye:
  - **Diabetes Mellitus**: Diabetic Retinopathy (proliferative & non-proliferative), Cataract, Refractive Error, Ophthalmoplegia. The commonest cause of third and fourth nerve palsy is DM.

Exam: "الصورة دي مهمة قدًا"



Proliferative Diabetic Retinopathy (PDR)
New vessels in the retina due to diabetes. The vessels are fragile and can bleed. If it bleeds it will lead to vitreous hemorrhage and retinal detachment.
(New dilated tortuous proliferative vessel in the retina)



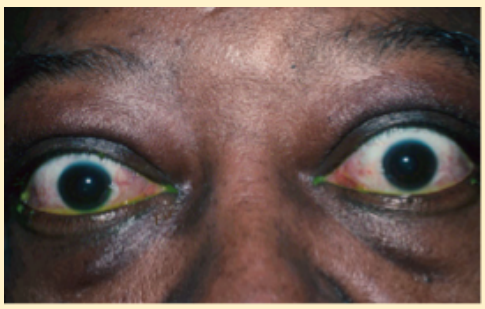
Pan-retinal Photocoagulation (PRP)
Laser treatment of the other picture.
Early detection and treatment will change the ischemic retina to anoxic retina. New vessels will be destroyed and central vision will be saved. There will be visual field defect, but the macula will be saved and the vision will be maintained.
(Retinal scars)

  - **HYPOparathyroidism**: Cataract "انتبهوا مش هايبر"
  - **Wilson's disease**. copper abnormality, deficiency of α-ceruloplasmin. Causes cataract and corneal changes (Kayser-Fleischer, corneal ring: a brownish-yellow ring around the cornea of the eye)
  - **Thyroid eye disease:**
    - Exophthalmos, Lid retraction (upper part of sclera is seen)
    - The **commonest** cause of **unilateral & bilateral proptosis** is thyrotoxicosis.
      "بعض الاقزام يجي يقولك unilateral proptosis بتقولوا خلا ده مش thyroid، لأ! It can be thyroid"

Exam

Manifestation of exophthalmos

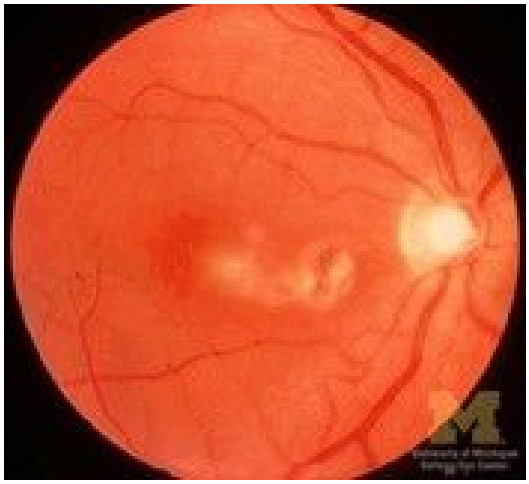
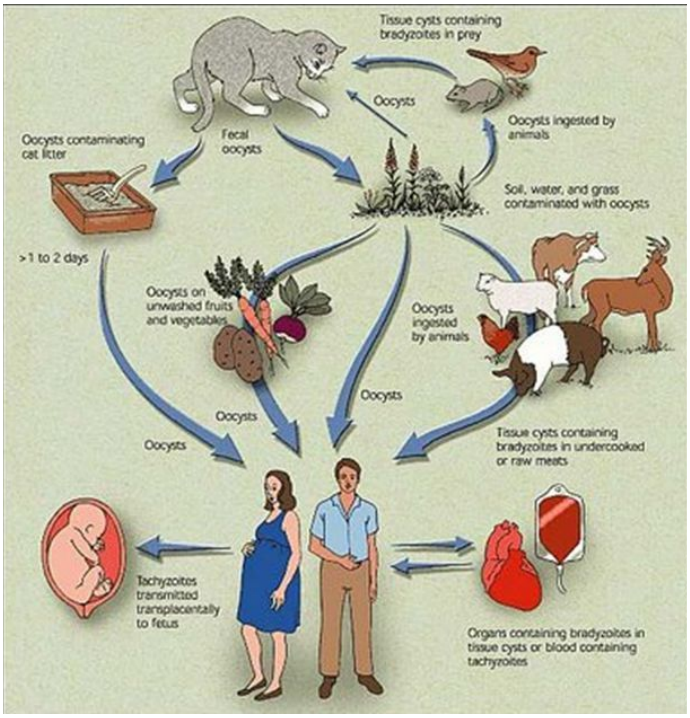
"أحيانًا في السيناريو questions يجي يقولك the upper part of the sclera is seen يعني ايه؟ طبعًا normally أي حد بيص في حد عندكو تلاقوا الـ lid عندكو مغطي الـ upper part of the cornea لو نزل شويه اسمها ptosis لو طلع شويه اسمها lid retraction، فالتعبير في السيناريو بيختلف محد حيجي يقولك exophthalmos or lid retraction محد حيقولك لأ! حيقولك lid lag sign فتفهمي على طول أن ده wide part of the sclera is seen thyrotoxicosis"



Extra from 435 team
- Thyroid eye disease, also known as **infiltrative ophthalmopathy** "Graves ophthalmopathy". Since they have **increased IOP** we perform visual field exam.

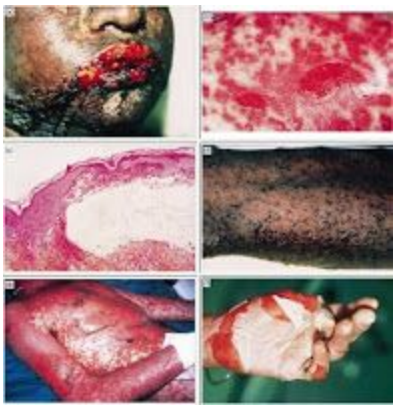
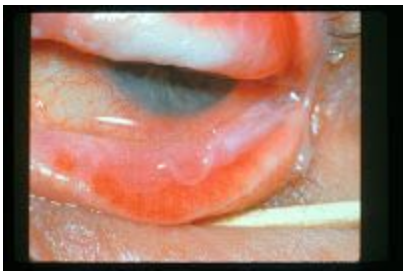
• Infections

- Syphilis, Toxoplasmosis, Rubella. Syphilis and Rubella also can cause retinitis (inflammation of the retina)

Toxoplasma infection	
	

• Mucocutaneous disorders

- Steven-Johnson Syndrome (SJS), Pemphigus.

Mucosal Lesions In Stevens-Johnson Syndrome	Eye Lesions of Stevens-Johnson Syndrome
	

• Elastic tissue

- Pseudoxanthoma elasticum: degeneration of the retina in which the patient can develop neovascularization.

• Chromosomal abnormalities

- Trisomy: 13, 15, 21.

• Eye poisoning the eye is a delicate indicator of poisoning

- **Morphine addict:** lead to meiotic pupil.

Constricted pinpoint pupil = morphine overdose or pontine hemorrhage

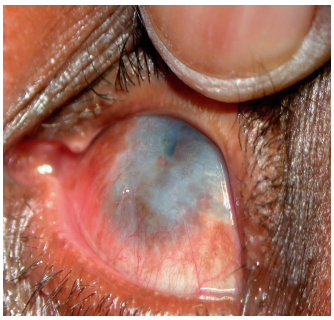
They have the same presentation the only difference is **hyperpyrexia with pontine hemorrhage**.

- Lead poisoning, vitamin A (example for acne treatment) intoxication lead to papilledema.

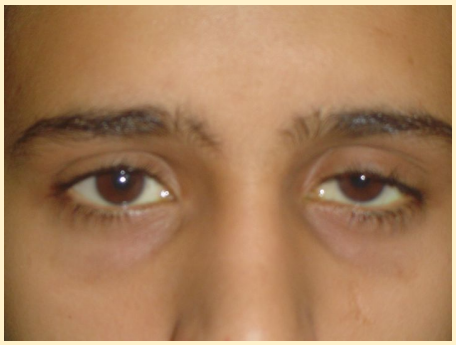
• Allergy

- **Vernal keratoconjunctivitis (VKC)**: causes cobblestone papillae. حساسية الرمد الربيعي

"مشهورة في نجران وجيزان، it is a dusty area و most of the children have allergy فتجيلهم حاقة اسمها vernal catarrh أو spring catarrh، للأسف معظمهم بيروحوا يشتروا steroids من نفسهم، فيجيلهم حاقة اسمها، cataract و glaucoma. هذا الموضوع مهم جدًا و لازم تذاكروا في الـ drugs الـ steroids و الـ complications، طبعًا سواء كان drops أو tablet أو injections أو inhaler even"



Exam: important



An example of steroids overuse with VKC

Q1: What do you see?

Unilateral ptosis in left eye.

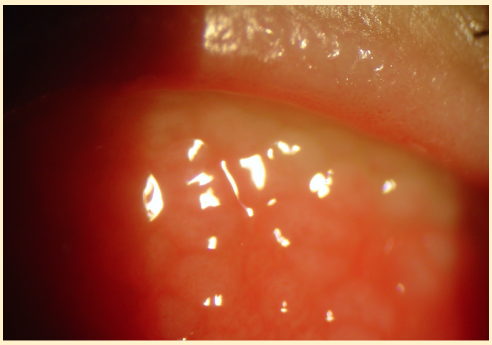
Q2: Cause?

Mechanical ptosis (not paralytic) due to cobblestone papillae.

Q3: Spot diagnosis?

Vernal keratoconjunctivitis (VKC)

or vernal catarrh or spring catarrh.



Q1: Name of the lesion?

cobblestone papillae.

Q2: Type of glaucoma may develop and why?

Steroid- induced glaucoma (Secondary glaucoma).

Chronic use of topical steroids lead to increased IOP

Extra from 435 team
- We start the treatment of VKC with Antihistamine and cold water and leave steroid as the last resort although it is the best treatment for them but can lead to glaucoma and cataract.

Ophthalmic Evaluation

Objectives of the comprehensive ophthalmic evaluation

- Obtain an ocular and systemic history.
- Determine the optical and health status of the eye and visual system.
- Identify risk factors for ocular and systemic disease.
- Detect and diagnose ocular diseases.
- Establish and document the presence or absence of ocular symptoms and signs of systemic disease.
- Discuss the nature of the findings and the implications with the patient.
- Initiate an appropriate response. e.g. further diagnostic tests, treatment, or referral.

History Taking

- Ocular and systemic history.
- It is a gathering information process from the patient guided by an educated and active mind.
- It is a selective guided and progressive elicitation and recognition of significant information.
- History by skilled person can arrive at the proper diagnosis in 90% of patients.
- It gives vital guidance for: physical examination, laboratory work and therapy.
- Failure to take history can lead to missing vision or life threatening conditions.

Chief complaint "Patient's own words"

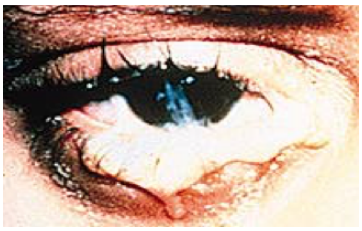
- "She cannot see with the right eye", you should not come to conclusion that her problem is nearsightedness and write down "Myopia of right eye (RE)".
- The patient needs will not be satisfied until he/she has received an acceptable explanation of the meaning of the chief complaint and its proper management.

History of presenting illness

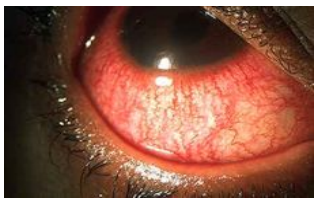
- Detailed description of the chief complaint to understand the symptoms and course of the disorder.
- Listen and question and then write down in orderly sequence that make sense to you.
- **Questions to ask:**
  - **The time sequence when, How fast, what order did events occur?**
  - **Frequency, intermittency.**
  - **Location, Laterality.**
  - **Severity.**
  - **Associated symptoms.**
  - **Documentation (old records, photo) e.g ptosis, proptosis, VII N palsy.**
- **Examples:**
  - Gradual painless decrease vision both eyes for 1 year.
  - Sudden painless decrease vision of right eye for 10 minutes.
  - "cannot see with my right eye"!!
    - Only distance vision blurred?
    - Blind spot is present in the center of visual field?
    - Right side of visual field of the right eye lost?
    - Right visual field of both eyes lost?
    - A diffuse haze obscures the entire field of right eye?
  - Each of these has different diagnostic implication.
- Most patients. have difficulty providing precise and concise description

Disturbances of vision	Ocular pain or discomfort
- Blurred or decreased central vision. - Decreased peripheral vision. - Altered image size: micropsia زغيرة, macropsia كبيرة, **metamorphopsia** معوجة - Diplopia (monocular, binocular) double vision - Floaters خيالات، الذبابة الطائرة (like with myopia) - Photopsia (flash of light) - Color vision abnormalities. - Dark adaptation problems. - Blindness: ocular, cortical. - Oscillopsia (shaking of images).	- Important question to differentiate between mild and severe pain: "حسأل سؤال واحد: أنت صليت الفجر ولا ما صليت؟، بيقولك أنا ما بنامش لغاية الفجر، شوفوا الكلام هنا مهم ما بيقولك أنا ما بيجيليش نوم إنما بيقولوك آه الحمدلله أنا نمت وصحيت وصليت الفجر، معنى ده ما بيكونش pain خالص يكون itching أو foreign body sensation أو حرقان في العين، وفيه ناس بتعمل مشاكل عشان الـ sick leave، بس أنا عايز أعرف هو mild ولا severe " - Foreign body sensation - Ciliary pain: aching, severe pain in or around the eye, often radiating to the ipsilateral forehead, molar area. - Photophobia. - Headache, like migraine. - Burning. - Dryness. - Itching: patient rub the eye vigorously (allergy) - Asthenopia (eye strain)
MCQs: **(1) metamorphopsia** is a sign of age-related macular degeneration. **(2)** 23 years old male with high myopia complaining of **photopsia** & **floaters**. **Diagnosis?** **Retinal detachment (RD)** [RF: young & myopic]	

Abnormal ocular secretions like with conjunctivitis

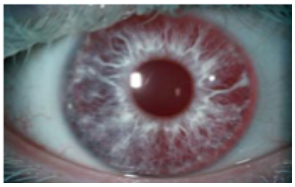
Lacrimation, epiphora	Discharge	
		• Purulent • Mucopurulent • Mucoid • watery

Dryness

Redness, opacities, masses	Anisocoria	
		* **Anisocoria:** different pupil sizes Occurs with third nerve palsy, topical medications or trauma to the eye. * **Anisoconia:** different corneal sizes Occurs with congenital glaucoma "One cornea is big and the other is small"

• Family history

- Many eye conditions are inherited: RE, glaucoma, strabismus, retinoblastoma, neoplastic, vascular disorders.
- Familial systemic disease can be helpful in ophthalmic evaluation and diagnosis: Atopy, thyroid diseases, DM, certain malignancies.
- Ask about any eye problem in the family background? specifically about corneal diseases, glaucoma, cataract, retinal diseases or other heritable ocular conditions.
- Ask questions designed to confirm or exclude your tentative diagnosis. ''Significant is equal to expected''
    - Significant positive.
    - Significant negative.
- Predict the physical and lab. finding likely to be present.
- Any discrepancy between the history and physical examination requires explanation

Albinism (family history is important)			
			

OSCE: you might have a history station with an SP. "لا قدر الله"

Example: a case of chronic visual loss, how to approach the patient?

1. Always start with "Hello, I'm a fourth year medical student, can I take history from you?"
2. Start asking questions about the causes of chronic visual loss, example:
   - What is your **age**? (why? To exclude or if it goes with age-related macular degeneration)
   - Did your vision decreased **suddenly** or **gradually**? (why? To differentiate **acute** from **chronic**)
   - Are you diabetic (why? To exclude or if it goes with diabetic retinopathy)
   - Are you taking any antiglaucoma medication? Do you have family history of glaucoma? (To exclude or if it goes with chronic glaucoma)

Note: if the answer to all the above mentioned questions is no then **cataract** is the most likely diagnosis.

- Are you taking steroids?

- Don't take patient's words literally.

" لو نزلتوا الطوارئ! مشاكل، بيجي المريض يقولك أنا مش شايف! تجي تكشفه تلاقيه بيشوف 6/6، الـ patient كان في البيت ما بيشوفش، بيقولك قعدت ربع ساعة ما شوفتش بس بعديها شوفت فلازم تاخذ منه التفاصيل!"

- Questions to ask:

- **Duration**: was it all day? In 2 days? 1 week? 1 year?
- Is it **temporary**?
- Do you have blurry vision?
- Is there a zigzag line? (sometimes it starts in one eye then it goes to the other eye. The patient close her/his eye for 20 minutes and every 5 minutes she/he opens her eyes to see if it disappears)

- Examples of transient visual loss (amaurosis fugax):

- Transient ischemic attack.
- Migraine:
  - Not a disease at all and does not interfere with life but يخلي نفسيتك زي الزفت
  - Luckily it goes away. The attacks com daily when you're young → weekly → monthly by age 30 → decreases by age 40 → once in a year by age 50 → goes away by age 60.
  - More common in females
  - **Pathophysiology:** vasoconstriction of cerebral and retinal blood vessels followed by vasodilation.
  - **Example of typical history of migraine:**

"واحد قاعد يزاكر ومرهق وبعدين عينو بتزغلل بعدين ما بيشوفش كويس وبعدين بيشوف أو لما يجي وقت الـ exam بيجي في آخر الـ exam يقولك عيني زغللت ومش شايف وأبتدي أصدع عاوز أرجع"

  - When they have attack they: close their eyes, dim the light or keep away from people.

"بيقولوا ده ما بيكلمناش ده انسان مش كويس، هذا الـ impression للي بيجي الناس اللي حواليه، بيقولوا دي على طول كذا بتتغير، لا هيا مش بتتغير دي جالها migraine ومش قادرة تتكلم مع حد وبتقفل النور، الغالب لو أنو غسل راسو بميه ساقعه (باردة) بيبقى أفضل"

Advice from doctor: if you have a migraine take Inderal 10 mg before the exam to prevent the attack.

- It is important to differentiate between **vision loss** & **blurry vision**!

"بيقولك أنا مش شايف وهو في الحقيقة شايف وبيسوق بس بيخبط عمال يعمل accident لأن عندو فيه defect في الـ periphery في الـ visual field، زي الناس with advanced glaucoma الـ visual field بتاعو مضيع ما يشوفش الناس اللي حواليه فيعمل accident"

Extra from 435 team

- **Example** of pt who has acute loss of sight or blurred vision for 20 minutes only or less? Migraine is the best example of a patient who will come complaining of loss of sight(blurred vision) with headache and nausea.
- **Important Terms**:
    - Asthenopia is tired and fatigue eye
    - Red eye has two types: one around the limbus called ciliary injection PAINFUL and one in the periphery called conjunctival injection NON-painful
    - Anisometropia different refractive errors between both eyes
- Floaters (pt see something moving around and nobody else see it. this is because that there is an object in vitreous chamber when crosses the central part of vision > pt see it. pt with DM or have hemorrhage in eyes will have floaters).
- If the patient can't see ask about distant or near because the patient might be more than 40 years old and cannot see near which is normal for this age which is called Presbyopia.
- **DDx of Acute Visual Loss**
    1. Age related macular degeneration(AMD).
    2. Vascular occlusion(DM,HTN).
    3. Retinal detachment.
    4. Acute glaucoma.
        - **Morning** visual disturbances > Eye **dryness**.
        - **Blurring** of vision for **20 min** > Migraine aura .
        - Patient still complains of problems with his\her vision although all his exams and investigation are **normal** > Do visual field exam (could be stroke!).
        - There are two types of color blindness. complete and partial (most common), both are diagnosed by ishihara's test.
    5. A "Convergence insufficiency" is a condition that can cause pain especially in pediatric patients.
- Refractive errors never cause pain Ciliary injection (red eye) is mainly caused by 4 things:
    1. Acute conjunctivitis
    2. Acute iritis
    3. Acute glaucoma
    4. Acute keratitis (corneal ulcer; a combination of conjunctivitis and iritis)
- Foreign body sensation (ask pt about to have hair or sand in their eyes)

Physical Examination

- **The purpose is to evaluate:**

1. Function:
- Visual.
- Non-visual:
  - Eye movement.
  - Alignment.

2. Anatomy.
- The adnexa (lid and periocular tissues)
- The globe.
- The orbit.

The picture illustrates how ophthalmic examination changed over the years from a simple torch exam to the slit-lamp.

- **Ophthalmic examination:**

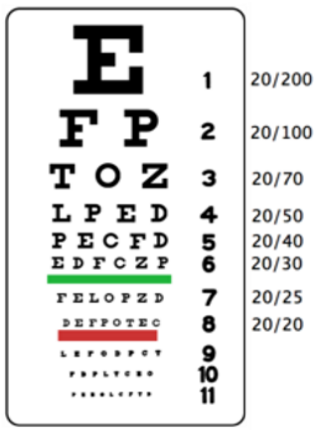
1. Visual acuity.	**2. External examination.**	**3. Motility and alignment.**
4. Pupil examination	**5. Slit lamp biomicroscopy.**	**6. Tonometry**
7. Ophthalmoscopy	**8. Gonioscopy** "don't forget it"	**9. Retinoscopes**

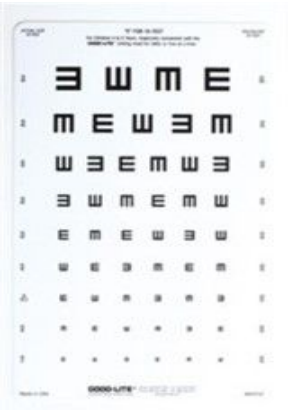
1. Visual acuity: subjective

- **Vital sign (MUST)** first thing the nurse checks in the clinic: blood pressure, temperature & visual acuity. Sometimes the optometrist checks the ocular alignment, example: child with a squint. Sometimes measuring the intraocular pressure (IOP) with air-puff Tonometry.

- **Good vision:**
  - Intact neurological visual pathology.
  - Structurally healthy.
  - Proper focus.

- **How to test vision?** You'll take it in details later on.
  - Display of different-sized targets shown at a standard distance from the eye.
  - Snellen chart. | 20/20, 6/6 | Uncorrected, corrected

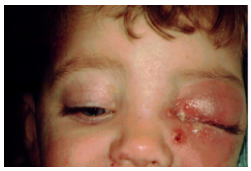
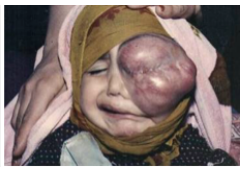
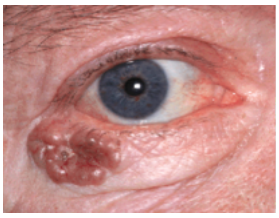
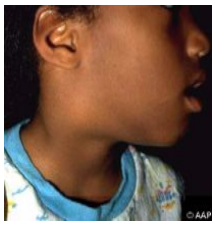
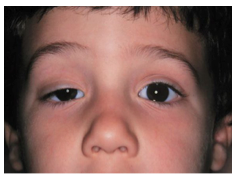
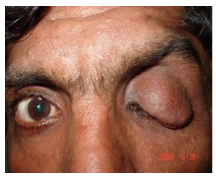
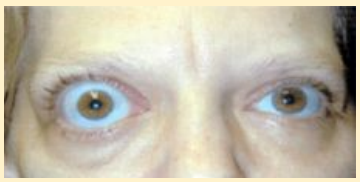




- **How to test poor vision?**
  - If the patient is unable to read the largest letter < (20/200), Move the patient closer e.g. 5/200.
  - **If patient cannot read:** "هذي تحفظونها صم! ومحد يسألنا في الامتحان يعني ايه"
    - **Count fingers (CF)**
    - **Hand motion (HM)**
    - **Light perception (LP)**
    - **No light perception (NLP)**

2. External examination

- Evaluate by gross inspection and palpation.

Ocular adnexa. (lid periocular area), Skin lesions, growths, inflammatory lesions	**Palpation of bony rim, periocular soft tissue**	**General facial examination** e.g. enlarged preauricular lymph node, temporal artery prominence
  **Retinoblastoma** IIt is important to check the red reflex, if white (leukocoria) you need to exclude retinoblastoma	 **Rodent ulcer** (basal cell carcinoma)	 **Oculoglandular syndrome:** Eye infections + enlarged preauricular lymph node
Ptosis "drooping of the eyelids"	**Proptosis, exophthalmos, enophthalmos "مهمة"**	
  **Right:** severe ptosis (might be mechanical or aponeurotic ptosis)	 **Exophthalmos:** العين خارجة برا	 **Enophthalmos:** عين طبيعية وعين داخلة على جوه

3. Motility and alignment

- Alignment, misalignment of the eyes:

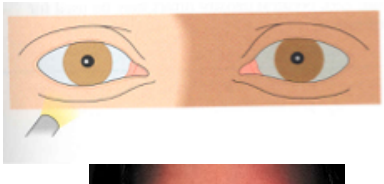
SAQ/OSCE: Q: What is this?
Esotropia (convergent squint)

"لو بتكتبي squint بس في الـ exam بتاخدي ZERO، ولو بتكتبي medial squint = ZERO مفيش حاجة اسمها medial، عيب! لو جبت واحد فراش بيقول medial squint، لازم تعرفوا الـ terms"
Esotropia: convergent الحول الأنسي | **Exotropia:** divergent الحول الوحشي

- Movements

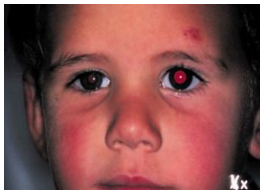
- Follow a target with both eyes in each of the four cardinal directions of gaze.
- **Note:** speed, smoothness, range, symmetry, unsteadiness of fixation (e.g nystagmus)

4. Pupil examination

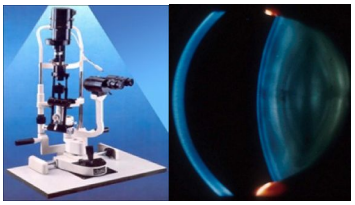


- Examine for size, shape reactivity to both light and accommodation.
  - Direct response and consensual response.
  - Afferent pupillary, defect (Marcus Gunn pupil)
  - Efferent pupillary defect.

- Pupillary abnormalities: Neurologic disease. previous inflammation- adhesion, acute intraocular inflammation, spasm, atony, prior surgical trauma, effect of systemic or eye medication, benign variation of normal



5. Slit Lamp Examination:

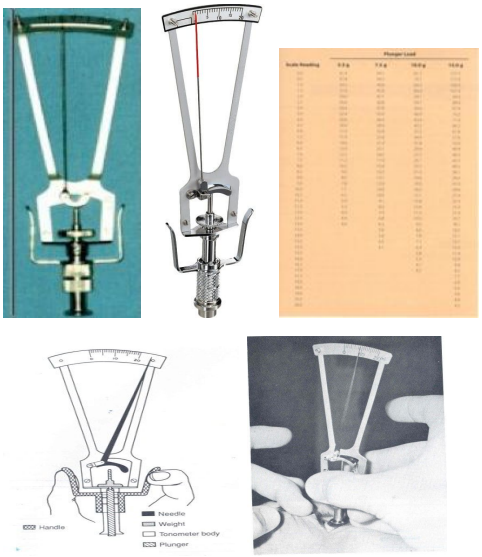


- Is a table-mounted binocular microscope with special illumination source.
- A linear slit beam of light is projected onto the globe – optic cross section of the eye.
- Slit lamp alone, the anterior half of the global (anterior segment) can be visualized.

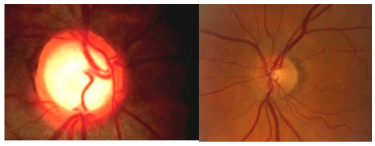
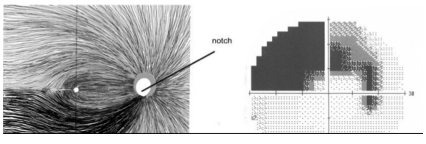
You should know what you're looking at! You'll see Cornea → anterior chamber → lens

6. Tonometry: important

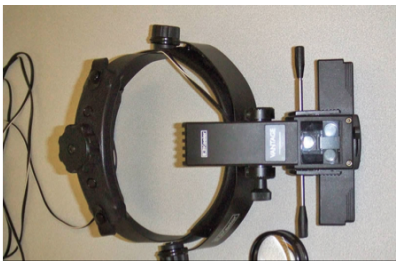
- The globe is a closed compartment with constant circulation of aqueous humor.
- This maintains the shape, and relatively uniform pressure within the globe.
- **Normal pressure 10 – 21 mmHg.**

Types of Tonometry		
Schiotz Tonometer depends on ocular indentations	**Digital Tonopen Tonometer** Used with children.	**SAQ:Identify? + Use?** Measures IOP **Goldmann Applanation Tonometry**
		
Perkin Tonometer	**Air-puff Tonometer** في استقبال العيادة	
		

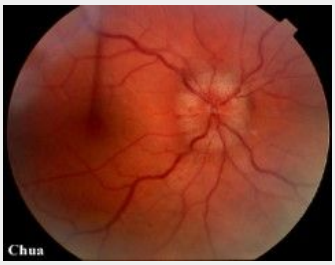
- **Glaucoma:** intraocular pressure (IOP), disc, visual field defect (VFD). Optic nerve disorder manifested by VFD.

Optic cupping	Retinal nerve fiber layer & VF
	

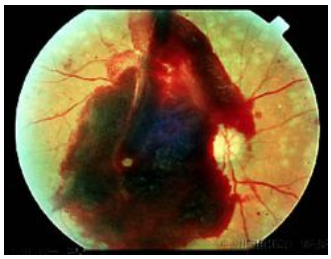
7. Ophthalmoscopy you should know the difference between direct & indirect.

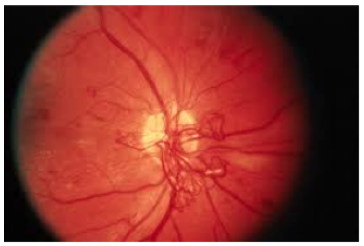
Direct you'll take it in details later on	**Indirect**
- Handheld instrument. - Standard part of the general medical examination. - Portable  " بتقولوا طيب ما درستونا الـ indirect بس you should know the difference!" **Direct vs indirect:** * Large image | Small image * Monocular | Binocular * Virtual Erect | Real Inverted	1. Provide much wider field of view. 2. Less magnification (3.5X with 20D lens) 3. Brighter light source – better view. 4. Binocular – stereoscopic view. 5. Allow entire retina examination till the periphery. **Disadvantage:** 1. Inverted retinal image. 2. Brighter light is uncomfortable to the patient.  

Central vein occlusion



Optic disc edema



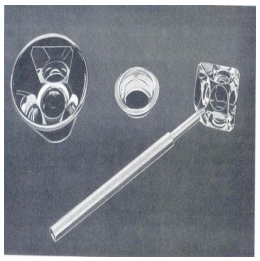


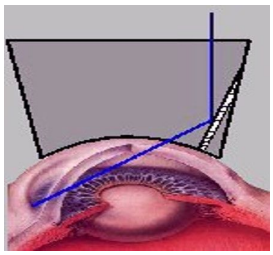
Proliferative diabetic retinopathy

Hemorrhage (vitreous) | New vessels in the dic

8. Gonioscopy important

- lens to test anterior chamber angle to differentiate between open/closed angle - acute/chronic glaucoma.
- Special lenses: goniolens.
- Other lenses allow evaluation of the posterior segment.





IMPORTANCE OF GONIOSCOPY

9. Retinoscopy refraction بنعمل بيه الـ

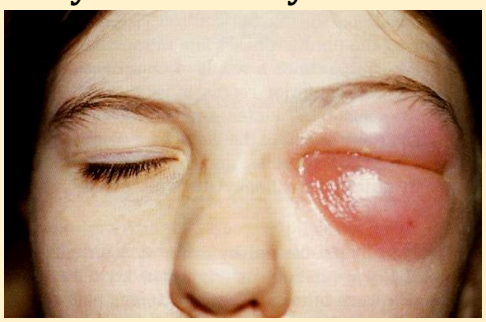
Questions

MCQ: A 10-year-old boy came for his annual eye check-up. Upon examination, the doctor noticed cherry red spot in the macular area. What could this sign represent?

A. Hydroxychloroquine toxicity.
B. Niemann pick disease.
C. Recurrent Optic neuritis
D. Severe Central Retinal Vein Occlusion.

Answer: B

SAQ: 34 year old lady with history of sinusitis "هز ا بالزبط اللي يجيلكم"



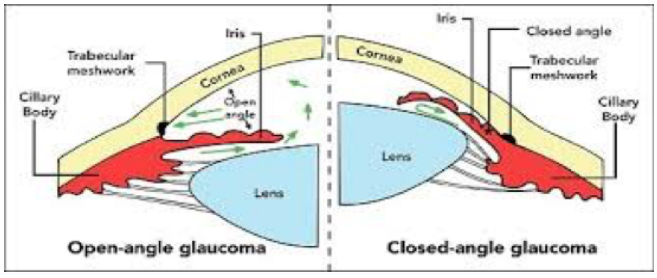
Q1. What is your diagnosis?

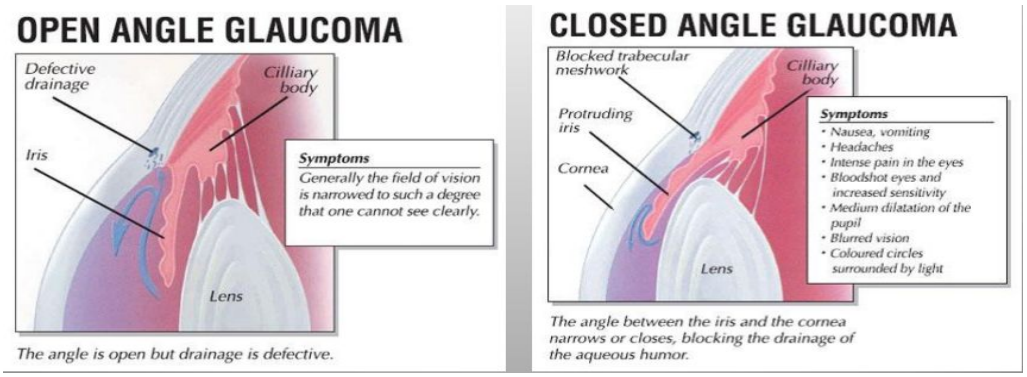
Orbital cellulitis (if no history of sinusitis → preseptal cellulitis)

Q2. What is the treatment? IV antibiotics

A patient with hx of **sinusitis** will get **orbital cellulitis (limited ocular motility)** and the treatment is **IV Antibiotics.**
If she **didn't have sinusitis** then its **preseptal sinusitis (full ocular motility)** and treatment is **oral Antibiotics**

Extra from Slides





Basic Anatomy & Physiology

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

Objectives:

- Touch embryology of the eye.
- Explore anatomy of the orbit
- Explore anatomy and physiology of EOM.
- Explore anatomy of the eyelid and conjunctiva.
- Explore anatomy of the globe .
- Explore anatomy of the visual pathway.
- Understand the physiology of: vision, accommodation, pupillary reflex & tear drainage system.

Done by: Lamya Alsaghan, Gadir Assiry, Razan alSabti.
Edited & revised by: Lamya Alsaghan.
Resources: Slides + Notes + Lecture Notes of Ophthalmology + 434 / 435 Team.

Embryology & Development

• Embryology of the eye

- This highly specialized sensory organ is derived from germ cell layers:

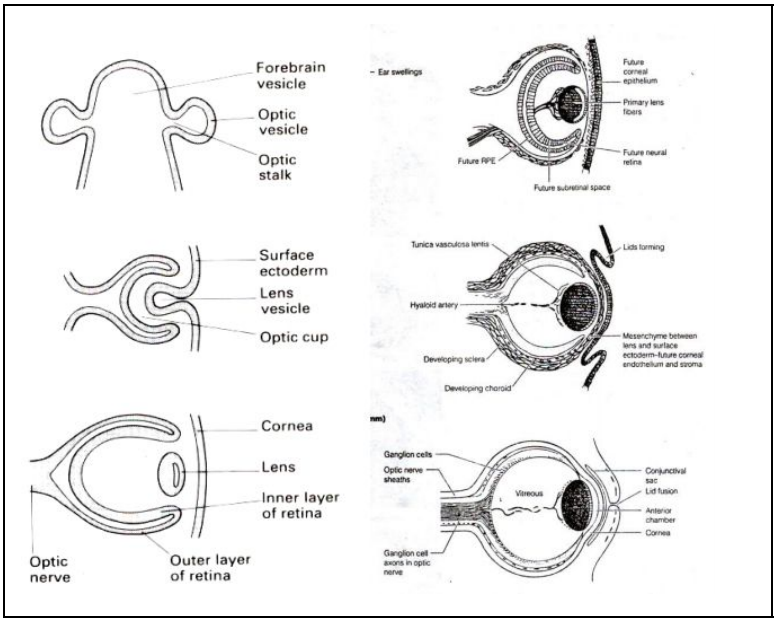
1. **Mesoderm:** give rise to **ciliary body and muscles** (**vascular**, **muscular** & **supportive tissues**)
2. **Ectoderm:** **END**ODERM does not contribute in the eye embryology.
    a. Neural ectoderm give rise to **retina and optic nerve** + epithelium of the iris and conjunctiva
    b. Surface ectoderm give rise to the **epithelium of the cornea and lens**

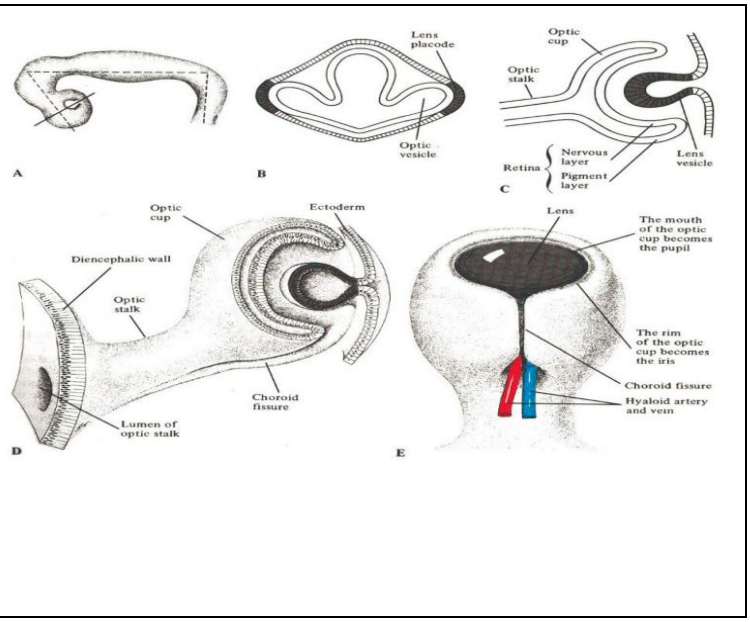
An interesting piece of information:
Part of the cornea, all of the lens (surface ectoderm invaginate to form the lens vesicle) and skin originate from the ectoderm, that is why:
* Atopic dermatitis (eczema) is usually associated with cataract.
* Very painful. Example: when you hit your eyes with the edge of the paper → corneal abrasion → pain.

SAQ: you might get a picture of corneal abrasion.
Q1: identify the picture: corneal abrasion **Q2:** What is the stain used? Fluorescein **Q3:** treatment: eye patch

* Cornea and lens are the **only** 2 parts in eye that can be changed surgically until now.
Keratoplasty for the cornea. **Pseudophakia:** replacing the lens with an artificial lens in cataract surgery.
الجلد القرنية والعدسة لهما نفس المنشأ، وهي الأجزاء التي يمكن تبديلها في العين (إِنَّ الَّذِينَ كَفَرُوا بِآيَاتِنَا سَوْفَ نُصْلِيهِمْ نَارًا كُلَّمَا نَضِجَتْ جُلُودُهُم بَدَّلْنَاهُمْ جُلُودًا غَيْرَهَا لِيَذُوقُوا الْعَذَابَ ۗ إِنَّ اللَّهَ كَانَ عَزِيزًا حَكِيمًا) (سورة النساء - آية 56) يقصد بالعذاب هنا "الألم"

- The eye is essentially an outgrowth from the brain (neural ectoderm).
- Started as ***optic vesicle*** connected to the forebrain by ***optic stalk***.
Invagination of both the optic vesicle to form optic cup and the optic stalk to form choroidal (or optic) fissure inferiorly.
Ectoderm → Neural tube → optic grooves → optic vesicle → optic cup & optic stalk
Coloboma: congenital malformation in the eye, that results from a defect in fusion in inferionasal part. any defect in any other place (superionaa, lateral temporal…) is NOT a congenital problem.





• Development of the eye <u>after birth</u>

- At birth, the eye is relatively large in relation to the rest of the body.
- The iris has a bluish color due to little or no pigment on the anterior surface. Usually babies are born with light colored eyes, with age pigmentation darkens the eyes or it stays light like people with european descent.
- During early infant life, the cornea & sclera can be stretched by raised IOP → enlargement of the eye.

Difference between **infantile and childhood glaucoma** is noted by the <u>size of the eye</u> (very important to know)

	Congenital glaucoma	
	Infantile glaucoma	**Childhood glaucoma**
Patho-physiology	Non-working drainage system (non-functional trabecular meshwork) → high intraocular pressure (IOP)	
Age	Since birth - before 2 years	After 2 years
Elasticity	Present. Sclera and cornea can be stretched	Absent
Size of eye	Enlarged **(buphthalmos)**	Same size. <u>NO</u> enlargement

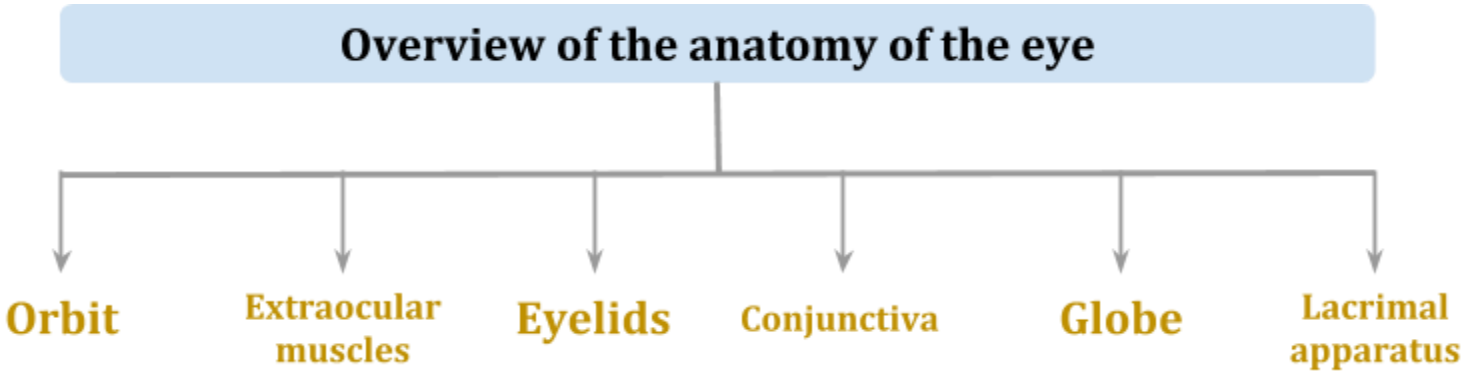
- The eye reaches full size by the age of 8.

Usually by age 2 the eye reaches its full size (adult globe) however, it is accepted up to age 8.

- The lens continues to enlarge throughout the life. The only part of the eye that continues to enlarge which explains **phacomorphic glaucoma**[1] or the acute glaucoma that occurs after 40 years.

By age 40: (1) Lens capsule lose its elasticity (2) Refractive index increases (presbyopia)

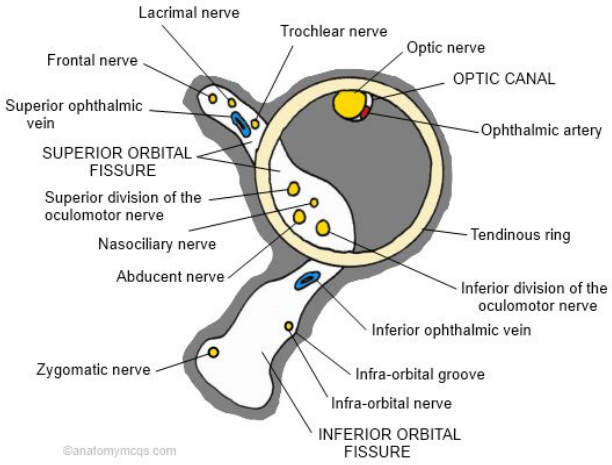
Anatomy & Physiology



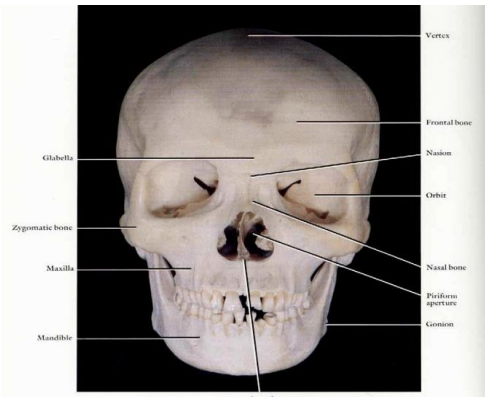
[1] term used for secondary angle-closure glaucoma due to lens intumescence. The increase in lens thickness from an advanced cataract, a rapidly intumescent lens, or a traumatic cataract can lead to **pupillary block** and angle closure.

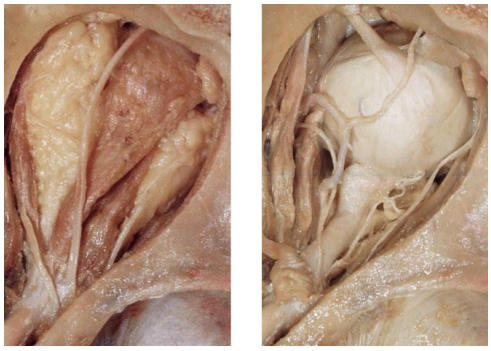
The Orbit

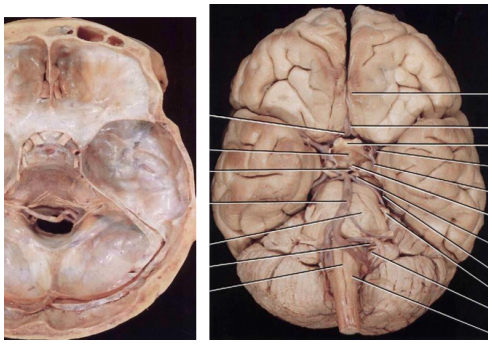
- A socket, contains & protect the eye.
- **Seven bones contribute the bony orbit:** frontal, zygomatic, maxillary, palatine, sphenoid, ethmoid, lacrimal.
- The weakest parts are the floor & the medial wall.
- Surrounded by nasal sinuses. that is why if you have sinusitis you might have orbital cellulitis as well.
- **Important openings are:** you have to know the structures running through all of them
    - Optic foramen (canal): ex: optic nerve & vessels.
    - Superior orbital fissure: ex: nasociliary nerve & inferior division or third cranial nerve.
    - Inferior orbital fissure:

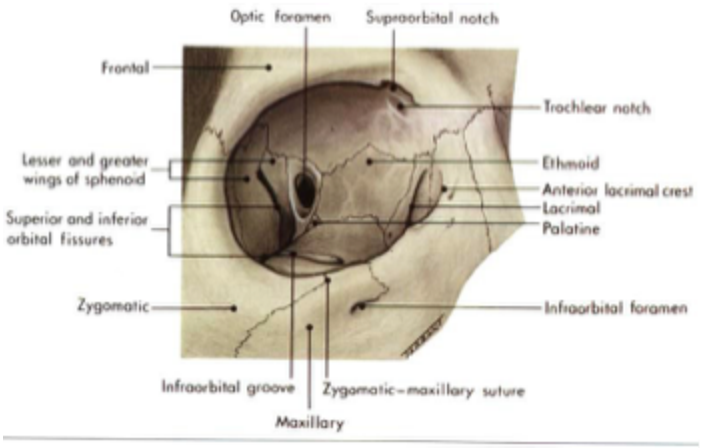


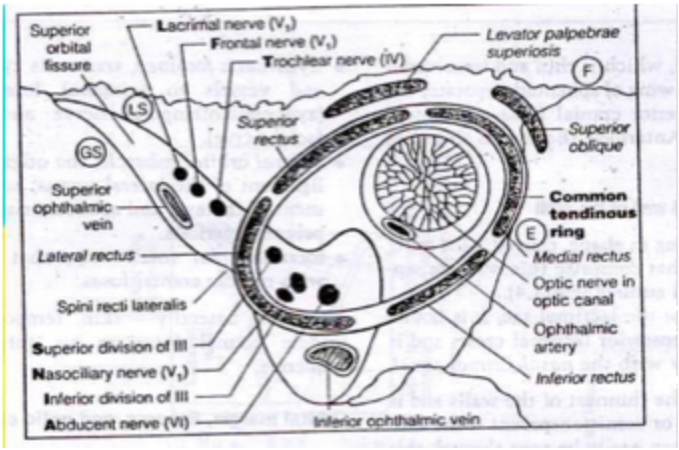
Optic foramen	Superior orbital fissure	Inferior orbital fissure
1. Optic nerve. 2. Ophthalmic artery. 3. Central retinal vein.	ANY STRUCTURE NOT MENTIONED IN OTHER OPENINGS! III, IV, and VI cranial nerves, lacrimal nerve, frontal nerve, nasociliary nerve, orbital branch of middle meningeal artery, recurrent branch of lacrimal artery, superior orbital vein and superior ophthalmic vein.	1. Infraorbital nerve (part of maxillary nerve) 2. Inferior ophthalmic vein. 3. Infraorbital vein & artery, 4. Zygomatic nerve. 5. Parasympathetic fibers to lacrimal gland











Extra from 435 team

- The eye lies within the bony orbit, which has the shape of a **four - sided pyramid**:
  - At **posterior apex:** optic canal; transmits optic nerve to the chiasm, tract & lateral geniculate body.
  - **Superior & inferior orbital fissures:** allow passage of blood vessels and cranial nerves which supply orbital structures.
  - The **lacrimal gland**: lies anteriorly in the superolateral aspect of the orbit.
  - On the **anterior medial** wall: lies the fossa for the lacrimal sac.

- Orbital margin is formed by the frontal, maxilla, and zygomatic bones.

- The **orbital cavity** is pyramidal, with its base in front and its apex (cone) behind.
  - **Roof:** formed by the orbital plate of the frontal bone, which separates the orbital cavity from the anterior cranial fossa and the frontal lobe of the cerebral hemisphere.
  - **Floor:** formed by the orbital plate of the maxilla, which separates the orbital cavity from the maxillary sinus. floor is the most easily part to get fractured (cause: blowout fracture) **"This is a common Q" Fracture of the floor is called orbital blowout fracture** and is due to blunt trauma which causes increased IOP and fracture of the orbital floor. This fracture causes impaction of inferior rectus muscle so the eye will be displaced inferiorly "defective elevation" and posteriorly "enophthalmos" and the inferior orbital nerve will be paralyzed. The patient will complain of diplopia **"vertical diplopia".** when u ask pt to look up and one of the eye did not move upward + hx of trauma think of blowout fracture.
  - **Medial wall:** formed from before backward by the frontal process of the maxilla, the lacrimal bone, the orbital plate of ethmoid (which separates the orbital cavity from the ethmoid sinuses), and the body of sphenoid. The weakest parts are the floor (because it has many openings) & the medial wall. that is why when someone has sinusitis he will have orbital cellulitis.
  - **Lateral wall:** formed by the zygomatic bone and the greater wing of sphenoid.

- **Other openings in the orbit:**
  - **Supraorbital notch:** Situated in the superior orbital margin, transmits the supraorbital nerve and blood vessels.
  - **Infraorbital groove and canal:** Situated in the orbital plate of the maxilla, the transmit the infraorbital nerve (a continuation of the maxillary nerve) and blood vessels.
  - **Nasolacrimal canal:** Located anteriorly in the medial wall; it communicates with the inferior meatus of the nose, and transmits the nasolacrimal duct.

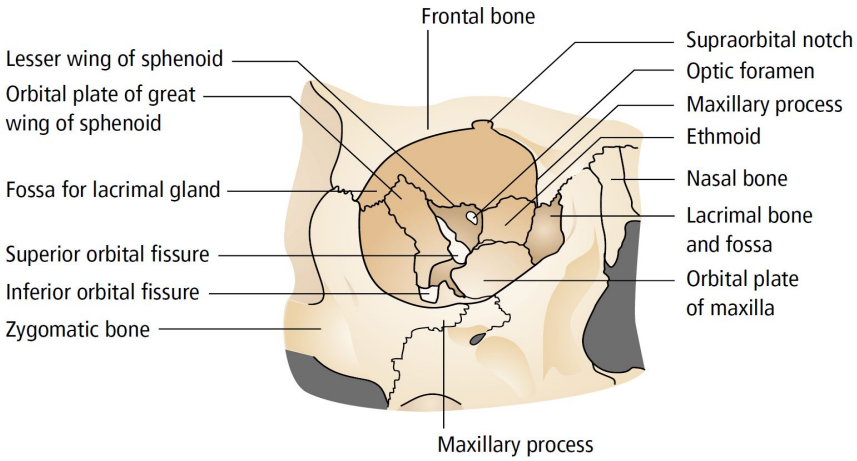
- Nasal bone fracture is the most common facial fracture.

- Zygomatic bone is the strongest, because it's the most likely to get trauma.

- Ethmoid bone is very thin, so it is known as Lamina Papyracea **"thinnest part of the orbit".** Patient with a **sinusitis →** invasion of the Infection from the ethmoidal sinus to the orbit results in **orbital cellulitis,** which is a serious complication.

- Both upper and lower divisions of oculomotor nerve (CN III) pass through Superior orbital fissure.

- MCQs: Infraorbital nerve: supplies the skin of the lower area of the lid. so when get senario says: **patient with paresthesia of the lower lid, what nerve is affected?** infraorbital nerve.



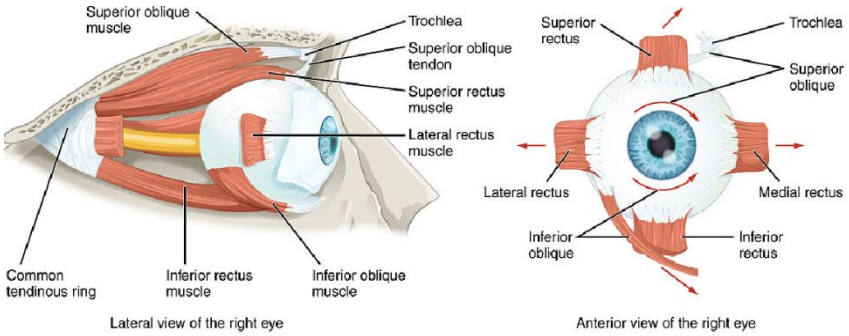
Extraocular muscles

- There are **6 muscles** responsible for the **movement of the eye**:
  - **Four recti:** superior, inferior, medial (the largest), and lateral.
  - **Two oblique muscles:** superior and inferior.

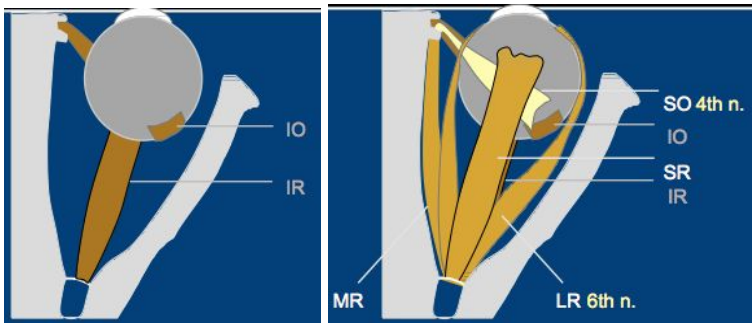
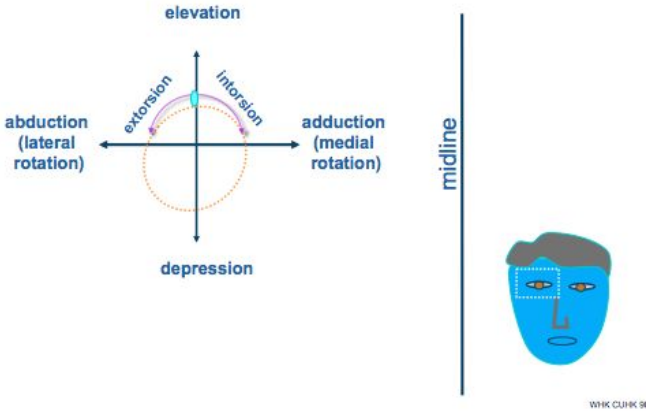
You need to know the origin & insertion of each muscle				
Muscle	**Origin**	**Insertion**	**Nerve supply**	**Action**
Superior rectus	Superior part of common tendinous ring of zinn	Superior & anterior aspect of the sclera	Oculomotor (CN III)	**Primary: elevation** Secondary: adduction Tertiary: incycloduction (**intorsion**)
Inferior rectus	Inferior part of common tendinous ring of zinn	Inferior & anterior aspect of the sclera	Oculomotor (CN III)	**Primary: depression** Secondary: adduction Tertiary: excycloduction (**extortion**)
Medial rectus	Medial part of common tendinous ring of zinn	Anterio-medial aspect of the sclera	Oculomotor (CN III)	**Primary: medial rotation or adduction**
Lateral rectus	Lateral part of common tendinous ring of zinn	Anterio-lateral aspect of the sclera	Abducens (CN VI)	**Primary: lateral rotation or abduction**
Superior oblique	Body of the sphenoid bone	Sclera - posterior to the superior rectus	Trochlear (CN IV)	**Primary: depression** Secondary: abduction Tertiary: incycloduction (**intorsion**)
Inferior oblique	Anterior aspect of the orbital floor.	Sclera - posterior to the lateral rectus	Oculomotor (CN III)	**Primary: elevation** Secondary: abduction Tertiary: excycloduction (**extortion**)

- Intorsion & extortion are micromovements of the eye (not visible)
- **All are supplied by Oculomotor nerve (III), except:**
  - Superior oblique → Trochlear nerve (CN IV)
  - Lateral rectus → Abducens nerve (CN VI)
- **Hence for clinical testing:**

Muscle	**Superior oblique**	**Inferior oblique**	**Superior rectus**	**Inferior rectus**
Direction to look	Down & in	Up & in	Up & out	Down & out
Movement of obliques are the opposite. **[O: Oblique = I: In], [R: Rectus = O: Out]**				



Note that there is a **difference** between **normal function & clinical testing! Helpful [link]**
Normal function: (1) SO: down & out (2) IO: up & out (3) SR: up & in (4) IR: down & in.
Clinical testing: (1) SO: down & in (2) IO: up & in (3) SR: up & out (4) IR: down & out.
SR & IO: both elevate the eye **IR & SO:** both depress the eye | we need to eliminate one to test the other
Why? for example SR vs IO: up & out. Orienting the visual gaze axis perpendicular to the inferior oblique muscle fiber direction to trap the IO so SR is the only muscle that is mediating elevation. Likewise with the other muscles. [**testing pure muscle action**]

Attachments of eye muscles	Action of eye muscles
	

Extra from 435 team

- Notes:

1. Superior oblique is the most superior muscle in the orbit.
2. Medial recti are strong muscles that keeps the eye in its normal position. During sleep, the eye tends to move laterally due to the relaxation of the medial recti. It's important that during preoperative assessment of a patient with strabismus. Check the angle of deviation before injecting anesthetic drugs; the eyes changes angles during sleep.
3. **Abducens nerve** is the only nerve passing through the **cavernous sinus** (around internal carotid), other nerves pass in the wall of cavernous (not through it). A trauma at this structure will cause: **medial squint.**

- Attachment of the Recti Muscles to the Eye:

- The **recti** are attached **in front** of the equator[2], unlike the obliques which are attached behind the equator.
- All muscles are originated from behind the eye, except inferior oblique (anatomically and physiologically), and superior oblique(physiologically only)[3]

- Patient with a 3rd nerve palsy, how can you rule out 4th nerve palsy? Important OSCE question
The clinical manifestations of 3rd nerve palsy: Ptosis + all muscles are paralyzed except the LR, so the eye will be abducted and little down + The patient can not look downward because the IR is paralyzed.
- To know if the 4th nerve is intact or not, ask the patient to look downward and if the 4th nerve is intact, then the eye will be in intorsion due to the action of SO.

[2] equator: the largest diameter of the eyeball.
[3] SO: is originated from behind the eye (anatomically), BUT the function of the muscle is like its originated from anterior because of the rounded tendon.

The Eyelids

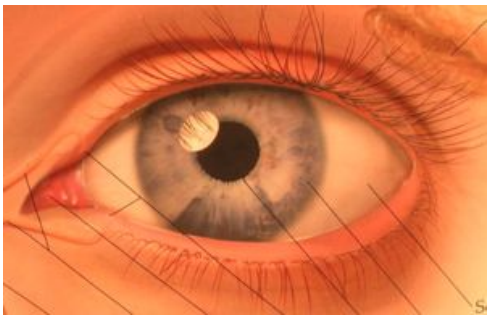
- There are upper & lower eyelids.
- They provide a protective covering for the eye. fish don't have eyelids because the water acts as a covering.
- The lids are:
  - **Closed by:** *orbicularis oculi,* supplied by facial nerve (CN VII)
  - **Opened by:** **IMPORTANT**: difference between ptosis due to parasympathetic & sympathetic injury!
    - *levator palpebrae superioris,* supplied by oculomotor nerve (CN III) **parasympathetic supply.** Ptosis occur with third nerve palsy due to levator palpebrae muscles paralysis.

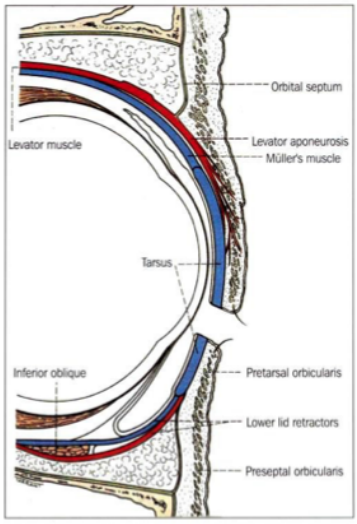
Patient came to the ER with ptosis (third nerve palsy) and the eye is out because of the lateral rectus, how can you test if the third nerve palsy is with/without fourth nerve palsy? ask the patient to look down. If fourth nerve is intact → intorsion. If not → unable to do intorsion **[that is at resident's level you'll not be asked about it]**

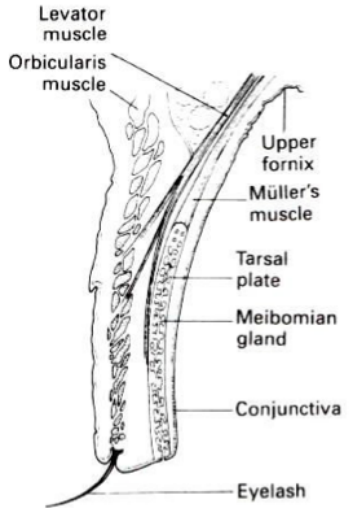
- *Muller's muscle,* **sympathetic innervation** injury → paralysis of Muller's muscle → ptosis like with horner syndrome. Mnemonic: **MAPLE** + sometimes heterochromia

(**M:** miosis (small pupil), **A:** anhidrosis (dry skin), **P:** ptosis, **L:** loss of ciliospinal reflex[4], **E:** enophthalmos)

- *Lower lid retractors*







Extra from 435 team

- Tarsus is the skeleton of the eyelid.
- Contraction of the peripheral fibres of the orbicularis muscle results in a protective, forced eye closure, while that of the inner, palpebral muscle results in the blink.
- It spreads the tears, and keeps ocular surface wet all the time.
- Orbital septum is a strong fibrous tissue, that serves to separate som eye structures from other structures, and it's an important barrier against infection.[5]
- It has a special meibomian glands (modified sebaceous glands) which secretes oily material that retards tear and make its stay longer in front of the cornea. divided into:
  - Anterior lamella: Skin and orbicularis (fibrosis of this leads to ectropion).
  - Posterior lamella: Tarsus and levator and conjunctival (fibrosis leads to entropion).
- Eyelashes are important as a protective to the eye (because the hair which is directed outward give the orbicularis more time to close). the area where eyelashes get out is called lead margin.
- Inflammation of lead margin is called Blepharitis.
- With entropion, upper eyelashes will be directed inward, and may damage cornea with time, with ectropion, lower eyelashes will be directed away from the eye, and this will lead to tearing and eye will become dry.

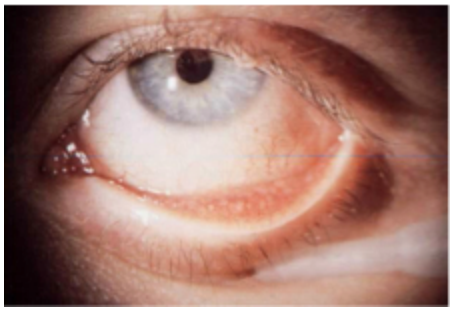
[4] The ciliospinal reflex (pupillary-skin reflex) consists of dilation of the ipsilateral pupil in response to pain applied to the neck, face, and upper trunk.
[5] infection anterior to orbital septum (preseptal cellulitis), behind septum (orbital cellulitis, very dangerous).

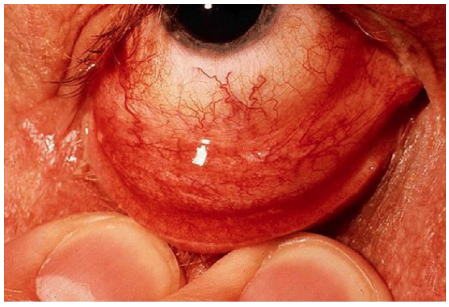
Conjunctiva

- Three parts:

1. **Bulbar conjunctiva** part that is covering the sclera (sclera is the white visible part)
2. **Palpebral conjunctiva** behind the eyelid
3. **Forniceal conjunctiva** at the fonix inside the eye "في الآخر"

- Limbus. | - The stroma: **adenoid** layer and **fibrous** layer (no adenoid tissues until 3 months after birth)[6].
- Follicles & Papillae. | - Injection & chemosis.

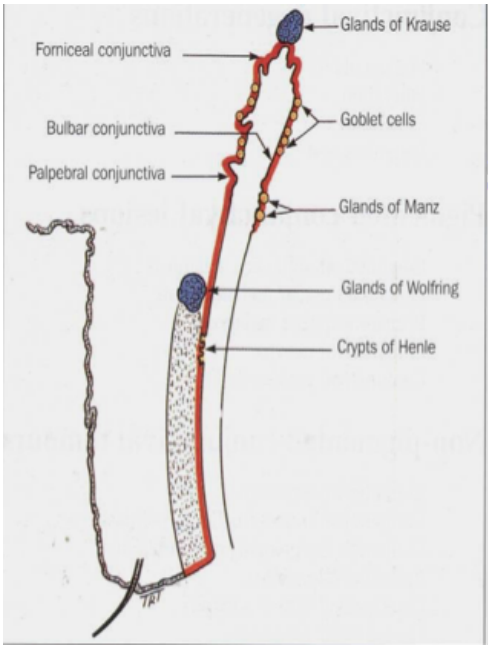




You can see:
- the three parts of the conjunctiva: Bulbar, palpebral & forniceal.
- **Accessory lacrimal glands:** Goblet cells, Glands of of krause, Glands of Manz, Glands of Wolfring

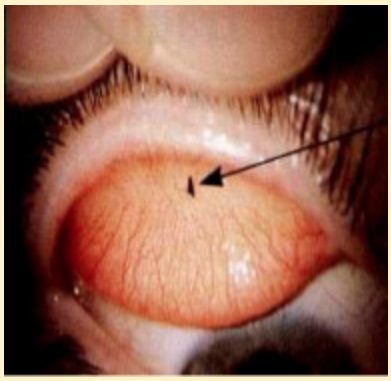
They are called the:
Sensory lacrimal glands → normal secretions (tears)
Lacrimal glands → reflex secretions like crying or happiness tears.
Tears ≠ lacrimal glands

Trachoma is an infectious disease. It used to be endemic in Saudi but now is eradicated. You can see the scarring of old trachomas in some patients.
No tear film → severe dry eye



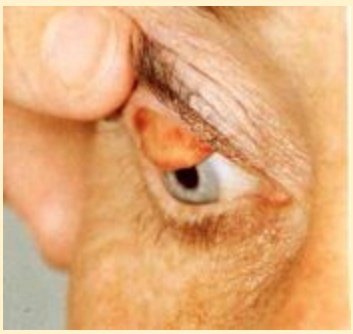
SAQ:

Q1: Identify the picture?
Foreign body in palpebral conjunctiva.
Q2: treatment?
Foreign body removal & topical antibiotics



SAQ:

Q1: Identify the picture?
Scars in palpebral conjunctiva due to old trachoma.
Q2: treatment?
Lubrication



Most previous batches didn't know how to answer this question

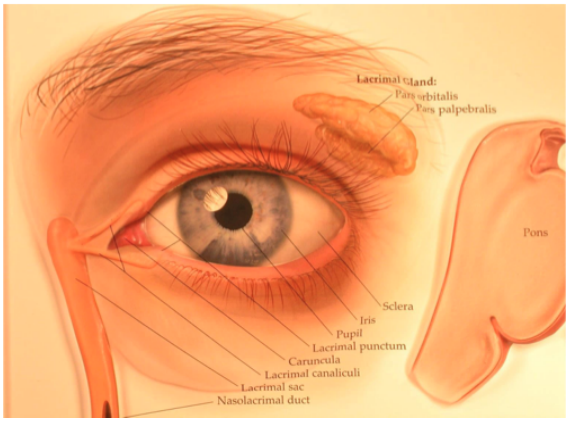
Extra from 435 team

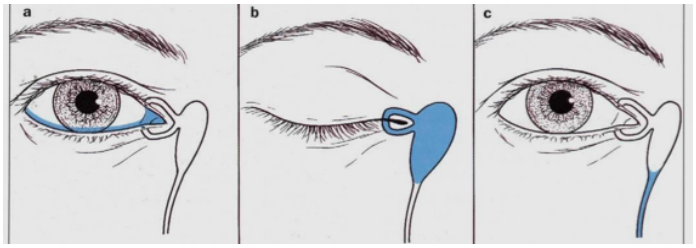
- It is the transparent membrane covering the sclera, it's the outer cover of the eye, normally it has vessels and sometimes they are prominent, but if there are no vessels or redness, then the patient might be anemic.
- In a case of vernal keratoconjunctivitis, the bulbar conjunctiva may have tranta's spots. "discrete whitish raised dots along the limbus"
- Forniceal conjunctiva: it has a structure called Cul-de-sac, which serves as a reservoir for tears and drugs.
- Injection : is peripheral hyperemia of the anterior ciliary vessels which produces a deep red or rose color of the corneal stroma. Causes of ciliary injection: keratitis, Uveitis, Acute glaucoma.
- Chemosis : It is a swelling of the conjunctiva due to abnormally permeable capillaries

[6] inflammation of adenoid is called (Follicular Conjunctivitis), and inflammation of fibrous layer is called (Papillary Conjunctivitis)

The Lacrimal Apparatus

- Lacrimal gland has two parts: bulbar & palpebral. It connects with the eye by a duct. Works by reflex tearing.
- Tear secretion & drainage. if there is secretion with no drainage = persistent tearing
- Layers of precorneal tear film.
- Fluorescein test, done to adults & pediatrics: we put a stain (fluorescein) in the eye → goes away after 15 min If it does not go down → indicative of nasolacrimal duct obstruction.

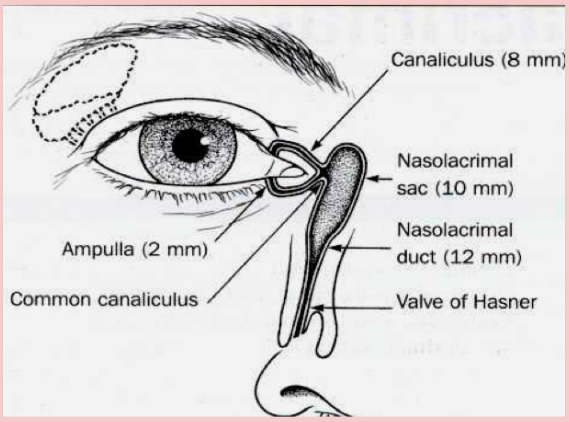




Fluorescein test

You need to know the length "هذه الصورة تحفظونها صم"

Ampulla (2 mm), canaliculus (8 mm), common sac (10 mm), nasolacrimal duct (12 mm): bony part & soft part, valve of hasner → inferior meatus of the nose.

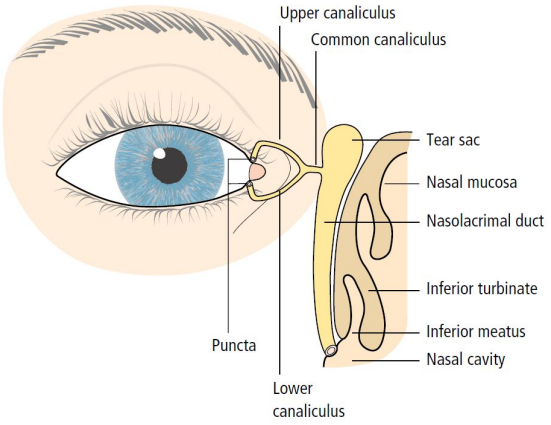


Babies born with teary eyes have a problem with the nasolacrimal duct (blocked)

* Closed by bony part → nothing to do
* Closed by membranous part → observe for 1 year → goes spontaneously → persists → syringe & probing "تسريب"

Extra from 435 team

- Duct opens in superior conjunctival fornices.
- Tears drain into the upper and lower puncta and then into the lacrimal sac via the upper and lower canaliculi (Figure). They form a common canaliculus before entering the lacrimal sac. The nasolacrimal duct passes from the sac to the nose.
- Lacrimal gland secretes tears into the upper fornix of the conjunctival sac which are spread over the surface of the cornea as a tear film by blinking of the lids.
- Tears accumulate at the inner canthus and drain into the lacrimal sac via the puncta and canaliculi. (tears do not stay)
- the sac is continuous inferiorly with nasolacrimal duct which opens into the nasal cavity just beneath the inferior turbinate.
- The cascade : punctum (the start of the drainage system) > canaliculi> nasolacrimal sac > nasolacrimal duct > Inferior meatus (valve of Hasner is at the meatus).



- For the lacrimal gland, normally it's not palpable.
- The canaliculi connect the punctum to the nasolacrimal sac, not the duct.
- Blinking help tear drainage into the system by creating negative pressure.
- Infection of the lacrimal gland is called (Adenitis), and of lacrimal sac (Dacryocystitis).
- Running eye and nose means intact nasolacrimal duct.
- Tear film. 3 layers consists of: Mucin gel layer, Aqueous layer (produced by lacrimal gland), Oil layer: Meibomian glands.

Eye (globe)

- Two spheres with different radii:
- Cornea (transparent), window of the eye.
- Sclera (white), opaque shell.

- Cornea in stronger than sclera.
- Sclera is opened in most ruptured globe (open globe) cases in the ER due to blunt trauma. Why? Special arrangement of vertical & horizontal collagen → transparency of the cornea. Corneal injury → becomes white due to scarring (like sclera). Scleral fibers are interwoven → white & opaque.

- The eye measures approximately 24 mm in all its main diameters.

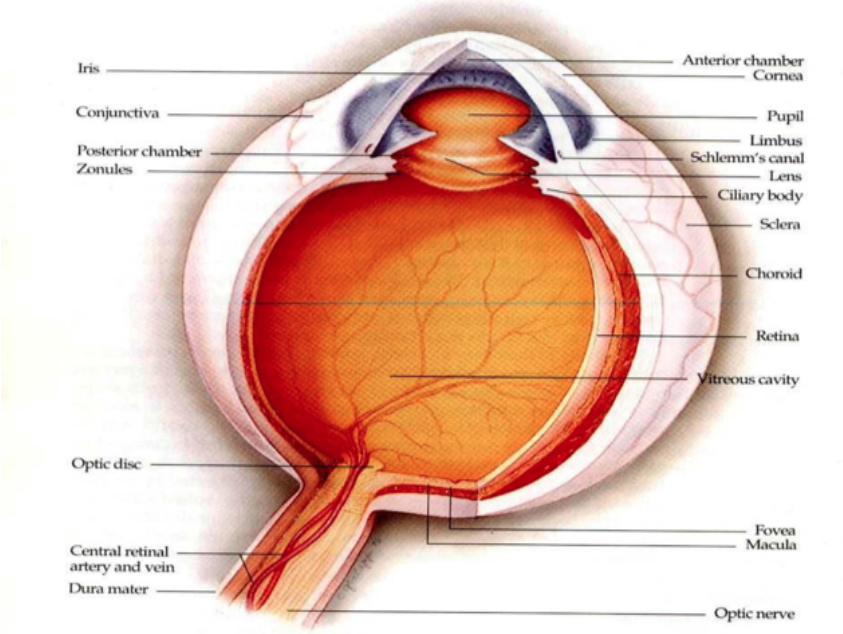
- Three layers:
- **The outer** (outer protective layer): inelastic coat, transparent **cornea** and opaque **sclera**.
- **The middle**: (vascular nourishment layer) coat, **The Uvea:** choroid, ciliary body and iris.
- **The inner** (neurosensory)**:** The **retina**, extends forwards to within 6 mm of the limbus & **optic nerve**

- If there is no vascular tissue the eye will atrophy because all secretions are coming from the ciliary body.

SAQ:

You might get a picture pointing to a structure.
So, it is very important to know all the structures.
Examples:

a. Lens.
b. Vitreous cavity.
c. Fovea.
d. Optic nerve.
e. Iris.



Extra from 435 team

- Eye consist of globe (eyeball, extraocular muscles) and adnexa (lacrimal gland and sac).
- **Coats of the Eye:**

1. Fibrous coat: Made up of a posterior opaque part, the sclera, and an anterior transparent part, the cornea. Both are formed of collagenous fibers with different arrangement. Lamina cribrosa is the area of the sclera that is pierced by the nerve fibers of the optic nerve. Cornea is in contact posteriorly with the aqueous humor.
functions are: protection and vision

2. Vascular pigmented coat : Consists from behind forward, of the choroid, the ciliary body " mainly", and the iris. The choroid is composed of an outer pigmented layer and an inner, highly vascular layer. The ciliary body is continuous posteriorly with the choroid, and anteriorly, it lies behind the peripheral margin of the iris. It is composed of the ciliary ring, the ciliary processes, and the ciliary muscles. "The ciliary body has pigmented and non-pigmented epithelium, the non-pigmented one secrets the aqueous"
if ciliary body affected (uveitis) will cause atrophy of the eye.

3. Inner layer: composed of the optic nerve and the retina. The retina extends forward to within 6 mm of the limbus.

Eye Chambers

- Three optically clear spaces:

- **Anterior chamber:** in front of the iris (between the cornea and the iris)
- **Posterior chamber:** immediately behind the iris. (between the iris and zonule fibers)

These two chambers which communicate through the pupil are filled with clear aqueous humour السائل المائي

- Aqueous humour: (1) secretion: non-pigmented ciliary epithelium. **(2)** pathway: posterior chamber → pupil → anterior chamber **(3)** drainage: at the angle → canal of schlemm → trabecular meshwork.

- Glaucoma: closed-angle glaucoma (if closed), open-angle glaucoma (open but non-functional)

- **Vitreous cavity:** filled by gel-like structure, The Vitreous. السائل الزجاجي

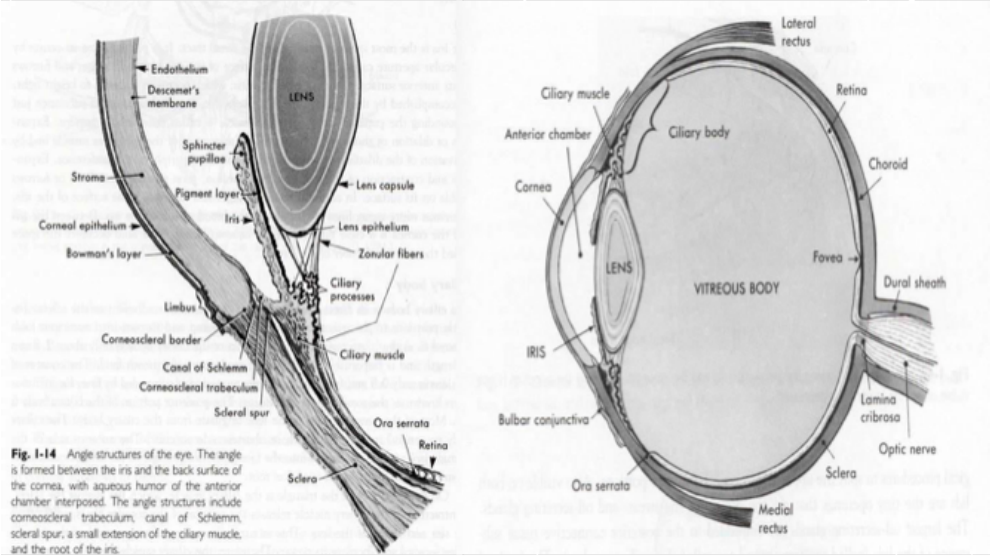


Fig. I-14 Angle structures of the eye. The angle is formed between the iris and the back surface of the cornea, with aqueous humor of the anterior chamber interposed. The angle structures include corneoscleral trabeculum, canal of Schlemm, scleral spur, a small extension of the ciliary muscle, and the root of the iris.

- The Intraocular pressure (IOP)

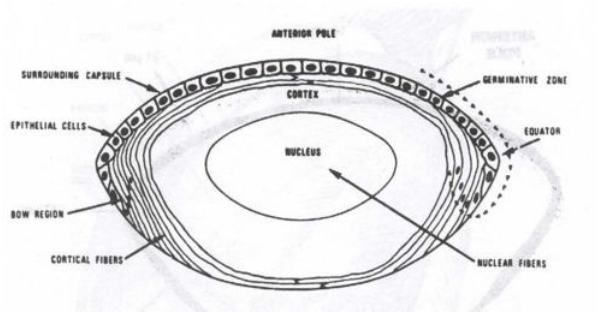
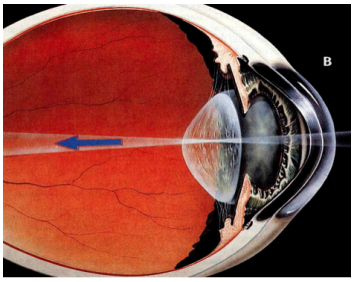
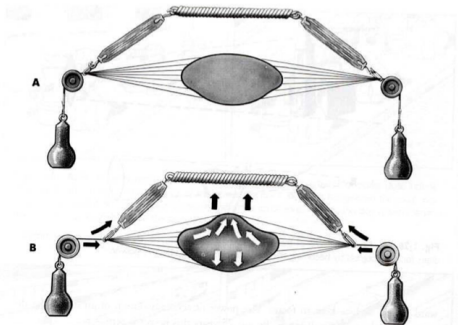
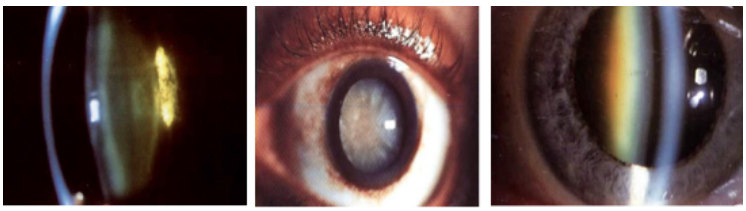
- The pressure within the eye is maintained at a steady level by continuous formation & drainage of aqueous.
- **The intraocular pressure, (IOP), is normally 10 – 21 mmHg; increased IOP called Glaucoma.**
  If IOP becomes high it might lead to glaucoma & optic nerve atrophy.
- High IOP almost always due to an obstruction of aqueous outflow.

Extra from 435 team

- It should be always clear, abnormal blood collection in it is known as hyphema and it is mainly due to trauma.
- Collection of pus in the anterior chamber is known as hypopyon.
- Glaucoma happens because of problem in absorption not because of increase secretion.
- **434 notes:**
  - the aqueous is secreted with the help of carbonic anhydrase enzyme. Drainage: Trabecular meshwork into the schlemm's canal (85%), and the other 15% got drained through the uveoscleral outflow (absorbed by the iris and the ciliary body to the suprachoroidal space).
  - If the patient has a predisposing factor of glaucoma (e.g. hyperopia) and the pupil remained mid-dilated for a long period, the iris will adhere to the lens, and this will cause pupillary block, which in turn leads to accumulation of the aqueous in the posterior chamber and hence raising of IOP leading to the development of acute glaucoma. The initial treatment of acute glaucoma is carbonic anhydrase enzyme inhibitors to reduce the volume of accumulated aqueous like: Acetazolamide "Diamox" and Sulfonamide. These drugs act mainly on the trabecular pathway. Prostaglandins found to be active on the other pathway and has cosmetic side effects, it elongates and thickens eyelashes. The initial intervention is long term treatment is clear lens extraction or laser iridotomy.
  - Anterior segment is anything in front of the lens including anterior chamber, cornea, iris, and lens itself. Posterior segment is anything behind the lens including vitreous, retina and optic nerve. When the patient complains of decreased vision but has normal anterior and posterior segments, then he might have amblyopia "lazy eye".

The Lens

- The crystalline lens is the only structure continuously growing throughout the life.
- **Structures:** capsule (anterior & posterior), epithelium and lens fibers, nucleus and cortex (in between)
- Children have embryonic nucleus (soft), adults have hard nucleus.
- Zonules or suspensory ligament (about 70,000) attaches the nucleus to the ciliary body on each sides. Which is important in accommodation (contract or relax depending if you're looking near or far away). After 40 years, the elasticity of the zonules diminishes (no contraction) → inability to see what is near.
- **Disease of the lens:**
  - Cataract.
  - Congenital anomalies and effect of systemic diseases.

Structures of the lens	Changeable refractive media
	 Light passes: cornea → anterior chamber → lens → retina
Zonules contraction & relaxation	**Cataract**
 **A:** contraction of zonules → far vision **B:** relaxation of zonules → bulging of lens → near vision	 **There are 3 types of cataract:** **A: Nuclear** cataract **B: Cortical** cataract **C: Posterior subcapsular** cataract. Occurs with steroid use or trauma (in both pediatric & adult patients)

Extra from 435 team

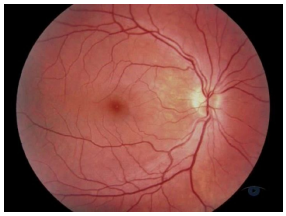
- It is a transparent, biconvex structure enclosed in a transparent capsule. It is situated behind the iris and in front of the vitreous body and is encircled by the ciliary processes. **(It's a changeable refractive media).**
- The lens consists of an **elastic capsule**, which envelops the structure; a **cuboidal epithelium**, which is confined to the anterior surface of the lens; and **lens fibers**, which are formed from the cuboidal epithelium at the equator of the lens.
- It is mainly ectodermal in origin, so it is very sensitive to pain.
- Attack of high pressure cause destruction of the epithelium under anterior capsule cause opacity.

- 434 Notes :

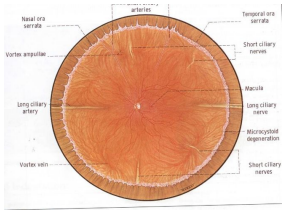
- **Important question:** Post kidney transplant patient **on steroids** came complaining of decreased vision, what is the possible cause? The answer depends on the IOP, if it is normal, then he might have posterior subcapsular cataract. If his IOP is elevated, then he might have glaucoma.
- Two approaches of cataract surgery: **phacoemulsification** and standard extracapsular cataract extraction (ECCE).

Retina & Vitreous

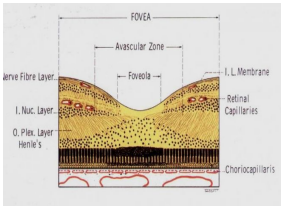
- Optic nerve head, macula, fovea, retinal background, ora serrata & retinal vasculature.
- Remember: there are two parts of the retina without blood vessels: macula (fovea) & ora serrata.
That is why most ophthalmic surgical procedures are done there **(pars plana)** ex. vitrectomy.
- Retina has 10 layers: **(1)** the inner limiting membrane **(2)** the nerve fiber layer **(3)** the ganglion cell layer **(4)** the inner plexiform layer **(5)** the inner nuclear layer **(6)** the outer plexiform layer **(7)** the outer nuclear layer **(8)** the outer limiting membrane **(9)** the photoreceptor layer and **(10)** the retinal pigmented epithelium.
- Vitreous attachment. | - Retinal detachment. | - Effect of systemic diseases.



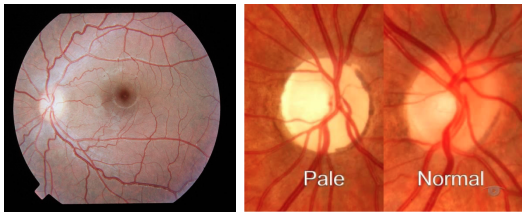
Normal retina:
* Optic nerve
* Macula (fovea)



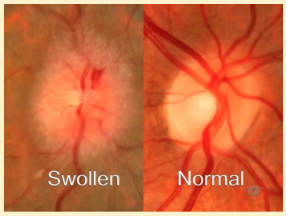
* Retinal artery & vein.
* Optic disc.



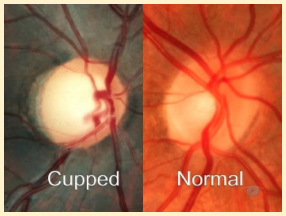
Macula → fovea → avascular zone → Foveola (sharp vision 20/20 with color)



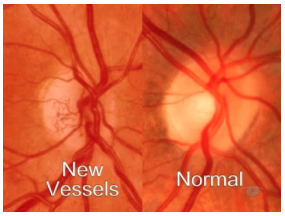
Normal optic disc has small cup (0.3 to 0.4 cup-to-disc C/D ratio).
Optic cup: pale area in central fovea **pic**



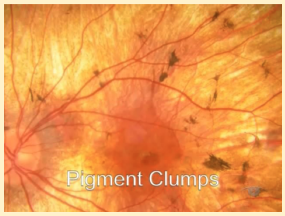
SAQ: Disc edema. If bilateral = papilledema (can be due to intracranial or brain tumor)



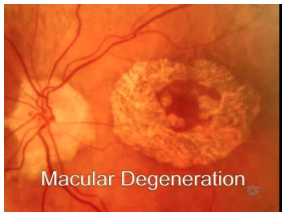
SAQ: If C/D ratio is more than the range = large or physiologic or pathological (glaucoma) cup. How to diagnose glaucoma? Measure IOP (ocular tonometry) & visual field.



New vessels (mostly optociliary shunt vessels) due to sella turcica meningioma **OR** central vein occlusion **OR** diabetic retinopathy.



SAQ: very important
Retinitis pigmentosa: Pigmented clumps + pale optic disc



Age-related macular degeneration

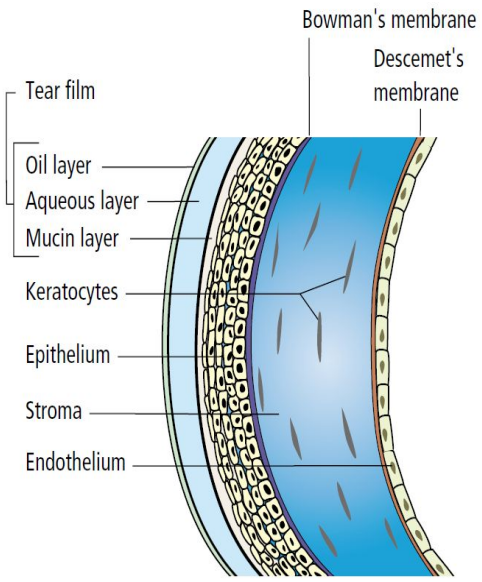
Extra from 435 team

- The retina consists of an outer pigmented layer and an inner nervous layer. Its **outer** surface is in contact with the choroid and its inner surface is in contact with the vitreous body.
- At the center of the posterior part of the retina is an oval, yellowish area, the **macula lutea**, which is the area of the retina for the most distinct vision. It has a central depression, the **fovea centralis.**
- The optic nerve leaves the retina about 3 mm to the **medial** side of the macula lutea by the optic disc. **The optic disc** is slightly depressed at its center, where it is pierced by the **central retinal artery.** At the optic disc, is a complete absence of rods and cons, so that the optic disk is insensitive to light and is referred to as the **"blind spot".**
- Photoreceptors contains visual pigment [large protein (opsin) attached to retinal (vitamin A aldehyde)]
- Light splits the opsin from the retinal with initiation of a graded electrical potential →Transmitted through the visual pathway to be processed in the visual cortex (occipital lobe) → vision sense.
- Macula: an area of the eye near the center of the retina where visual perception **is most acute**.
- Fovea: The fovea is the region in the center back of the eye that is responsible for acute (central) vision. **The fovea has a high density of cones. (while periphery of retina has higher density of rods).**
- Rods is used for night vision and cones for day vision
- Vitreous humor: It is a gel-like substance occupied the space behind the lens and in front of the retina at the back of the eye, it comprises a large portion of the eyeball. It consists of water, collagen, salt and sugar. It maintains the shape of the eye, and it is a clear solution to the light can easily pass through it.

Cornea

It has 5 layers

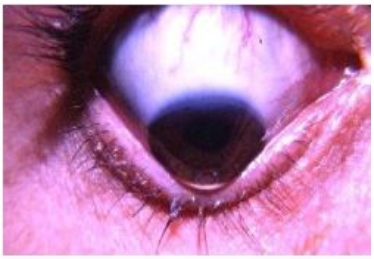
Layer (anterior to posterior)		condition
Epithelium	➢ continuation of the bulbar conjunctiva ➢ ectodermal origin	Abrasion. Treatment by covering the eye for 24hrs and defect will heal without scarring. Never use topical anesthesia in this case.
Bowman's membrane	clear **acellular** layer	Abrasion. Risk of scarring
Stroma	contains collagen fibrils that accounts for around 90% thickness	
Descemet's membrane		Disturbance of this layer is common in congenital glaucoma. Management is surgical via keratoplasty
Endothelium	Single layer	It end in the angle, we have to know if it's open or closed which is about schwalbe's line , trabecular meshwork , scleral spur , ciliary body and iris if all structures are seen that mean **open angle glaucoma while if all structures closed that mean closed angle**.



You should know all 5 histological layers of the cornea.

Extra from 435 team

- The cornea is 500-530 micron(0.5mm) in thickness. It consists of **five layers: epithelium, bowman's layer** "called anterior limiting membrane"**, corneal stroma, descemet's layer** "called posterior limiting membrane"**, and corneal endothelium.**
- It protects the internal ocular structures. Together with the lens, it refracts and focuses light onto the retina.
- Nutrition of the cornea is supplied almost entirely by the aqueous humour.
- It is transparent because: it is **avascular** and it has **regularly** arranged collagen fibers.
- The epithelium is from surface ectodermal origin, thus it is very sensitive and also it can regenerate after being injured.
- The thickness of the cornea affects the IOP reading. After Lasik, the cornea becomes weak and a false low reading of IOP could occur, so the diagnosis of glaucoma might be missed. One possible risk of Lasik is keratoconus, which is central thinning and plugging of the cornea. This might end with corneal scarring and keratoplasty "corneal transplantation"
- The patient with keratoconus when he looks down, his lower eyelid becomes V shaped because of the plugging, and this is called **Munson's sign. "important"** **(**Keratoconus is a strong contraindication of Lasik.) So, When bowman's membrane gets injured, it will heal with a scar "opacity".

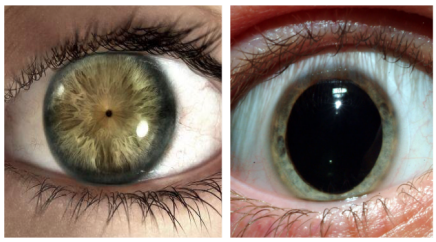


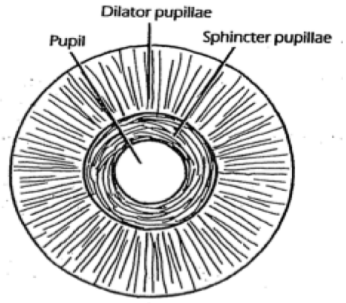
- **SAQ:**
  - Corneal abrasion usually does not cause scars unless the bowman's layer or the stroma are injured.
  - The descemet's membrane is a very strong membrane, but in case of congenital glaucoma, it will be stretched and the aqueous will invade the cornea, which will disturb corneal fibers arrangement and cause corneal edema. If congenital glaucoma doesn't get corrected early, the condition will worse and end up with corneal scarring and keratoplasty might be needed. **So the treatment of congenital glaucoma is surgical. Important Q:** When the patient got blunt trauma in the eye, which one is more susceptible to injury, cornea or sclera? **The answer is sclera**
  - Actually, both Cornea and sclera are made up of collagen fibers but they differ in the arrangement. The corneal fibers are arranged in vertical and horizontal planes, making it more clear and 100 times stronger than sclera. The fibers of the sclera are arranged in haphazard fashion, which make them weaker and more opaque.

Iris & Pupil

- The iris has two muscles:
  - **Dilator pupillae:** dilation of pupil (mydriasis) supplied by sympathetic fibers
  - **Sphincter pupillae:** constriction of pupil(miosis), supplied by parasympathetic fibers from the oculomotor nerve. Predominant.

Difference between two pupils (small & large) is called: **Anisocoria.**





Extra from 435 team

- The iris is a thin, contractile, pigmented diaphragm attached peripherally to the anterior part of the ciliary body.
- It forms the pupil at its centre, the aperture of which can be varied by the circular sphincter and radial dilator muscles to control the amount of light entering the eye.

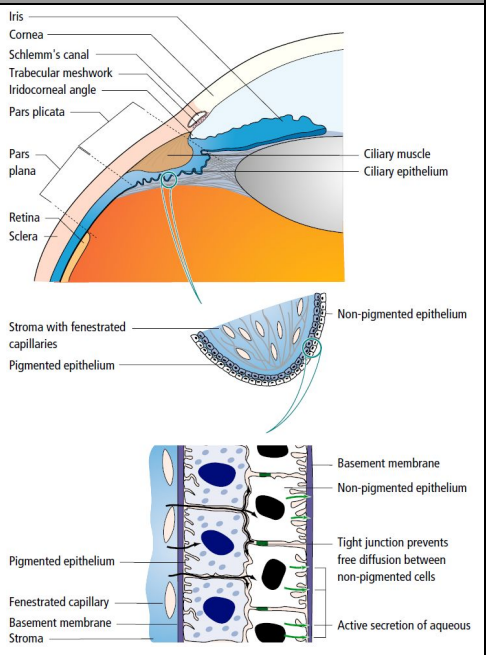
- 434 notes:

- In patients with 3rd nerve palsy and spared pupil like in diabetes, or in patients with semi dilated\fully dilated pupil and 3rd nerve palsy, what is the recommended investigation?
- The answer is MRA "magnetic resonance angiography" because the most common cause of surgical 3rd nerve palsy is aneurysm, which can be detected using MRA.

Ciliary Body

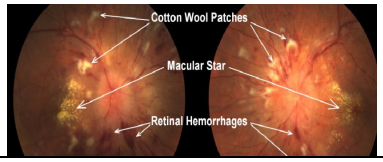
Extra from 435 team

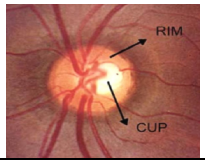
- The structure of the ciliary body is triangular in cross-section, and it encircles the inside of the eye behind the colored iris. It connects posteriorly with the choroid and anteriorly with the iris.
- The ciliary body is subdivided into three parts:
  - The ciliary muscle (responsible for changes in lens thickness and curvature during **accommodation**)
  - The ciliary processes at the front (pars plicata):They are responsible for the secretion of **aqueous humour**.
  - The pars plana at the rear. Pars plicata has the ciliary processes, and the zonular lens fibers from the crystalline lens pass deep into the valleys between them.



- 434 notes:

- In fundus examination, you should comment on: **the optic disc, the retinal vessels, and the macula.**
- Patient with chronic visual loss + the fundus is not seen = do B-scan ultrasound. "Gold standard"

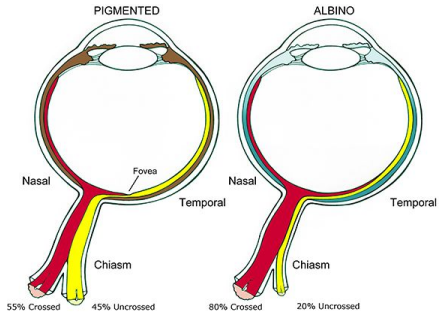




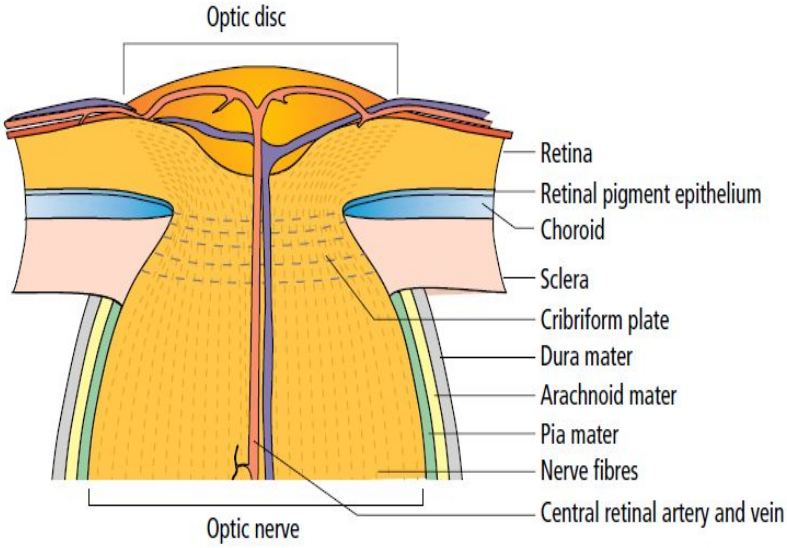
Optic Nerve

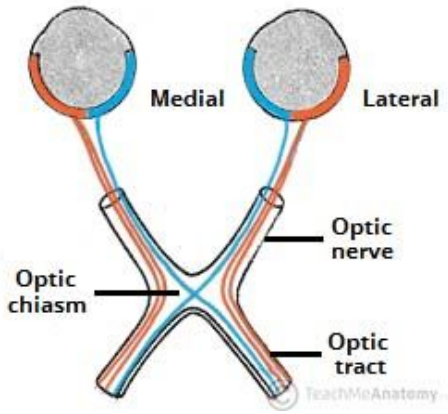
Extra from 435 team

- Optic nerve > optic disc > rim and cup(when increased is sign of glaucoma, or could be physiological and we differentiate two types by measuring IOP and visual field)
- contains around 1.2 million nerve fibers, which are axons of the retinal ganglion cells.
- the length of optic nerve in these structures: important to know
    - 1 mm in the globe.
    - 25 mm in the orbit.
    - 9 mm in the optic canal.
    - 16 mm in the cranial space



- **Partial decussation occurs and about 53%** of the fibers cross to form the optic tracts (if pt have carotid lesion will develop binasal hemianopia).
- **The optic nerve** leaves the retina about 3 mm to the medial side of the macula lutea by the optic disc. The optic disc is slightly depressed at its center, where it is pierced by the central retinal artery. At the optic disc, is a complete absence of rods and cons, so that the optic disk is insensitive to light and is referred to as the "blind spot".
- **Optic disc:** the terminal part of the optic nerve where ganglion cells fibers leaves the eye.
- **Optic cup:** a central depression within the optic disc.
- The normally cup-disk ratio is 3/10 (0.3) Measurement is crucial to diagnose certain diseases like glaucoma.





Optics of the Eye

Extra from 435 team

- The eye is like a camera. Light must have a clearly pathway to be clearly focused on the sensory receptors of the retina, i.e., Clear cornea, anterior chamber, lens and vitreous cavity.
- The Refractive power of the eye is about 58 -62 diopters.
- The cornea is the major refracting element of the eye with a power of approximately 40 diopters. If the curvature is greater in one meridian than the other→ Astigmatism.
- The refractive power of the lens is about 17-21 diopters at rest. Accommodation can change the power of the lens markedly depending on the age.

- 434 notes:

- Young kids have a strong accommodation up to 30 diopters as their lenses has 70.000 zonule fibers.
- When ciliary body contracts → relaxation of the zonules → plugging of the lens and thus increased depth of focus and you will be able to see near objects.
- When ciliary body relaxes → contraction of the zonules → thinning of the lens, so you will be able to see far objects clearly.
- **Important:** *With increased age, usually from 40s, people experience a problem with near vision "Presbyopia", why?

Because of: (1) Loss of elasticity of the lens (2) Increased refractive index of the lens due to nuclear sclerosis.

- Presbyopia is treated with convex lens.
- **Hyperopia "farsightedness"** → treated with convex lens.
- **Myopia "nearsightedness"** → treated with concave lens.
- Patient with big eyes ">25 mm in diameter" the image will be reflected in front of the retina leading to myopia, to correct it we give the patient a concave lens, thus causes a diversion of light rays.
- Patient with small eyes "<21 mm in diameter" the image will be reflected behind the retina leading to hyperopia, to correct it we give the patient a convex lens that causes a conversion of light rays.

Vision / Visual Pathway

- Function of the eye: receive the image and change it into language that the brain can understand.

- The retina:

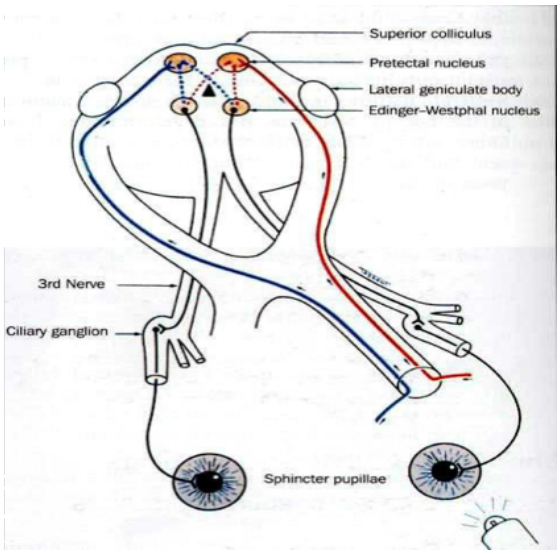
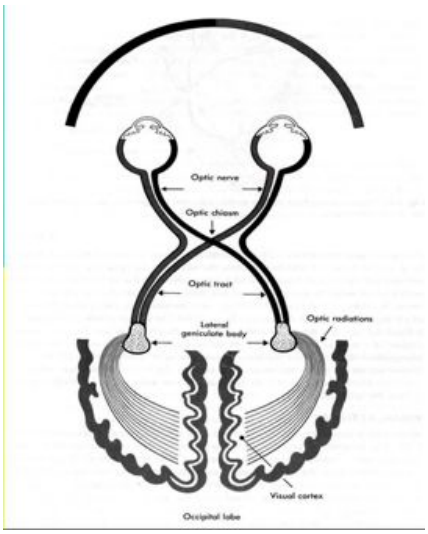
- It is divided into retinal pigment epithelium & neurosensory retina.
- Photoreceptors contains visual pigment (vitamin A aldehyde: 11-cis-retinal) that changes into Rhodopsin (chemical) upon light stimulation (electrical stimulation)

- Visual Pathway: THREE order neurons:

1. **Bipolar cell:** lies within the retina. from photoreceptors to retinal ganglion cell.
2. **Ganglion cell**: from retinal ganglion cell to synapse in lateral geniculate body
3. **Third neuron:** from lateral geniculate body and terminates in visual cortex.

Extra from 435 team

- The central retina contains yellow pigment, Xanthophyll, the so called macula lutea (yellow spot).
- Light splits the opsin from the retinal with initiation of a graded electrical potential →Transmitted through the visual pathway to be processed in the visual cortex (occipital lobe) → vision sense.

Pupillary light reflex by CN III	Visual pathway
	

Summary

Question	Answer
The socket that contains the eye is called	**ORBIT**
The orbit is formed by …..... Bones.	**Seven**
The optic nerve pass through ……..	**Optic foramen or canal**
The eye has …... Extraocular muscles, …... Recti and …... obliques.	**Six , 4 , 2**
All extraocular muscles are supplied by …… except …… & ……	**Oculomotor nerve, SO4, LR6.**
When we test a patient 4th nerve we should ask him to look …….	**Down and in.**
The space that lies behind the cornea & in front of the iris called ……..	**Anterior chamber**
The fluid that fills the chambers of the eye called ……..	**Aqueous humors.**
The cavity that lies behind the lens called …….	**Vitreous cavity**
The main refractive structure in the eye is ……..	**the cornea.**
The second refractive structure in the that can change its power ……..	**the lens**
The structure that determines the color of the eye is ……..	**the iris**
when the lens get opacified this condition called ……....	**cataract**
The central part of the retina is called ……...	**the macula.**
The central part of the macula is called ……...	**fovea**
The central part of the fovea is called	**foveola**
The optic nerve lies in ……. part of the retina.	**nasal**
The percentage of the decussating fibers is ……...	**53%**

Ocular Manifestation of Systemic Diseases

[**Color index:** Important | Notes: **F1**, **F2/A** | Extra] **EDITING FILE**

Objectives:

➢ Not given.

Done by: Bedoor Julaidan.
Edited by: Lamya Alsaghan, Rawan Aldhuwayhi, Munerah AlOmari.
Resources: Slides + Notes + 435 Team.

*** Don't let the number of pages deceive you. It is mostly pictures.**

*** Prof. Abu El-asrar emphasized on the importance of (Kanski's Clinical Ophthalmology) book "just search for a disease mentioned in the lecture & look at its pictures" LINK**

*** Dr. Esam (organizer) said a lot of MCQs & SAQ pictures will come from this lecture.**

*** Special thanks to Bedoor Julaidan for her amazing note taking skills!**

One last thing… grab a cup of coffee

Diabetes Mellitus

• Overview

– By far the most common disease that can affect the eyes and even can cause blindness and now considered to be irreversible cause of blindness is diabetes.
– The commonest cause of legal blindness in individuals between the ages of 20 and 65 years. This is relatively young people. Which means that blindness due to DR has a major impact on the country because the cost of taking care of blind people is very huge. So it has a major socioeconomic problem.
– The risk of blindness is about 25 times greater in diabetics than in non-diabetics.

<table>
<tr><th colspan="2">Risk factors</th></tr>
<tr><th>Modifiable</th><th>Non-modifiable</th></tr>
<tr><td>✻ Glycemic control: tight control of blood sugar especially if started early in the course of diabetes is very beneficial to prevent and stop progression of diabetic retinopathy, not only diabetic retinopathy but also other microvascular disease such as: nephropathy and neuropathy. This evidence came from many studies, one of the oldest is diabetes control and complications study that was multi-centered study, patients were followed for 6 and half years and was mainly focusing on type 1 diabetes. Young diabetics were randomized to receive conventional insulin treatment (1 or 2 injections per day) vs tight control of blood sugar in the form of insulin pump or several insulin injections per day. The group who had tight control they had mean HA1C 7.2%. Then at the end of follow up, it was clear and obvious that tight control of blood sugar protected against development and progression of diabetic retinopathy. Another big observation that after termination of study, all the patients resumed the previous medication, so those patients who were tightly controlled are no longer tightly controlled. Then it was found that even that the 2 groups have equal blood sugar levels, those who had tight control early in the course of diabetes were still protected. So early tight control after the onset of diabetes is very important. This phenomenon is known as “metabolic memory”.</td><td rowspan="3">✻ Duration: if we look to the risk factors related to the incidence of DR, it is the strongest and unfortunately cannot be avoided . It’s estimated that by 10-15 years of diabetes about 90% of patients with DM type 1 will develop some sort of retinopathy, and about 60% of type 2 diabetes will have some sort of retinopathy.

– Patients with type 1 diabetes tend to have more aggressive disease “more aggressive fibrovascular proliferation”.

– Patients who develop type 1 diabetes in childhood, the risk to have retinopathy is very minimal before the age of puberty.</td></tr>
<tr><td>✻ Blood pressure: tight control of blood pressure is very important. In the UK prospective study “which was mainly looking at type 2 diabetes”. Patients were randomized into tight control and conventional treatment, but the study looked also to the effect of controlling blood pressure “tight vs conventional treatment”. It was found that should be controlled (<140/80 mmHg).</td></tr>
<tr><td>✻ Other important factors like: exercise, controlling obesity, blood lipid, pregnancy, nephropathy (renal transplantation may improve DR), smoking, cataract surgery and anemia.</td></tr>
<tr><td>– The combination of poor glycemic control in addition to high blood pressure is very destructive to the retinal circulation and we see it every day among our patients.
– Another study called ACCORD found the same phenomenon that control of blood sugar was very important. It also found that group of drugs used to control dyslipidemia (fenofibrate) by unknown mechanism was protective. It’s now an important argument that all diabetics should use fenofibrate.</td></tr>
</table>

• Ocular Manifestations

– **Iris:** Rubeosis Iridis
– **Lens:** Cataract diabetics are more prone to have it. (glucose affects osmolarity → the lens gets opacified)
– **Iridocyclitis:** inflammation of the iris and of the ciliary body. Also called "anterior uveitis" and "iritis"
– **Retinopathy:** the major problem is retinopathy, most common one.
– **Optic neuropathy.**
– **Third, Fourth & sixth nerve palsies:** especially in those who are not having good control.

Diabetic Retinopathy (DR)

– Most common microvascular complication of DM and can lead to retinal degeneration.

<table>
<tr><th colspan="3">The story behind diabetic retinopathy (2 components)</th></tr>
<tr><td>Neuropathy</td><td colspan="2">Very early in the course of diabetes, the retinal neurons are suffering even without vascular retinopathy. So, there is very early phenomenon of neuropathy that retinal neurons are suffering and many of them die early because of apoptosis as result of hyperglycemic exudative stress “centers of apoptosis are expressed by retinal neurons even in subjects without DR”.</td></tr>
<tr><td rowspan="2">Microvascular disease
“what we see clinically”.

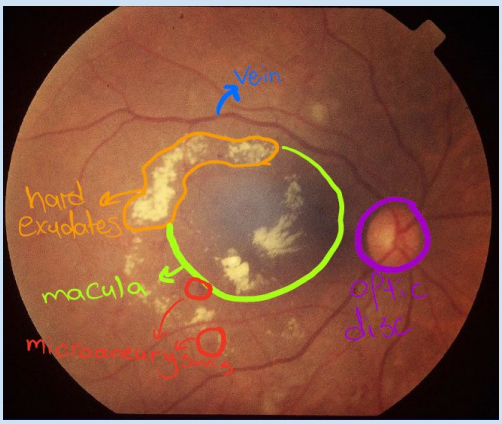
Has 2 major changes</td><td>1. Progressive vasculopathy</td><td>2. Progressive microvascular occlusion</td></tr>
<tr><td>Characterized by breakdown of blood retinal barrier (vessels of retina are lined by endothelial cells with tight junctions lying on basement membrane and surrounded by pericytes. The tight junctions of endothelial cells are responsible for integrity of blood retinal barrier). Very early in the course of diabetes, you have disruption of tight junctions proteins such as occludin and ve cadherin and this means that blood vessels become leaky so they leak fluid and lipoprotein and this will cause edema, and edema of macula is an important cause of moderate visual loss in diabetic retinopathy.</td><td>This will lead to retinal hypoxia and ischemia. Then retinal ischemia will activate transcriptional factors such as HIF-1-alpha “hypoxia- inducible- factor 1 alpha”. This HIF-1-alpha will upregulate many <u>angiogenic factors</u> and the most famous is vascular endothelial growth factor “VEGF”. VEGF is known to be hyperpermeability factor, It will cause breakdown of blood retinal barrier in addition, it’s an angiogenic factor (it induces proliferation, migration and tube formation of endothelial cells) which ends with formation of new vessels (proliferative disease). These new vessels are always accompanied by fibrous tissue. At the end, this fibrous tissue will cause traction of retina → tractional retinal detachment and the new vessels can bleed → the patient comes to ER with dramatic visual loss “suddenly he/she woke up and he/she cannot see”, and the first change that cause dramatic visual loss is vitreous hemorrhage. This is natural history of disease.</td></tr>
</table>

– Screening:

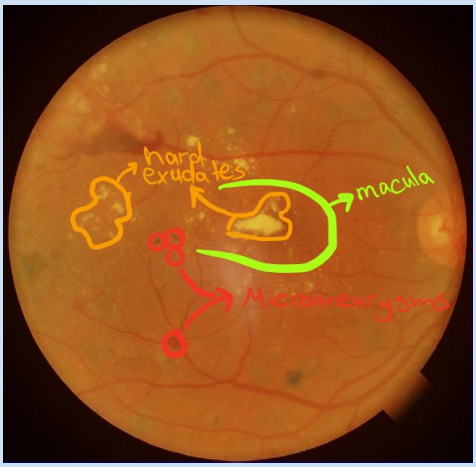
- You have to know that the **only way to prevent blindness** due to diabetic retinopathy is to do **regular screening for all diabetic patients**. Why? Because the patient might have blinding disease and he is still asymptomatic, and the time when he becomes symptomatic due to bleeding is too late.
- **The main objective of screening program** is to detect blinding disease at the stage when still asymptomatic (**still treatable with laser photocoagulation**). We should also discuss injecting **anti-VEGF agents** (3 drugs available: Bevacizumab “Avastin”, Ranibizumab “Lucentis”, Aflibercept “Eylea”) that help in controlling diabetic retinopathy.
- The screening is done by using a camera **“non mydriatic fundus camera”**. The camera will take a picture and the picture will be sent to computer and the doctor will look into the picture grading the severity of retinopathy (if the patient need laser or only follow up). **This is the international guideline to use non mydriatic fundus camera for screening.**
- Patients with type 1 diabetes don’t need screening unless they have **5 years** or more of diabetes. Patients with type 2 diabetes need immediate screening because many of them have diabetes which wasn’t recognized. If the retina is ok then the screening is done **every year.** If mild non PDR → done after **6 months**. If it’s severe non PDR, PDR or macular edema → **refer to laser.**

Non-Proliferative Diabetic Retinopathy (NPDR)

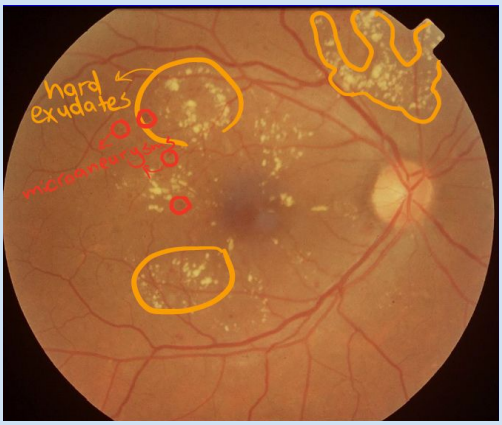
This is the right eye of patient with diabetes. This is optic disc with optic nerve head. This is the macula. If you look to the retinal vessels, the **veins are always darker than arteries and broader**. You can see in the macula, the collection of hard exudate (composed of lipoprotein and lipid laden macrophages). These hard exudates are result of **leakage**, it's a sign of **macular edema**. You can see small red dots, theses dots are microaneurysms and these are the source of leakage so the management here is to do **focal laser photocoagulation** (What are the targets of focal laser? Red dots. Focal laser has been shown to be associated with better outcomes compared to no treatment) Nowadays we have available anti-VEGF agents that can be injected to the eyes, So if the edema is involving the center of macula like in this patient .we can enhance the effect of laser by giving injection of anti-VEGF agents



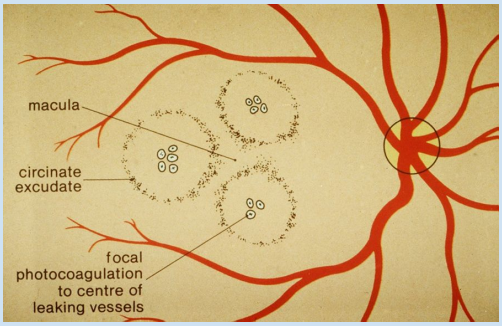
This is another example of **macular edema**, you can see the **hard exudates** (hard exudates are the result of leakage "breakdown of blood retinal barrier"). And you can see the red spots that we need to treat with laser photocoagulation

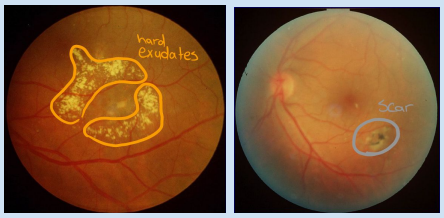
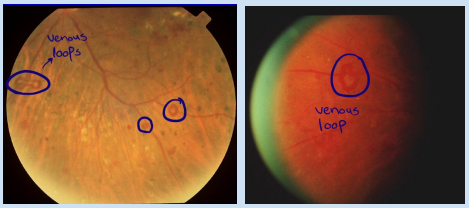
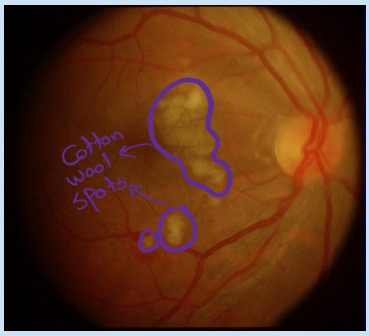
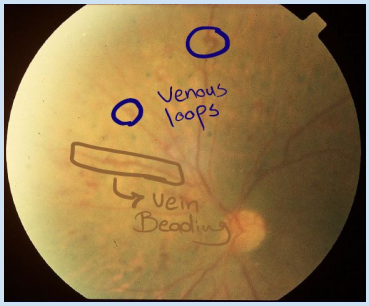
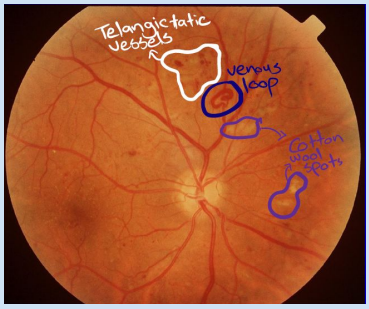
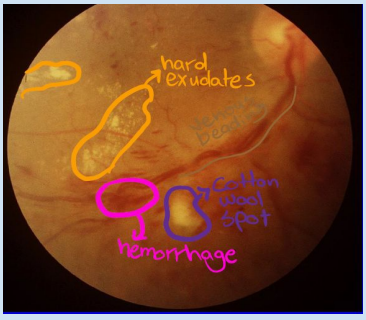


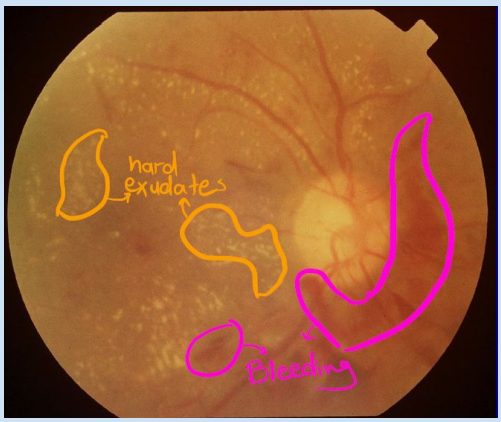
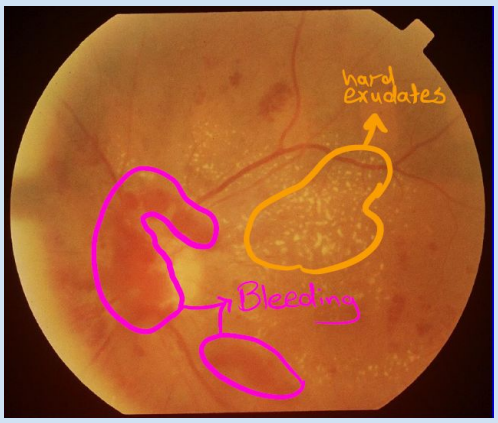
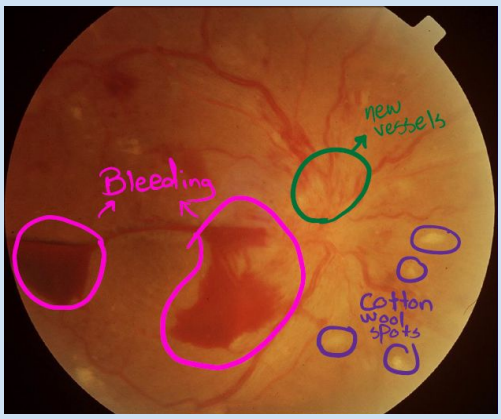
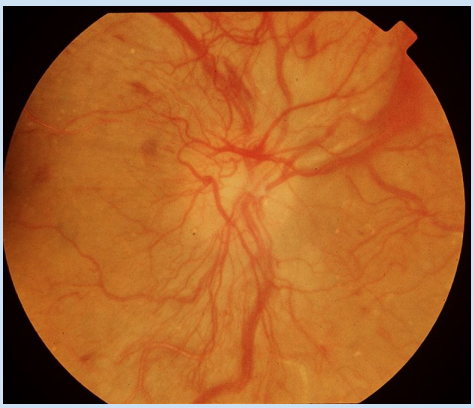
Another example of more extensive **hard exudates**, and you can see the red dots "we should close by focal laser photocoagulation"

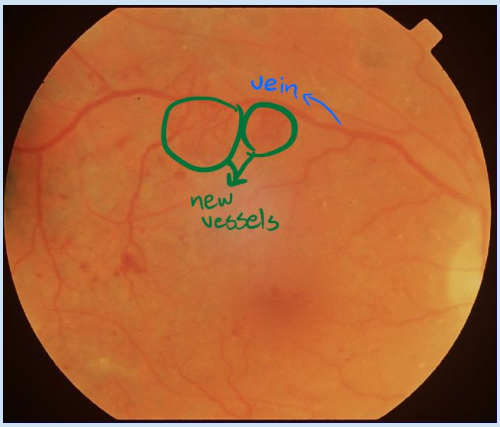


This cartoon shows you how we do focal photocoagulation
(hard exudates are the rings)
(microaneurysms are the small dots inside the rings)

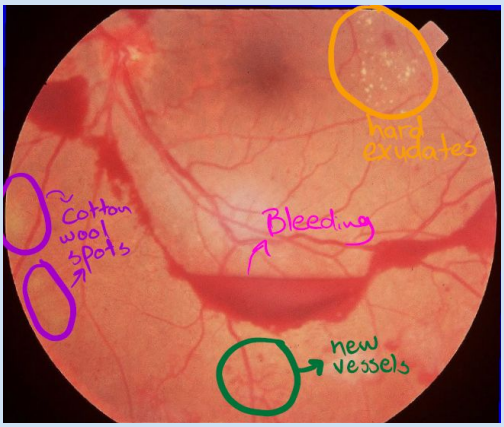


This is a patient who had a large ring of hard exudates and these are laser scars, usually it takes up to 6 months for hard exudates to absorb then after 6 months as you can see in the second picture, there is a complete resolution of hard exudates.	
You can see venous loops.	
All the previous pictures showed leakage component of retinopathy. Other component as we said is progressive occlusion of retinal arterioles and this will cause ischemia. There are signs in the retina that can tell me that the retina is very ischemic before development of new vessels, and these signs we call them **severe nonproliferative retinopathy**. One of these signs is **cotton wool spots** (the result of infarction of retina due to occlusion of retinal arterioles) Why did they call it like that? Because it's similar to small pieces of cotton	
The most important sign of **severe retinal ischemia** and the most reliable sign is venous changes. What are these changes? **Venous loops**. Also look to the course of this vein, there are dilated areas and constricted areas **(beading)**. So venous looping and beading are very important signs of retinal ischemia and the most important signs of retinal ischemia are venous changes.	
Venous loops. They call it sometimes **omega sign.** You can see the cotton wool spots. We have another sign of retinal ischemia called **Intraretinal microvascular abnormalities**, these are dilated telangiectatic vessels within the retina, the origin is not well known it can be collaterals, it can be new vessels still within retina. You can see here the dilated telangiectatic vessels. These are all signs of **severe non proliferative retinopathy** and we tend to treat with **pan retinal photocoagulation** in this stage.	
You can see venous beading (dilated and constricted parts), cotton wool spots, hard exudates. The patient has signs of ischemia and leakage. here are a lot of intraretinal hemorrhages (this is another sign of severe retinal ischemia, presence of **intraretinal hemorrhages in 4 quadrants** is a sign of severe non- proliferative retinopathy). To summarize **what are the signs of severe non- proliferative retinopathy**: **Venous changes, (IrMAs) Intraretinal microvascular abnormalities, hemorrhages in 4 quadrants** "cotton wool spots are less important"	

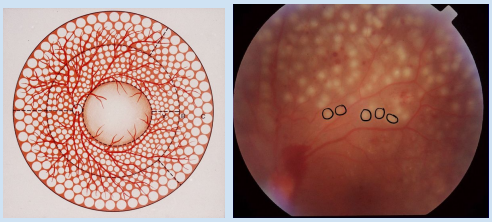
Proliferative Diabetic Retinopathy (PDR)	
	45% of patients with severe non-PDR will progress into proliferative disease within one year. What is the definition of proliferative disease? **Neovascularization** and the most important site for neovascularization is **optic nerve head**. In the pic, if you look to the optic disc you can see new abnormal blood vessels with bleeding. In addition the patient has hard exudates. This is a proliferative blinding disease and the patient needs urgent intervention in the form of **pan-retinal photocoagulation.**
	You can see bleeding of new vessels so this is a proliferative disease with hard exudates (we frequently see this combination)
	This a patient with proliferative disease and we can see here blood in front of retina, cotton wool spots, venous changes and new vessels on the optic disc
	This is another example of a very aggressive new vessels. The New vessels are coming from optic disc.



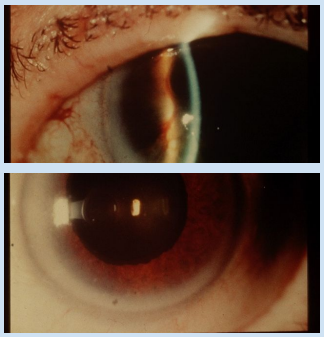
New vessels can develop outside the optic nerve and always from the veins. Here we can see new vessels originating from veins outside the optic nerve



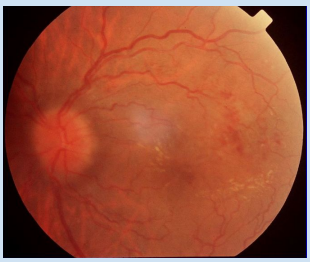
Proliferative disease. Patient now has bleeding 'subhyaloid hemorrhage'. You can see the neovascularization, hard exudates and cotton wool spots.



Left Picture: now the treatment of proliferative DR or severe non PDR is by **laser** (pan-retinal photocoagulation. Why is it call like that? Because you apply scattered laser burns throughout the retina sparing the optic nerve and macula and this automatically will be followed by regression of new vessels)
Right Picture: example of pan-retinal photocoagulation. These are laser burns. If there is extensive exudates and hemorrhages, PRP "panretinal photocoagulation" is done (the whole periphery gets cauterized except the posterior pole). results in loss of rods → **loss of vision at night**.



Another big complication of retinal ischemia is formation of new vessels on the iris and the angle of anterior chamber, this is called **Rubeosis iridis** (neovascular glaucoma). As a result of ischemia, the new vessels will not only develop on the retina, they develop also on the iris and it involves the angle "will close the angle by fibrous tissue", this will cause very aggressive type of glaucoma called neovascular glaucoma and it's a blinding disease. The angiogenic factors like VEGF will move into the anterior chamber and cause neovascularization of iris and the angle. This happens with any retinal ischemia like central retinal vein occlusion, but mainly with DR.



The following summarizes everything:

- **Changes seen in DR:**

1. Hard exudates on the retina and macular edema resulted from the leakage of and lipoproteins “yellow spots”.
2. Microaneurysms ‘blots and dots’.
3. Soft exudates ‘cotton-wool spots’ “white spots”.
4. Venous changes: beading vs looping (The most reliable signs of retinal ischemia in diabetic retinopathy).
5. Hemorrhage if neovascularization results in weak vessels and easy to break.
6. Neovascularization.

* Circinate exudate: when blood vessels leak it will result in Exudates and hemorrhages AROUND the source of leaking.

- **Non PDR**
  - Mild NPDR: microaneurysm only.
  - Moderate NPDR: microaneurysms – retinal haemorrhages - circumstances exudates – cotton wool spots – minimal intraretinal microvascular anomalies (arteriovenous shunt) – minimal venous changes (looping and beading).
  - Severe NPDR : all of the above + severe intraretinal microvascular anomalies (arteriovenous shunt) – severe venous changes (looping and beading) ‘Consider panretinal laser coagulation’

– Intraretinal microvascular abnormalities (IRMA): are arteriolar– venular shunts that run from retinal arterioles to venules, thus bypassing the capillary bed and are therefore often seen adjacent to areas of marked capillary hypoperfusion.
– Dilated telangiectatic capillaries Intraretinal Hemorrhage: The extent of involvement is a significant marker of the likelihood of progression to proliferative diabetic retinopathy.
– Diabetic macular edema “DMO”: Nowadays, Optic coherence tomography (OCT) is used to diagnose early macular edema even in patients with very mild edema that you can’t see it.
– **Treatment:**
1. Focal laser photocoagulation: Treatment of micro aneurysms with laser (After 6 months there will be a scar from the laser treatment but no hard exudate). Very effective in controlling diabetic macular edema.
2. Intravitreal injection of anti-VEGF agents (anti- vascular endothelial growth factor) in addition to laser, we frequently inject Intravitreal injection of anti-VEGF agents to help control diabetic macular edema. * Diabetic retinopathy patients have progressive macular vascular occlusion, which will cause ischemia of retina (hypoxia).

- **PDR**

– New vessels at the disc (NVD): describes neovascularization on or within one disc diameter of the optic nerve head.
– New vessels elsewhere (NVE): describes neovascularization further away from the disc; it may be associated with fibrosis if long-standing.
– New vessels on the iris (NVI): also known as rubeosis iridis, carry a high likelihood of progression to neovascular glaucoma.
– **Treatment:** Pan-retinal photocoagulation | Intravitreal anti-VEGF injection
– **Complications of Retinal photocoagulation:**
1. Anterior segment complications such as corneal or lenticular opacification
2. Transient visual loss.
3. Photocoagulation of the fovea.
4. Macular edema.
5. Hemorrhage.
6. Choroidal Effusion
7. Color vision alterations. Visual field defects and night vision problems.
8. Hemeralopia.

Graves' Disease

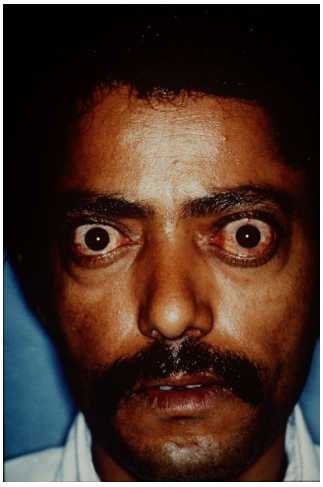
-wasn't mentioned by Prof. Abu El-asrar-

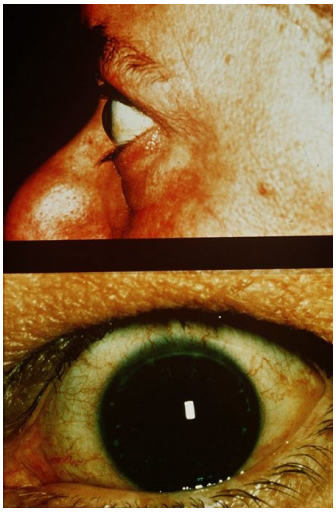
• Ocular Manifestations

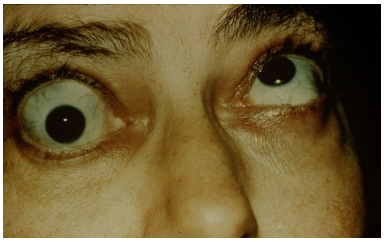
– Eyelid retraction.
– Infiltrative ophthalmopathy.
– Proptosis and exophthalmos.
– Dysthyroid optic neuropathy.
– Restrictive thyroid myopathy.
– Lid lag, chemosis, exposure keratopathy, ophthalmoplegia.

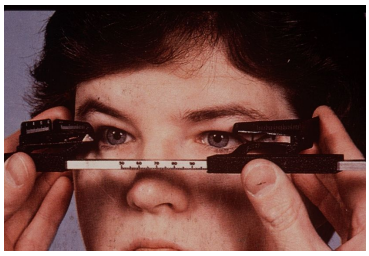
– Most common cause of both **bilateral** and **unilateral** proptosis in an adult.
– **Pathogenesis:**
  ○ Autoimmune disease characterized with serum IgG antibodies bind to TSH receptors in the thyroid and causes overstimulation and high thyroid hormone production.
  ○ Autoimmune antibodies infiltrate eye, cause inflammation of extraocular muscles and associated with increased secretion of glycosaminoglycans and osmotic imbibition of water.
– **Risk factors:** smoking (most important) – family history
– **Systemic manifestation:** pretibial myxedema, heat intolerance, weight loss
– **Investigations:**
1. Thyroid function test: **high T3, T4 and low TSH.**
2. Visual evoked potential: To exclude Optic neuropathy.

– **Treatment:**
1. Anti-thyroid medications or thyroid ablation with radioactive iodine (for disease itself)
2. Steroid, lubricants and eye protection before sleep (for eye symptoms)
3. If there is restrictive myopathy surgical intervention is required









Tuberculosis

Ocular features:

– Phlyctenular keratoconjunctivitis a hypersensitivity reaction of the cornea and conjunctiva to bacterial antigens, is characterized by discrete nodular areas of corneal or conjunctival inflammation
– Interstitial keratitis – Vitritis - Choroidal granuloma.
– Uvea TB can affect eyes causing uveitis "a study was done to reveal that Tuberculous uveitis is the second most common cause of uveitis as a referral in KAAUH". If uveitis is only involving the anterior part like iris → called anterior uveitis. If it's involving the posterior part like choroid → called posterior uveitis. If it's involving the whole uvea → pan-uveitis. TB is an important cause of uveitis, uveitis in TB can mimic anything
– Retinal vasculitis 'Eales disease'.

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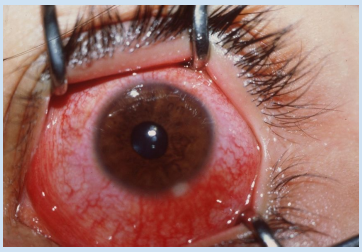
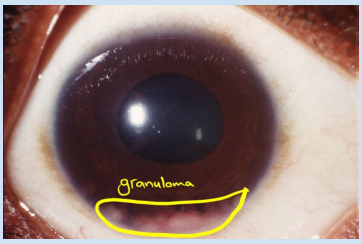
<td>  </td>
<td> 
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<td>This is a 16-year-old patient. Can you see the pinkish nodules in the angle? She had granulomas 'Phlyctenular keratoconjunctivitis' . When you see such granulomas you have to think about 2 conditions: either TB or sarcoidosis. This patient had many members of family with TB and she was managed with systemic anti-tuberculous treatment with improvement.</td>
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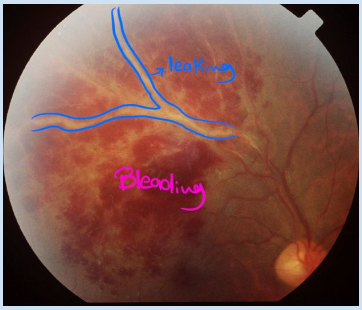
<td> 
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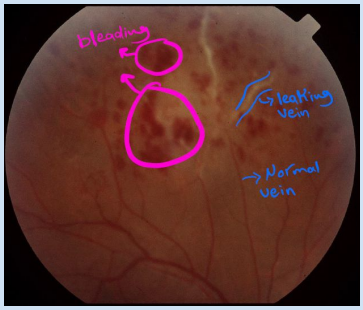
<td colspan="2"> 
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<tr>
<td>Patient came to the ER. You can see white veins (due to exudates) + hemorrhages "tuberculous retinal vasculitis" respond to anti-TB treatment, if you don't give anti-tuberculous treatment it will end by losing the eye.</td>
<td colspan="2">Another common manifestation of ocular TB is retinal vasculitis. If you look here to the retinal vessels, this is a normal vein but you can see it turned white with a lot of intraretinal hemorrhages meaning that this vein is inflamed and leaking blood. So this white appearing-vein is called cheesing and it's a sign of retinal vasculitis, it's due to peri-venous accumulation of inflammatory exudates and cells. TB is a common cause of occlusive retinal vasculitis. We have the advantage in the retina that we don't need biopsy to diagnose vasculitis, we see it clinically.</td>
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</table>

– TB is a chronic granulomatous infection usually caused in humans by Mycobacterium tuberculosis.
– TB is primarily a pulmonary disease but may spread by the bloodstream to other sites; ocular involvement (TB can involve any part of the eye) commonly occurs without clinically overt systemic disease. Extrapulmonary TB when you have an eye infection without pulmonary infection in 60% of cases.
– Tubercles uveitis is an important cause of blindness
– TB may be indolent and the first manifestation in the eye.
– Can be: 1. Direct infection 2. Immune response to tubercular protein
– TB is the second most common cause of uveitis in KSA, after Vogt- koyanagi-Harada disease, and the third cause is Behçet disease.

– Granulomatous inflammation that is the disposition of mutton-fat keratic precipitate, iris nodules, infiltration of the choroids, and retinal vasculitis. These are the most important manifestation of TB in the eye.
Mutton-fat keratic precipitation: collection of inflammatory cells on the corneal endothelium appear large with yellowish color. (can be seen as white dot inferiorly, mostly due to staph but could be caused by TB).
– **Investigations:**
1. First you should take a good history. (family history or history of exposure will increase the chance that the eye inflammation is caused by TB).
2. CXR to roll out that the patient has previous infection in the chest.
2. We rely more to tuberculin skin test, if it was strongly positive, 15 mm or more induration, this will support the diagnosis.
3. PCR and the interferon-gamma release assay (IGRA).
4. Aqueous or vitreous sampling rarely yields demonstrable (smear – acid-fast bacilli on Ziehl–Neelsen staining – or culture – Lowenstein–Jensen medium).
– **Treatment:**
1.Prolonged Anti-TB therapy *multi-drug therapy (4 drugs in 2 months, then continue for 6 months with 2 drugs). Isoniazid (with Vitamin B6 (pyridoxine) to prevent the development of peripheral neuropathy), rifampicin, pyrazinamide and ethambutol- Ethambutol can cause optic neuropathy-
2- Topical and systemic steroids may be used concomitantly to reduce inflammation- induced damage.

Leprosy (Hansen's Disease)

-Prof. Abu El-asrar skipped it-

– Ocular involvement is more common in the lepromatous type.
– **Signs:** Facial nerve affection, Loss of the lateral portions of the eyebrows and eyelashes (Madarosis), Interstitial keratitis, Iritis.

Syphilis

- **Congenital:** transplacental infection

– Interstitial keratitis
– Chorioretinitis

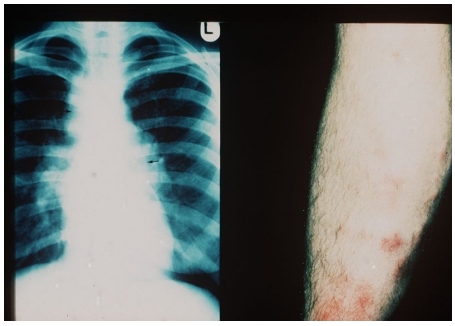
- **Acquired**

– Ocular chancre.
– Iridocyclitis.
– Interstitial keratitis.
– Chorioretinitis.
– Neuro-ophthalmic.
– When we see a patient with uveitis you always rule out syphilis (this is international recommendation) by doing serological testing of syphilis "VDRL, fluorescent treponemal antibody absorption (FTA-ABS)". Despite this, we diagnose syphilis very rarely here, but when we look to western countries like UK there are a lot of cases of syphilis "syphilitic uveitis" which means that until now so far we are protected against this bad disease. London is a city full of syphilis, the patient comes with syphilitic uveitis → receives treatment → cured then he will come again with another attack of syphilitic uveitis due to another exposure. They call syphilis the great mimicker because it can cause any type of eye inflammation and that's why we always do serology for syphilis in any patient with uveitis.

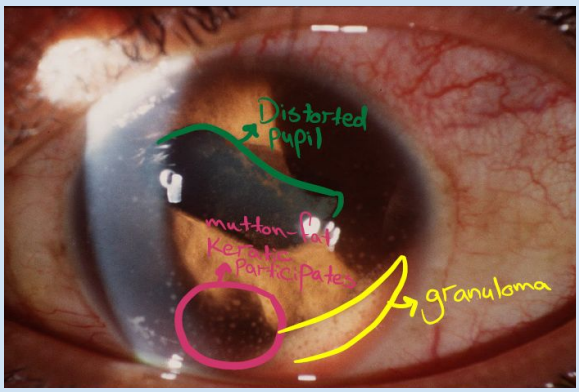
Sarcoidosis

• Eye lesions

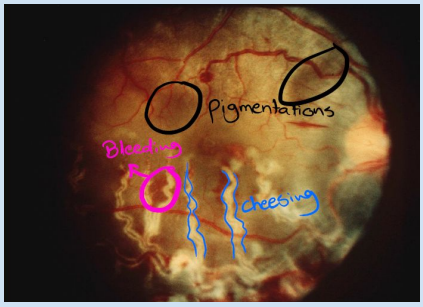
– Lid margin and conjunctival granuloma
– Acute iridocyclitis
– Chronic granulomatous iridocyclitis
– Peripheral retinal periphlebitis
– Choroidal granulomas
– Retinal granulomas
– Optic nerve head granulomas



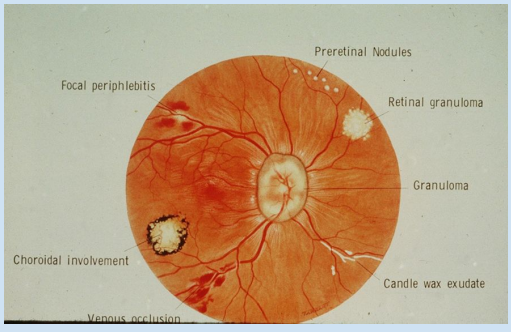
– Sarcoidosis is an important cause of uveitis. It's not common here but in a country like Japan, sarcoidosis is the most common cause of uveitis.
Sarcoidosis causes **non-caseating granulomas** when compared to TB that causes caseating granulomas. Retinal vasculitis can also be seen in sarcoidosis.
– When we suspect sarcoidosis as a cause of uveitis, we always ask for CT of the chest. What do you expect to see in CT? Hilar lymphadenopathy and also granulomatous infiltration of the lungs.



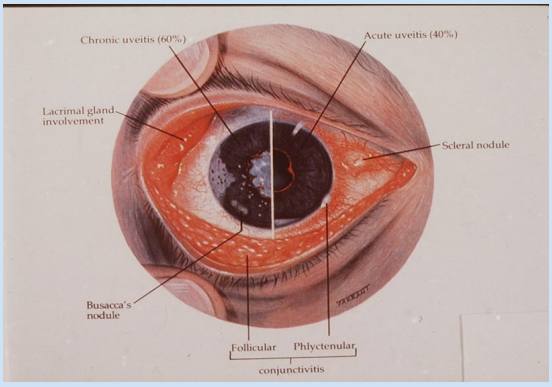
This a good example of a patient with sarcoidosis to show you important signs. You can see the cornea and behind the cornea is the anterior chamber then iris then lens. You can see whitish deposits on the back of cornea these are called **Keratic participates** which is an important sign in uveitis. Keratic participates in sarcoidosis are big therefore they're called **mutton-fat**. So the differential diagnoses when you see mutton-fat Keratic precipitates → sarcoidosis, TB, syphilis, Vogt- koyanagi-Harada and multiple sclerosis "MS is an important cause of uveitis" because mutton- fat precipitates are a sign of granulomatous inflammation. You can see in the angle a big granuloma. Distorted pupil caused by synechiae, when you have chronic granulomatous inflammation then you can have adhesions between the pupil and lens so when you dilate the pupil the pupil doesn't dilate.



This is a 14-year-old African American patient "you can see pigmentations of the fundus are black" (a lot of African American have sarcoidosis). In the pic, you can see a lot of vasculitis and cheesing. The patient responded very quickly to systemic corticosteroids



Can affect the optic nerve, it can occur on the retina and we can have vasculitis

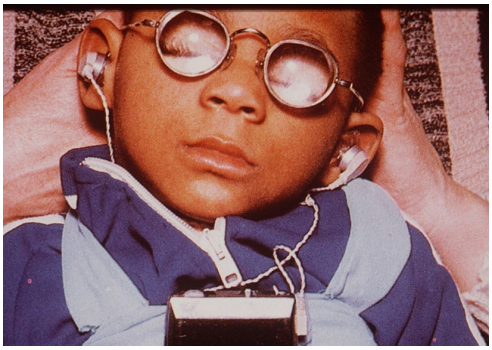


– **Systemic manifestation:**
  - The triad: erythema nodosum - bilateral hilar lymphadenopathy – polyarthralgia.
  - Could be the same as TB manifestation
– **Ocular manifestation:**
  - Candle-wax exudate in the retina
  - Optic nerve, Retinal, Choroidal Lid margin and conjunctival granulomas.
  - Mutton-fat keratic precipitates
– **Investigations:**
  - Tuberculin skin test: negative in sarcoidosis
  - Chest X-ray: showing bilateral hilar lymphadenopathy (BHL) (DIAGNOSTIC)
  - Elevated serum ACE levels and/or elevated serum lysozyme
  - Abnormal liver enzyme tests

  Biopsy should be taken to confirm the diagnosis, if we were in doubt.
– **Treatment:**
  - Steroid and NSAIDs.

Rubella

– Cataract
– Microphthalmos
– Retinopathy pigmentary retinopathy: salt and pepper.
– Glaucoma
– Anterior uveitis: unresponsive to steroids.
– They use VERY big glasses. they use hearing aid also.
– If the mother is infected with rubella virus, the baby can be born with congenital rubella syndrome (the baby will have cataract, small eyes "Microphthalmos", retinopathy and glaucoma). Systemically, they have heart disease and deafness.



Wilson's Disease

– Hepatolenticular degeneration.

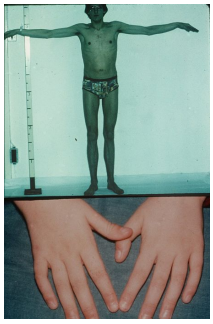
- **Ocular features:**

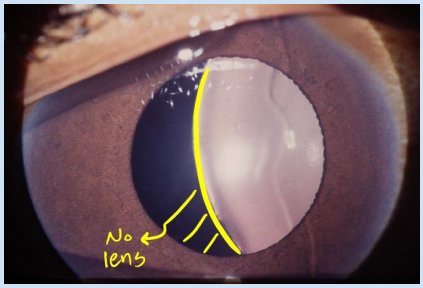
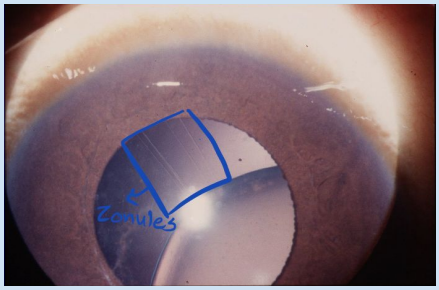
– Kayser-Fleischer ring consists of a brownish-yellow zone of fine copper dusting in peripheral descemet membrane detected with gonioscopy (Important sign)
– Green sunflower cataract
– There is excessive copper deposition in the tissues due to deficiency of the carrier protein which is called alpha 2 globulin "ceruloplasmin". So in the eye, the copper can be deposited at the peripheral part of Descemet's membrane and this will cause Kayser-Fleischer ring (the presence of this ring is diagnostic for Wilson's disease). Copper can also be deposited in the lens causing green sunflower cataract.
– **Systemic manifestation:** Liver disease, Basal ganglia dysfunction, Psychiatric disturbances.
– **Treatment:** Penicillamine.

Marfan's Syndrome

- **Ocular features**

– Lens subluxation the major feature of the disease is lens subluxation
– Angle anomaly
– Glaucoma
– Hypoplasia of the dilator M.
– Axial myopia
– Retinal detachment
– **Picture:** this is the systemic manifestation with arachnodactyly.

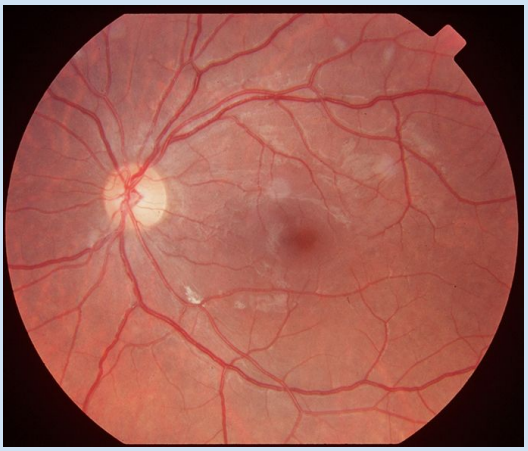


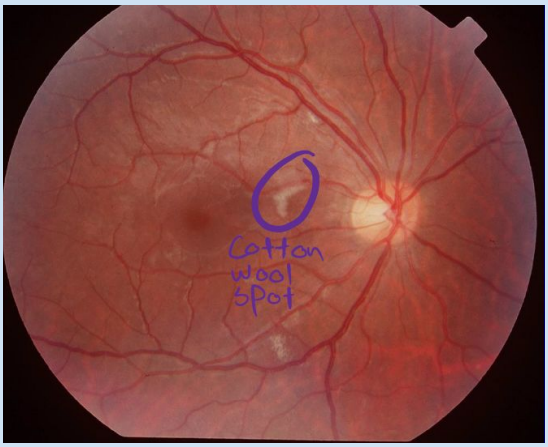
	
This iris and the pupil. What do you see behind the pupil? Subluxated lens, you can see the equator of the lens visible through pupil	Another patient with subluxated lens and you can see the stretched zonules

It is an autosomal dominant disease.
Systemic manifestation: Arachnodactyly (Long fingers), Heart diseases, Bone deformities

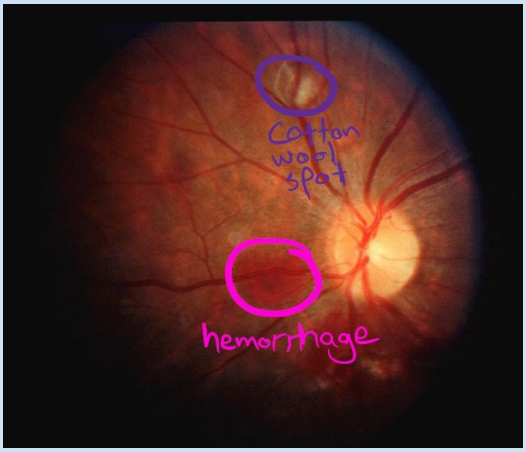
Systemic Lupus Erythematosus

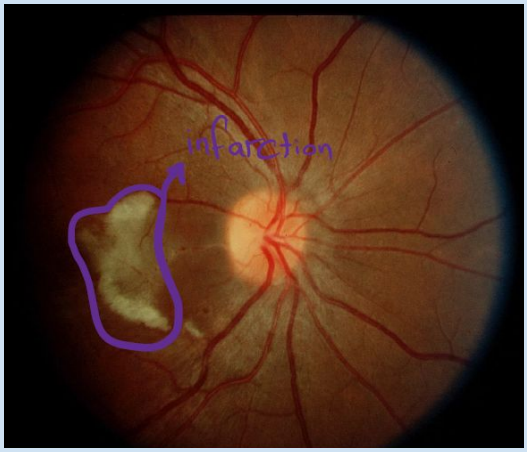
– If you see a patient with these facial features, what's your diagnosis? **SLE** "an autoimmune disease with multiple autoantibodies like: **antinuclear antibodies, anti ds DNA antibodies, the patient has high ESR, low C3 and C4**". If the disease is active, it can affect the eye particularly retina. The retinal affection will be more if the patient was also positive for antiphospholipid antibodies "lupus anticoagulants, anticardiolipin antibodies"
– What do you see in the retina? The disease mainly manifests as **retinopathy in the retina** "**cotton wool spots**" meaning that it causes occlusion of retinal arterioles. So the main pathology is micro-thrombosis of retinal arterioles causing multiple retinal infarctions "cotton wool spots with or without hemorrhages"
– Senario: young lady with Bilateral multiple cotton wool spots
(Always think of SLE)





Good example of cotton wool spots. This is a mild disease

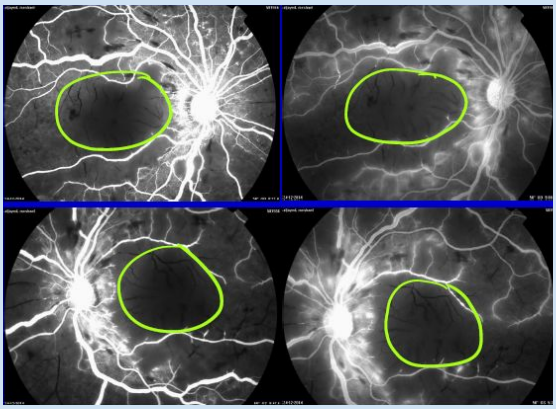




More severe disease: cotton wool spots & hemorrhages

Occlusion of this arteriole causing infarction

This is a very dramatic presentation. This was a young 22-year-old lady. The treating ophthalmologist thought that these white patches are retinitis, he didn't think about infarctions. The moment we saw the patient in the ER we made a diagnosis of possible SLE. The patient was admitted and she was positive for antinuclear antibodies, anti ds DNA antibodies referred to rheumatology for treatment.



Fluorescein angiography. You can see all these black lines, these are occluded retinal vessels with extensive ischemia and infarction. You can see that the macula is severely ischemic we ended with only 20/200 vision. In order for SLE to cause retinopathy, you need active disease not controlled medically.

Rheumatoid Arthritis

– A seropositive disease Which factor do you need in order to diagnose RA? Rheumatoid factor

- **Ocular features:**
  - K.C.S. Keratoconjunctivitis sicca "Dryness of the eye" (autoimmune disorder attacking the lacrimal gland). Positive Rose Bengal staining → K.C.S
  - Scleritis important cause of sclerites and melting of the sclera if not controlled
  - Keratitis.

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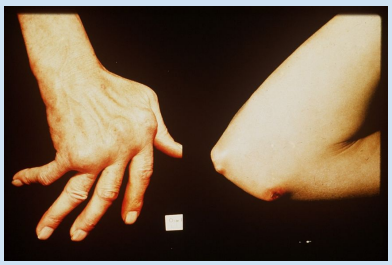
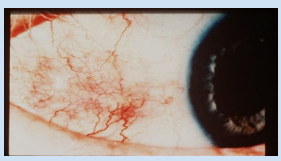
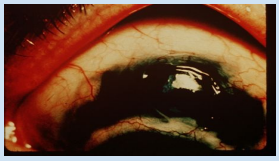
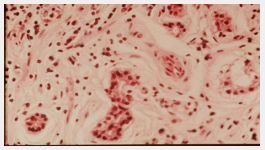
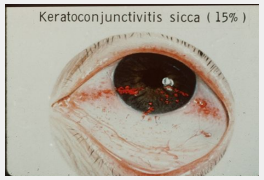
<td>  </td>
<td>   </td>
<td>   </td>
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<td>In the right picture: radial deviation, swollen fingers and nodules in the elbow.</td>
<td>*Melting of the sclera with "scleromalacia perforans" exposing the underlying different tissues
*To control this type of inflammation we need drug like cyclophosphamide</td>
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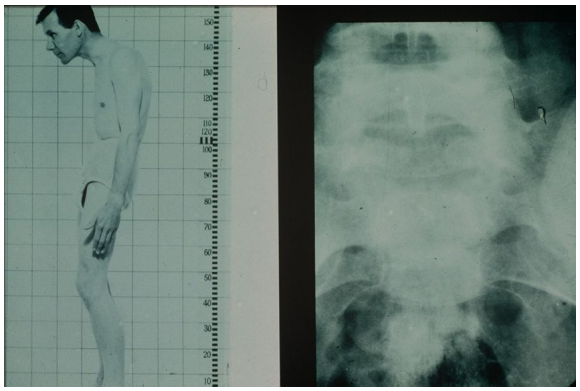
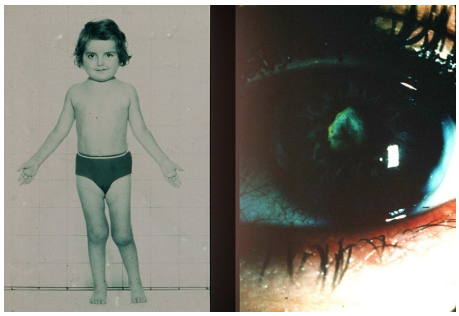
Ankylosing Spondylitis -Important-



– Seronegative
– X-rays of sacroiliac joints shows juxta-articular osteoporosis in the early stages .
– Acute recurrent **non-granulomatous** iridocyclitis
– This is an interesting disease for us. Whenever we see a **young male patient** in the ER having a **unilateral acute non-granulomatous anterior uveitis** **ankylosing Spondylitis** is the most important differential diagnosis, and to rule it out, **what kind of test should we do? HLA-B27 typing** is the most important test.
– This type of uveitis can happen in patients who are positive for HLA-B27. It can be systemic disease like: **ankylosing Spondylitis, psoriasis, Reiter's disease, IBD**. This type of uveitis can also occur without systemic disease.
– So whenever we see a young male patient with acute recurrent non-granulomatous (what do you mean non-granulomatous? You don't see mutton- fat precipitates which is sign of granuloma) iridocyclitis then we have to rule out ankylosing by HLA-B27 typing
– It's very important when we make a diagnosis like this to **refer** the patient because the patient can present for the first time to ophthalmology. So we have to refer the patient to rheumatology because at this stage if the patient has ankylosing spondylitis, you have to start **systemic treatment** early to prevent deformity.

– **Systemic manifestation:** Pain and stiffness in the lower back with limitation of movement, Calcification of spinal ligaments gives rise to a 'bamboo spine'.
– **Ocular manifestation:** Acute recurrent non- granulomatous anterior uveitis
– **Complications:** synchia. | – **Investigations:** HLA-B27-positive, X-ray: sacroiliac joints

Juvenile Chronic Arthritis
-Juvenile Rheumatoid Arthritis-



– The patterns of disease: **-important-**
1. Systemic onset: "Still's disease" uveitis is extremely rare
2. Polyarticular onset: uveitis is fairly rare
3. **Pauciarticular onset**: about 20% develop uveitis at the onset, **4 or less joints are affected**

– In the western countries, this is the most common cause of uveitis in children.
– This is a disease of children. The eye can be affected by blinding inflammation. The problem here is that children cannot complain and the eye looks quite, but the chronic inflammation can destroy the eye. That's why we have to know the **risk factors** for a child with juvenile chronic arthritis to develop uveitis **(1) being a girl (2) If arthritis developed before age of 4 3-positive antinuclear antibodies.**
– If the child presents with systemic onset "which is called still's disease": the child is presenting with fever, maculopapular rash, lymphadenopathy and hepatosplenomegaly and pericarditis. With this presentation, **uveitis is very rare**.
– Polyarticular onset: at presentation, 5 or more joints are affected and **still uveitis is rare**.
– **Uveitis is common with Pauciarticular onset.**
– Complications are common mainly glaucoma and cataract

– **Ocular manifestation:** Chronic non-granulomatous uveitis, Band keratopathy, Posterior synechiae.
– **Investigations:** Anti-ANA antibodies: will be positive in majority of pauciarticular type, Rheumatoid factor: positive in some polyarticular type, HLA-B27: it will be positive in some patient.
– **Treatment:**Topical and systemic Steroid and mydriatic agent to prevent posterior synechiae.

Behcet's Disease

– Anterior nongranulomatous uveitis
– Very common disease. In our recent publication, it was the third most common cause of uveitis here.
– It's a disease of multisystem vasculitis. The major cause of **visual loss** in patients with Behcet's disease is recurrent episode of **vaso-occlusive retinal vasculitis –This is very important-**.
– Patients with Behcet's disease have very important involvement of **polymorphonuclear leukocytes** in the pathogenesis of the disease **–very important-.**
– We see many patients who present with ulcers but after having recurrent episodes of **DVT.**
– How we make diagnosis of Behcet's disease? there is no specific lab study to diagnose Behcet's disease, the diagnosis is a **clinical** one based on specific signs and symptoms that were proposed by international Behcet's disease study group.
– The criteria required: **recurrent painful oral ulcer** (mouth ulcers should be in all patients. Why? Because if you look to epidemiological studies, mouth ulcers was the most common manifestation of the disease in about 97% of the patients). **In addition to the mouth ulcer, you need 2 of the followings: skin lesions, recurrent genital ulcers, eye manifestation –uveitis-**
– The country that has highest incidence of disease is Turkey. The disease is highly prevalent in what's called silk road "الطريق اللي كان يسلكه تجار الحرير ما بين حوض البحر المتوسط والصين". Is very common around mediterranean basin, China, Japan, Korea, Turkey and among us. You don't see it in Caucasians.

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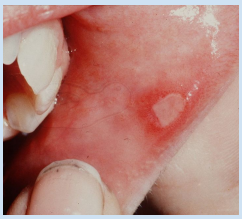
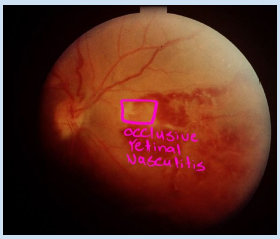
<td>  </td>
<td> 
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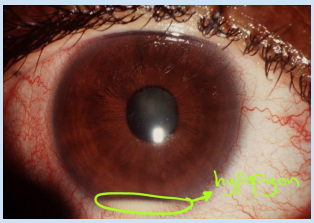
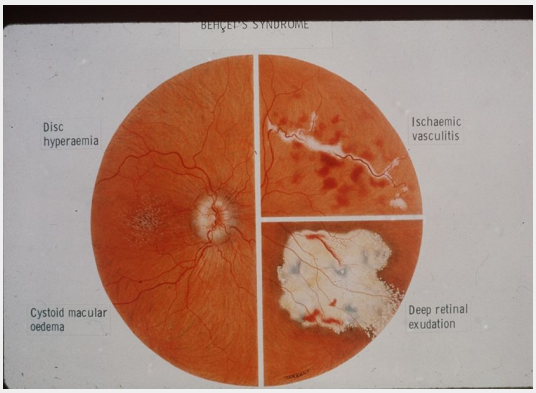
<td>  </td>
<td rowspan="2"> 
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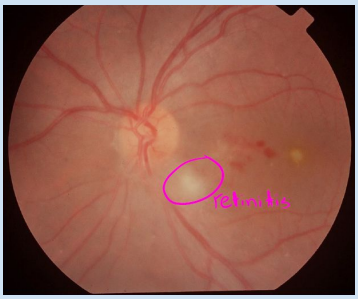
<tr>
<td>Aphthous ulcer is very painful and recurrent</td>
<td>Occlusive retinal vasculitis</td>
<td>Hypopyon due to polymorphonuclear leukocytes infiltrating into the anterior chamber</td>
</tr>
<tr>
<td>  </td>
<td colspan="3">It causes retinitis as you can see here , and it’s a blinding explosive disease. In the past, if you look into the literature they were telling whatever you do, the patient becomes blind. Now we have very effective treatment to stop blindness such as anti- TNF alpha agents “infliximab”. Infliximab very effective and prevents blindness in this disease. it’s the most effective drug available, it blocks an important pro-inflammatory cytokine called TNF alpha. Why is it very effective? Studies showed that if you analyze a sample from aqueous humor of patients with Behcet's disease, they have highest level of TNF-alpha which means that the inflammation is dependent on TNF-alpha in Behcet's disease.</td>
</tr>
</table>

– **Investigations:** HLA-B51 is positive | Pathergy test: pustule 24–48 hours after a sterile needle prick

Reiter’s Syndrome

-Prof. Abu El-asrar skipped it-

- **A triad of:** Urethritis, Conjunctivitis, Seronegative arthritis
- **Ocular features:** Conjunctivitis, Keratitis, Iridocyclitis

Sjogren’s Syndrome

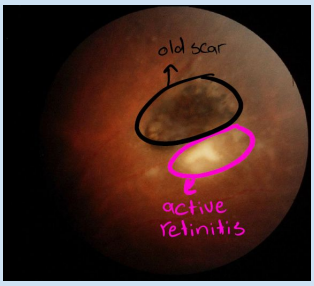
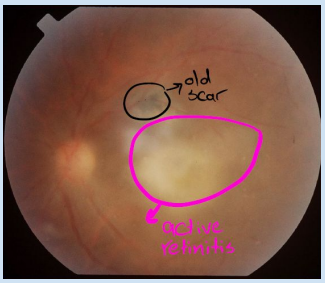
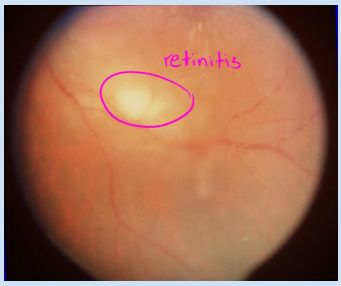
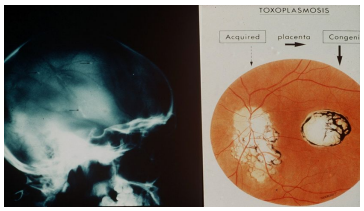
-Prof. Abu El-asrar skipped it-

– Autoimmune disease
– Involvement of: salivary glands, bronchial epithelium, vagina.
– Ocular features: K.C.S. Keratoconjunctivitis sicca “Dryness of the eye”
– Systemic manifestations: Dryness of skin and mouth and arthralgia and polyneuropathy.
– Investigation:

1. Schirmer tear test
2. Positive Rose Bengal staining (for keratoconjunctivitis sicca)
3. ANA, RF positive
4. Associated with HLA-B8/DR3

Toxoplasmosis

- Caused by Toxoplasma gondii after eating raw meat, obligatory intracellular protozoan parasite, can be:
  - **Congenital:** Convulsions, chorioretinitis, intracranial calcification
  - **Acquired:** Reactivation of old lesion, manifest manly as necrotising, inflammation of retina (retinitis).
- The drugs we use to treat toxo-retinitis if it's needed to be treated: Clindamycin, Sulphonamides, Pyrimethamine (Daraprim) steroids, sulphadiazine, cotrimoxazole minocycline, azithromycin
- The fourth most common cause of uveitis in the country. It's an infectious cause of uveitis
- The severity of infection of baby depends on the timing of infection by mother. So if it happens **in the first trimester** what will happen to the baby? **Abortion**. If it happens in the **third trimester**, the baby will end up with **congenital toxoplasmosis**
- If the mother is infected for the first time in her life, the baby will become **infected** (no antibodies to protect the baby), but if she is infected as a recurrent infection, the baby is **protected**. That's why at the start of pregnancy they always order antibodies screening for toxoplasma. If the mother has IgG positive antibodies meaning that she was exposed before so there is no fear, but if the mother was seronegative at the beginning of pregnancy and then during pregnancy became positive then the risk is very high
- The acquired toxoplasmosis affects retina causing toxo-retinitis (**focal necrotizing retinitis** which usually located to adjacent scar)

			
This is an example of a patient with an old scar meaning that the patient had previous infection of the retina. Nearby the old scar is an active retinitis (this is a recurrent disease)	Another patient. You can see old scar and the active retinitis. Unfortunately this patient **lost his central vision** because it involved the center of the macula → very aggressive retinitis	Another patient. You can see the aggressive retinitis	

- **Ocular manifestation:** posterior uveitis, Macular lesion, Retinochoroiditis "fluffy white with pigmented scar" Unilateral floaters, blurring and photophobia.
- **Investigations:** PCR and serology

Vogt-Koyanagi-Harada Syndrome

- Pigmented individuals
- Cutaneous signs, Neurological signs, Anterior uveitis, Posterior uveitis.
- This is the **most common** cause of **autoimmune uveitis** in the country | More common in **young women**.
- Autoimmune inflammatory response directed by T-lymphocytes attacking antigens related to **melanocytes** causing: **vitiligo** (melanocytes in skin), **alopecia** & **white hair (**melanocytes in the hair), **white lashes (**melanocytes in eyelashes), [Poliosis: absence or decrease melanin in head hair, eyebrows or eyelashes], **deafness and tinnitus (**melanocytes in the inner ear), **severe headache (**melanocytes in the meninges)

– It's a multisystem disease. It tends to affect pigmented individuals (you will not see it in Caucasians)
– The disease is blinding but if we treat the patient early in the course of the disease **(large dose systemic corticosteroid combined with immunomodulatory agent such as mycophenolate mofetil** "an anti-metabolite like azathioprine and methotrexate but much safer") then we can prevent all of these complications.
– You should know about this disease because the disease is very common and it's a multisystem disease.
– Big problem in the country that not many ophthalmologists know how to diagnose it early so when the patient comes with headache and inflammation of the optic nerve (optic nerve disc swelling), they make wrong diagnosis of pseudotumor cerebri and they refer patient to neurologist (a lot of investigations done to the patient: MRI, lumbar puncture) then the patient will become blind. So we have to have high index of suspicion for the diagnosis of Vogt-Koyanagi-Harada disease.

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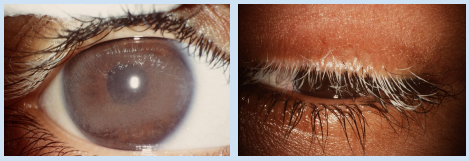
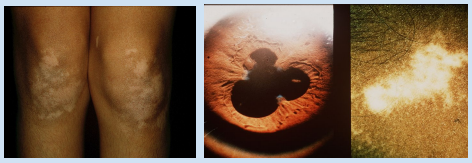
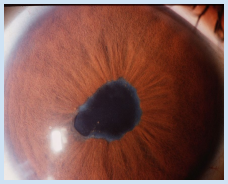
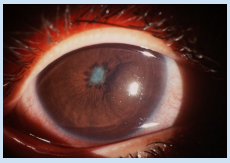
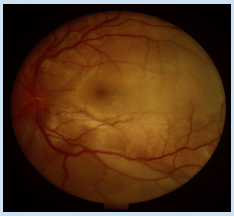
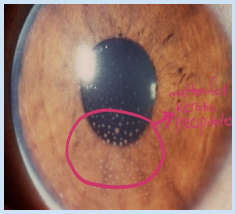
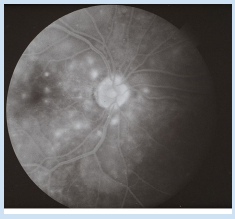
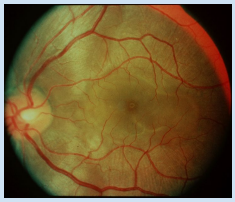
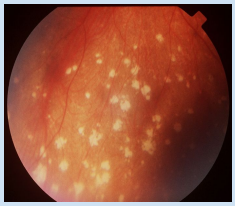
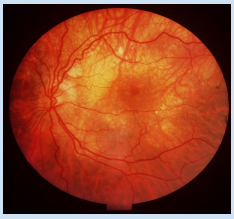
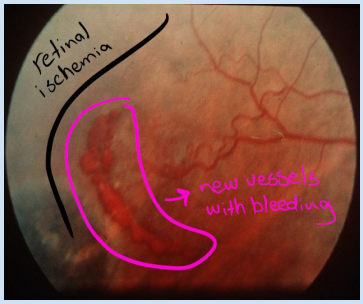
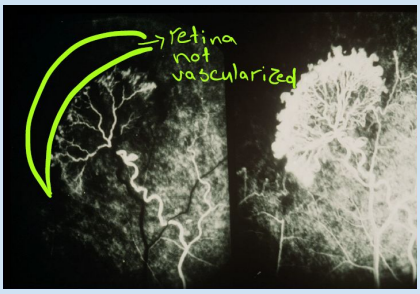
<tr><td colspan="2">  </td><td colspan="2">  </td><td>  </td><td>  </td></tr>
<tr><td colspan="2">White lashes</td><td colspan="2">Vitiligo</td><td>Posterior synechiae</td><td>Posterior synechiae and cataract</td></tr>
<tr><td>  </td><td>  </td><td>  </td><td>  </td><td>  </td><td>  </td></tr>
<tr><td>In the acute stage, the patient comes with exudative retinal detachment, because of inflammation within the choroid there is accumulation of the fluid under the retina. You can see the retina here is elevated</td><td>It can cause mutton-fat Keratic precipitates because it's a granulomatous disease</td><td>Starry sky appearance</td><td>Another example of exudative retinal detachment</td><td colspan="2">Right pic: Sunset glow fundus or orange fundes "if VKH is not treated" → it affects the retinal pigment layer</td></tr>
</table>

– **Ocular manifestation:** Bilateral granulomatous anterior uveitis, Bilateral multifocal posterior uveitis, Dalen–Fuchs nodules, 'sunset glow' fundus, Mutton - fat keratic precipitates, chronic manifestation, Acute phase manifested as inflammation of the choroid with exudative retinal detachment(accumulation of fluids under the retina)
– **Investigations:**
1. Associated HLA-DR1 and HLA-DR4
2. Lumbar puncture if diagnosis uncertain; CSF shows a transient lymphocytic pleocytosis, and melanin-containing macrophages.
– **Treatment:** High-dose steroid or infliximab in case of steroid resistance
– **Complications:** Glaucoma, Cataract, Choroidal neovascularization, Subretinal fibrosis, Retinal atrophy.

Sickle Cell Disease

- **Ocular features:**

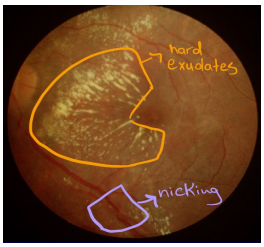
– Conjunctival comma-shaped capillaries
– Retinal changes: arterial occlusions, neovascular patterns, capillary closure
– Vitreous hemorrhage
– Sickle cell disease affects retina mainly and the sickling of the red blood cells will cause retinal ischemia (it will occlude peripheral retinal circulation causing retinal ischemia then you will have new vessels which can lead to bleeding looks like "Sea fans")
– Major complications of sickle cell disease: peripheral retinal ischemia- neovascularization- vitreous hemorrhage- traction retinal detachment
– To prevent bleeding you have to apply laser (scattered laser to the area of retinal ischemia)
– SCD retinopathy is differentiated from Diabetic retinopathy by the location of the new vessels, DR will be around the center, while SCD retinopathy in the periphery.

	Occlusion of peripheral retinal circulation causing ischemia. You can see new vessels with bleeding. To prevent bleeding you have to apply laser (scattered laser to the area of retinal ischemia)		Fluorescein angiography showing retina is not vascularized Picture on the left is early fluorescein angiogram shows massive ischemia, and on the right is delayed fluorescein angiogram which shows a big patch of complete filling of the new vessels with leakage around it(new vascular tufted filled with fluorescein)

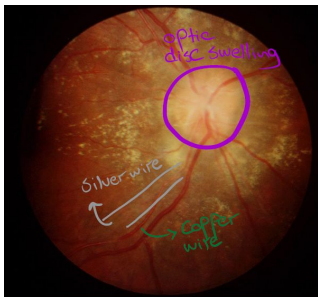
Hypertensive Retinopathy

- **Keith Wagener grouping:**
  - Stage I & II: arteriolar attenuation (silver wire and copper wire in the artery), increased light reflex
  - State III: Cotton wool spots, hard exudates, hemorrhages, macular star, retinal edema
  - Stage IV: all of the above +edema of the optic disc

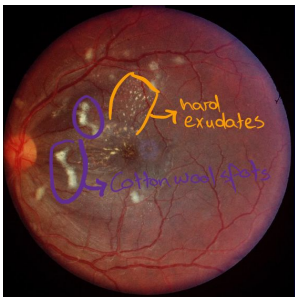
- **Ischemic choroidal infarcts (Elsching's spots):**
  - Retinal arterial macroaneurysm, ischemic optic neuropathy
  - As a compensatory phenomenon for high blood pressure, the first thing to happen is that the retinal arterioles become smaller (they attenuate) then the walls of arterioles become thicker so it will reflect more light this is will create what we call "copper wire and silver wire arteries". Then we see occlusion of retinal arterioles which appears as cotton wool spots and exudates then we can see hemorrhages as a result of severe hypertension and macular edema then the last stage we expect to see edema of the optic nerve head



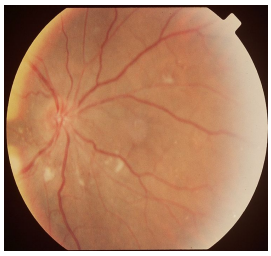
This is another good example of hypertensive retinopathy. Look to the hard exudates, but what is special about this hard exudates compared to diabetes? Here it's arranged **radially** and this is pathognomonic for hypertensive retinopathy you don't see it in other conditions.
You can see the artery with area of silver wiring and this is where the artery is crossing over the vein. There is a vein under the artery the vein becomes attenuated and this is called nicking "nipping" because the artery is becoming so thick and hard compressing on the vein under the artery causing this nipping
Here you can see the silver wire arteries, copper wires and radially arranged hard exudates



Hypertensive retinopathy. You can see optic nerve head swelling, look at the arteriole here you see the color here is white (this is what they call silver wire) and the rest is like copper wires (called copper wire)

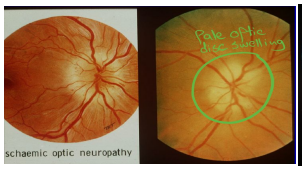


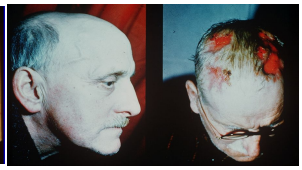
This is another young patient with pheochromocytoma. There are many cotton wool spots and you can see the radial distribution of hard exudates



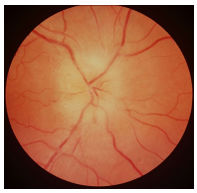
Giant Cell Arteritis

– Over 60 years, Females, smoking, low body mass index and early menopause | Large & medium sized vessels.
– Sudden visual loss due to anterior ischemic optic neuropathy profound unilateral visual loss.
– Amaurosis fugax which means recurrent attacks of loss of vision.
– Central retinal artery occlusion, cotton wool spots, anterior segment necrosis, oculomotor palsies (including a pupil-involving), cortical blindness.





You can see gangrene of scalp because of temporal arteritis
This is what happens: patient can present to the ER with blindness in one eye (no light perception). And when we look to the optic nerve we see white optic nerve and the margins are ill-defined "means it's swollen" and we call this **pale disc swelling** 'chalky white' edematous disc "characteristic feature" and this is a sign of ischemic optic neuropathy because the disease will cause occlusion of the small blood vessels that supply the optic nerve
Admission is required in such patients, because it's a life threatening disease, and this might be the first manifestation



This is another example. Patient presented to the ER and you can see white disc.
In this situation we always admit the patient , we need to confirm the diagnosis so we do ESR (high ESR) and we do temporal artery biopsy then if the diagnosis is confirmed you have to give the patient a large doses of systemic corticosteroids. Why? To protect the other eye because in the affected eye you cannot reverse blindness
We do an urgent temporal artery biopsy but if the patient has high ESR and C-reactive protein we can start systemic corticosteroids immediately

– **Non-arteritic anterior ischaemic optic neuropathy (NAION):** more common, caused by occlusion of the short posterior ciliary arteries resulting in partial or total infarction of the optic nerve head. Patient complains of sudden painless monocular visual loss; this is frequently discovered on awakening, suggesting a causative role for nocturnal hypotension.
– **Arteritic anterior ischaemic optic neuropathy (AAION):** Caused by giant cell arteritis (GCA). About 50% of patients with GCA have polymyalgia rheumatica (PMR)" pain and stiffness in proximal muscle groups, typically the shoulders and biceps, that is worse on waking , scalp tenderness and jaw claudication".

Ocular Emergencies & Red Eye

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

Objectives:

➢ Not given.

Done by: Monerah Alsalouli.
Resources: Slides + Notes + Lecture Notes of Ophthalmology + 435 Team + OphthoBook, Mayoclinic + Medscape + Master the boards.

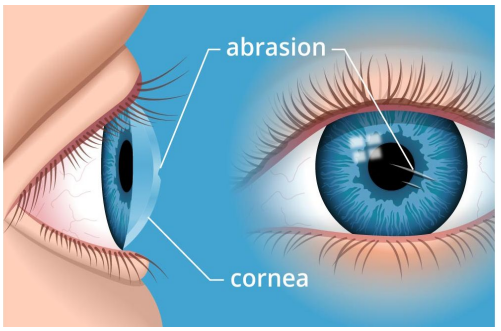
Don't freak out! This lecture is 2 lectures in one!

Ocular Emergencies

This lecture is so important (MCQs & future), you may face it yourself or one of your family members لا سمح الله.
Usually the outcome in emergency cases depend on immediate intervention (how did you manage the pt earlier), so despite the specialty you choose, you need to know these principles.

General Emergencies	Orbital/Ocular Trauma
Corneal abrasion **Corneal ulcer** **Uveitis** **Acute angle glaucoma** **Orbital cellulitis** **Endophthalmitis** **Retinal detachment** **Chemical injury**	**Corneal & conjunctival foreign bodies** **Hyphema** **Ruptured globe** **Orbital wall fracture** **Lid Laceration**

Corneal abrasion:



Corneal abrasions result from a disruption or loss of cells in the top layer of the cornea, called the corneal Epithelium. History of scratching the eye (fingernails or lenses). the epithelium has the ability to replicate.

Symptoms:

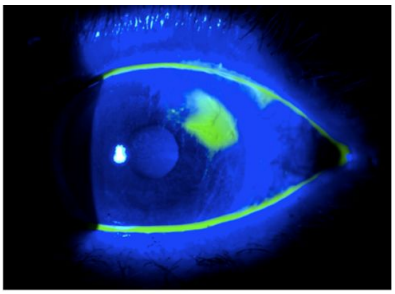
- Foreign body sensation.
- Severe Pain.
- Redness.
- Tearing.
- Photophobia experience of discomfort or pain to the eyes due to light exposure

"Corneal Abrasion can lead to Corneal Ulcer if untreated"

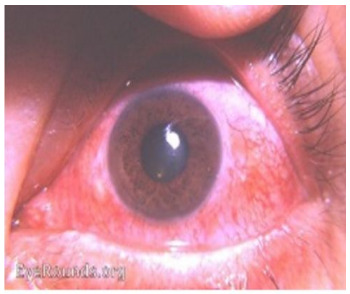
Treatment: it heals within 24 hrs.

Mostly will heal by itself but we fear of infections

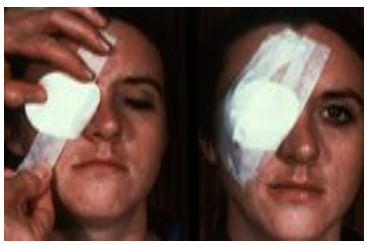
- Topical antibiotic "prophylactic to prevent infections"
- Pressure patch over the eye. احيانا ما يحتاج نغطيها
- Refer to ophthalmologist. See them everyday until it's gone
- Cycloplegia to dilate pupil to decrease pain.
- Important to treat to avoid infection.
- If infection is suspected do scraping biopsy to rule it out



SAQ: Fluorescein dye uptake
المنطقة الي تأخذ الصبغة هي المتأثرة



Corneal abrasion with NO opacities
القرنية صافية مو زي الالسر



Corneal ulcer: "microbial keratitis"

occurs secondary to disruption of the immune defence of the eye (lid and conjunctival inflammation) but is often due to trauma or contact lens wear. Can be due to Bacterial, viral, fungal or parasitic.

Symptoms:

- Ocular pain
- Redness
- Discharge
- Decreased vision
- **Corneal opacity** ± hypopyon "pus" white yellowish lesion on the cornea (ant. chamber)

Management:

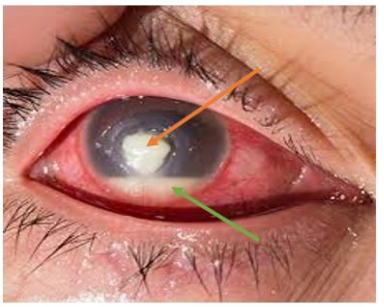
1. Prompt diagnosis of the etiology by doing corneal scraping (Slide, culture to diagnose)
2. Treatment with appropriate antimicrobial therapy are essential to minimize visual loss. كل ساعة
3. Then treat the inflammatory process.
4. Promote healing and treat the primary cause if present (e.g. lid deformity, dryness)

start by antibiotics, why ? because most common is bacterial, most serious (perforation) is bacterial, it takes long time to response if u treat as fungal.

- ➔ Gram **+ve:**
  - Mild to moderate: **Cefazolin**
  - Severe case: **Vancomycin**
- ➔ Gram **-ve:**
  - **Ceftazidime**

We give antibiotics every hour, why? because there is no immune system (no blood vessels)

Hypopyon is simply a pus collection in the anterior chamber.

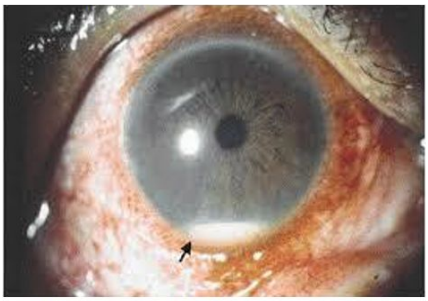


Corneal ulcer with Hypopyon

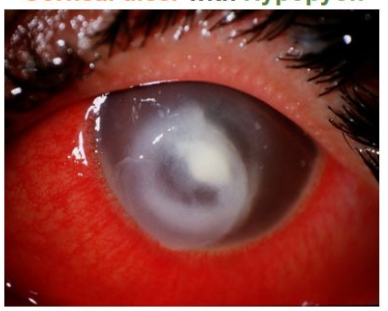
Contact lens wearer:

Any redness occurring for patients who wear contact lens should be managed with extreme caution.

1. Remove lens
2. Rule out corneal infection (i.e corneal ulcer)
3. Antibiotics for gram negative organisms (pseudomonas aeruginosa is the most common), treat it empirically as bacteria give **Ceftazidime**, if no response → antifungal, Because **Fungi** and **Acanthamoeba** are common causative organisms.
4. Do not patch
5. Follow up with ophthalmologist in 24 hours



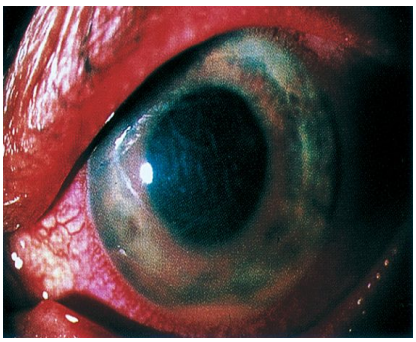
Hypopyon with No corneal ulcer



Bacterial keratitis with opacity

• Uveitis:

Inflammation of the uveal tissue **(iris, ciliary body, or choroid)**, however retina, blood vessels, optic disc, and vitreous can be involved. "the patient may have retinitis or hypopyon secondary to uveitis ".



It could be:

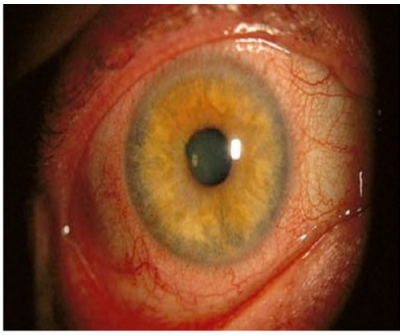
1- Anterior as iridoscleritis.
2- Posterior as choroiditis, retinitis.
3- Panuveitis (affect the anterior chamber, vitreous and retina and/or the choroid).

Etiology:

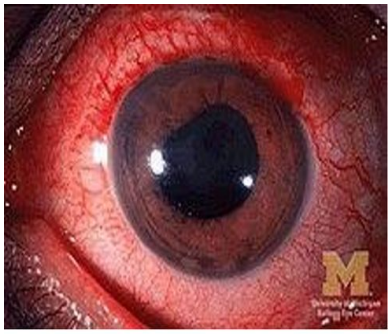
- Idiopathic 50%
- Inflammatory diseases:
  - HLA B27, Ankylosing spondylitis, IBD, Reiter's syndrome, Psoriatic arthritis.
  - Sarcoidosis (lung CT to diagnose), Behcet's, Vogt-Koyanagi-Harada Syndrome (panuveitis and ear involvement).
- Infectious:
  - Herpes virus.
  - Toxoplasmosis; transmitted through cats Poor outcome.
  - Secondary Tuberculosis; granulomatous uveitis (common in KSA, India) – give anti Tb and steroids Why? because you don't want the patient to have miliary TB.
  - Syphilis

Management:

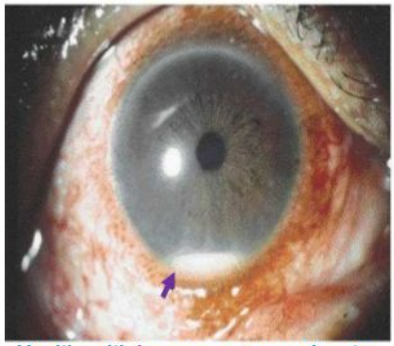
- Identify possible cause.
- Topical steroid "first".
- Topical cycloplegics to dilate pupils.
- Systemic immunosuppressive medication:
  "according to the workup,either shift to systemic or continue topical"
  - Steroid.
  - Cyclosporine.
  - Methotrexate.
  - Azathioprine.
  - Cyclophosphamide.
- Immunomodulating agents:
  - Infliximab (Anti TNF).



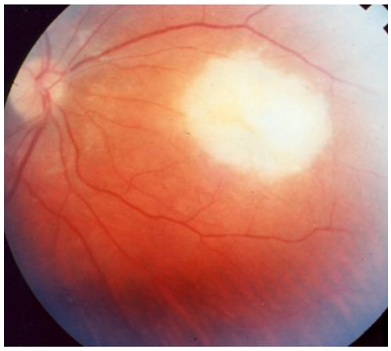
Ant. Uveitis with limbus inflammation



Uveitis



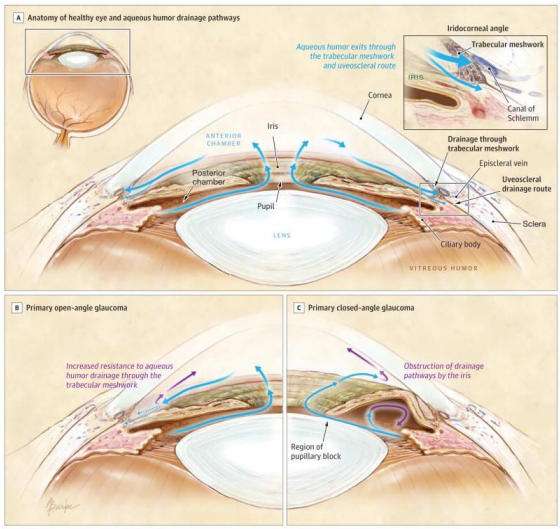
Uveitis with hypopyon secondary to Behçet disease



Patchy infiltration of uveitis secondary to toxoplasmosis

- **Acute angle glaucoma (موية زرقاء):**

Is caused by a rapid or sudden increase in intraocular pressure (IOP). High pressure inside the eye is caused by an imbalance in the production and drainage of fluid When the peripheral iris bunched up in the angel.
" Normal IOP 10 to 21 mmHg "



Result from peripheral iris blocking the outflow of fluid

Risk factor: more in hyperope pts since they have smaller eyes

symptoms:

- Pain
- Redness
- Mid-dilated pupil
- Decreased vision
- Colored halos around lights
- Severe headache or nausea and vomiting
- Increased Intraocular pressure

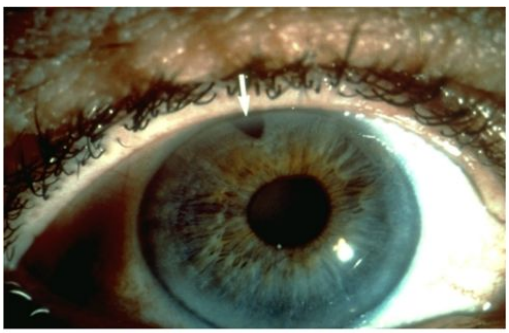
Typical history: symptoms increase while dimming the light. *Glaucoma increases at night more than morning because of **pupil dilation** at night.
e.g. patient came to the doctor complaining that he had eye pain whenever he watching a film and turn off the lights

Management:

- Medical treatment: is necessary to stabilize the eye and reduce the pressure before laser iridotomy(prevent prolapse of intraocular contents)"iv mannitol+ oral acetazolamide + topical beta blocker"
- Peripheral laser iridotomy will be curative in most cases. Aims to reduce the pressure and relieve the pain.

First reduce IOP by meds → then do iridotomy the ultimate treatment.
The prognosis depends on time of intervention. The earlier the better

Complications: Can cause severe visual loss due to optic nerve damage. The ultimate result



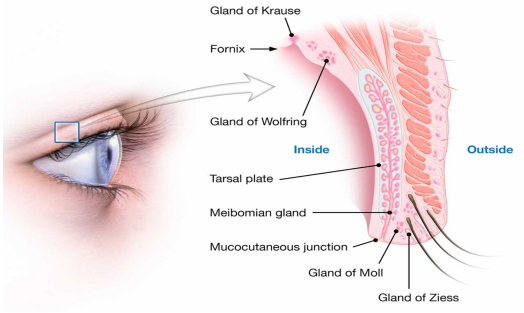
Post laser iridotomy

Halos around lights

Anatomy review:

Anatomy of upper eyelid: the septum is important as it separates the outside and inside the eye.

- Infection outside the septum is: **Preseptal cellulitis.**
- Infection inside the septum is: **Orbital cellulitis.**



• Preseptal cellulitis: *need to rule out orbital cellulitis.

also known as Periorbital cellulitis is an inflammation and infection of the eyelid and portions of skin around the eye, anterior to the orbital septum.

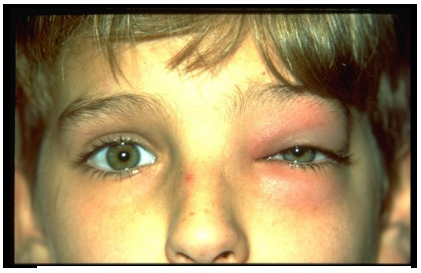
Symptoms:

- Lid swelling and erythema.
- **Normal** visual acuity, motility, pupils, and globe. Unlike Orbital cellulitis

Etiology:

- Skin wound
- Laceration
- Retained foreign body from trauma
- Vascular extension, or extension from sinuses (sinusitis) or another infectious site (e.g.,dacryocystitis, chalazion)
- Organisms:
  - Staph aureus most common
  - H.influenzae most common in < 5 yrs
  - Streptococci

Periorbital cellulitis



Periorbital cellulitis

Management:

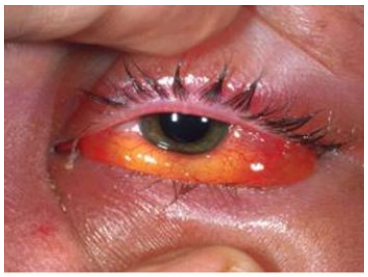
Need to be treated properly to avoid extension of the infection to the orbit which cause orbital cellulitis (inside).

- Warm compresses.(always warm for infections- we need vasodilation and subsequent increase in WBCs and chemotaxis)
- Systemic antibiotics "Augmentin" (no admission needed unless in case of a child)
- if not better or +ve history of trauma do → CT sinuses and orbit .

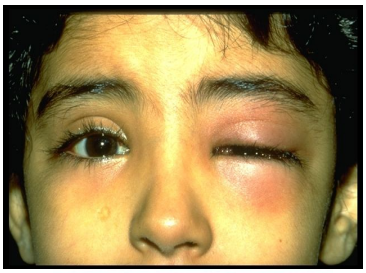
• Orbital cellulitis:

It most commonly refers to an acute spread of infection into the eye socket from either the adjacent sinuses or through the blood.

More serious than preseptal cellulitis because it may go to the brain and lead to death. Could be a consequence of preseptal cellulitis.



Orbital cellulitis

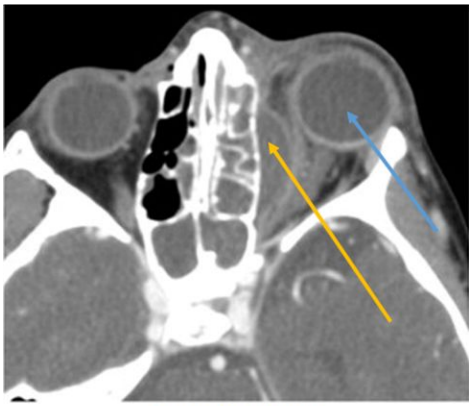


Orbital cellulitis

Symptoms: مو شرط كلهم

- Pain
- Decreased vision.
- Impaired ocular motility/double vision
- Afferent pupillary defect
- Conjunctival chemosis and injection Chemosis of the conjunctiva is a type of eye inflammation It occurs when the inner lining of the eyelids swells.
- Proptosis (bulging of the eye anteriorly out of the orbit)
- Optic nerve swelling on fundus exam

"Motility, pupil reaction, fundal exam, color vision need to be tested to check optic nerve function."



Orbital cellulitis
Collection of pus pushing the eye

Management:

- **Admission**.
- **Intravenous** antibiotics (vancomycin)
- Nasopharynx and blood cultures
- Surgery may be necessary

In case of subperiosteal abscess **Don't' drain immediately!** First give IV antibiotics until the inflammation subside, because you never know the abscess may go away spontaneously, if it doesn't go u need to drain it.
Complication: think of the brain: meningitis, abscess

• Endophthalmitis:

Endophthalmitis is the inflammation of the vitreous cavity, it's a EXTREME EMERGENCY as its a blinding disease that needs intervene within hours!
Potentially devastating complication of any intraocular surgery "Infection in vitreous cavity[1]"
Secondary to trauma or post-surgery (channel from outside to inside which cause bacterial entry and it found good environment to live in as there is no direct blood vessels in the vitreous to provide strong immunity) Sometimes the destruction is due to the inflammation not the infection itself.
Symptoms:

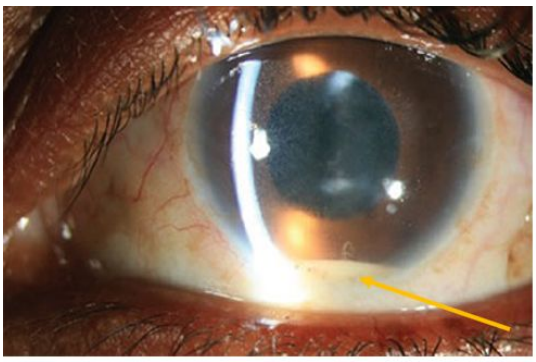
- Any **Early postoperative period** patient (within 6 weeks of surgery) complaining of PAIN or DECREASE VISION should be evaluated immediately

By looking at the eye it's sometimes difficult to differentiate between uveitis & endophthalmitis
what should we do??! **HISTORY!!!** Post surgery → Endophthalmitis
(e.g. patient Present 2-3 days post-op with Severe redness, lid edema and hypopyon on exam you find vitritis.
Management:

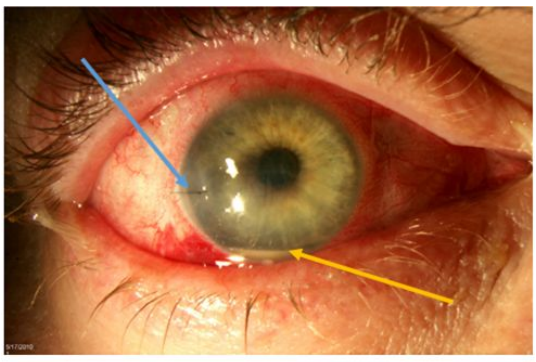
- Vitreous sample for culture.
- **Intravitreal** antibiotics injection plus topical antibiotics.

Broad spectrum antibiotics or **Vancomycin**

- In severe infection the vitreous will be like an abscess in this case surgery is needed to drain it (Vitrectomy).
- If visualization of vitreous is not possible in case of severe infection, do B scan (aka ultrasound)
- In decreased visual acuity (hand motion or less) Surgery is needed, if better give Intravitreal antibiotics only.
- Visual acuity will decide the treatment if Intravitreal antibiotics or surgery
- Do surgery – if no response to antibiotics and Endophthalmitis secondary to blebitis[2].



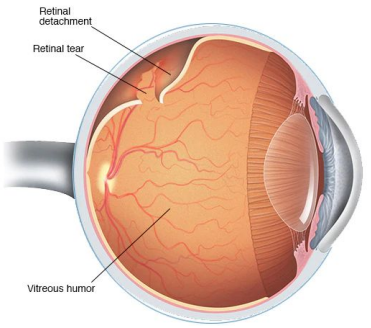
Endophthalmitis with hypopyon



Endophthalmitis (Note the sutured corneal wound and hypopyon)

[1] Other entity of endophthalmitis called "endogenous endophthalmitis, infection inside the body goes to the vitreous cavity
[2] Blebitis: is a presumed infection in or around a filtering bleb without vitreous involvement.

● Retinal detachment (انفصال الشبكية):



Separation between retinal pigment epithelium (RPE) and neurosensory layer. (neuronal layer from the pigmented layer). انفصال الطبقة الأخيرة عن التسعة اللي قبلها, not b/w retina and chroid..

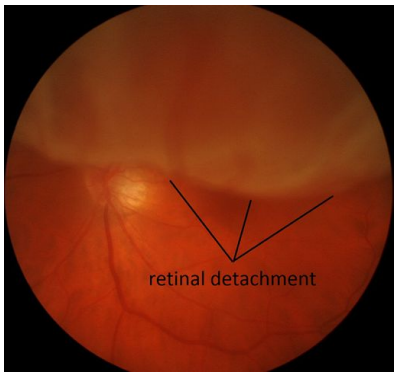
Retinoschisis: Separation between retina and choroid, no urgent intervention needed and usually congenital.

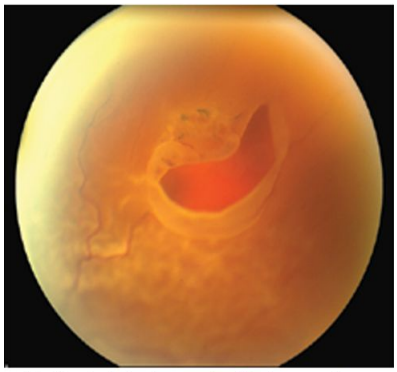
Rhegmatogenous retinal detachment: (emergency and need surgery) common in people with high myopia because they have peripheral breaks, fluid goes inside it and cause detachment.

Risk factors: people with high myopia -6 and above.

Symptoms:

- Flashes, floatersسر ابات تتحرك, a curtain or shadow moving over the field of vision.
- Peripheral and/ or central visual loss.
- History of scratching the eye
- Painless





Retinal detachment with horseshoe retinal tear

Management: Surgery Laser and vitrectomy.

The aim of the treatment is to close the causative break in the retina and to increase strength of the attachment between the surrounding retina and the RPE 'Retinal pigment epithelium' by inducing inflammation in that region.

- If involving the macula (Macula off) ⇒ poor prognosis and surgical intervention needed.
- In the periphery (Macula on) ⇒ better prognosis and can be treated by laser.

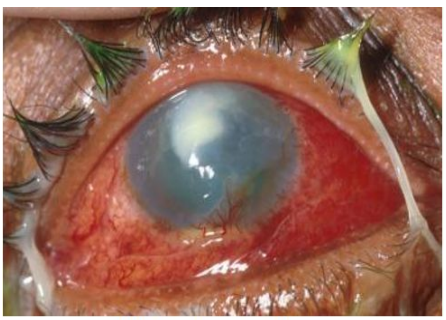
● Chemical injuries:

A vision-threatening emergency with a poor outcome. ليش نخاف منه اكثر شيء؟ cuz it could damage the stem cells

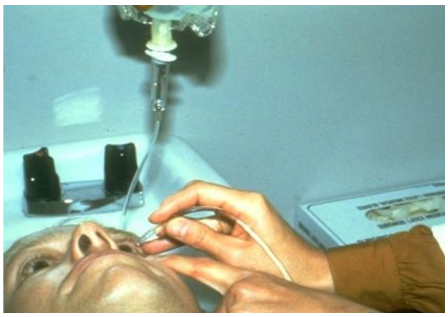
- The offending chemical may be in the form of a solid, liquid, powder, mist,vapor.
- Can occur in the home, most commonly from detergents, disinfectants, solvents, cosmetics, drain cleaners, **Alkaline** chemical injury is **worse** than acid more penetration.
- Can range in severity from mild irritation to complete destruction of the ocular surface.
- It may be aggressive and destroy eye surface "Epithelium" causing stem cell deficiency end up with blindness.
- Destruction of optic nerve common in case of glaucoma resulting from alkaline injury.

Management: poor prognosis

1. Immediate **irrigation** essential, preferably with saline or Ringer's lactate solution, for at least 30 minutes. *immediately before take history even*. اول شي نسويه
2. Irrigation should be continued until neutral pH is reached (i.e.,7.0)
3. Check for and remove foreign bodies in case of fireworks/Cement.
4. Instill topical antibiotic and anesthetic.
5. Frequent lubrications.
6. Oral pain medication.
7. Enhance healing

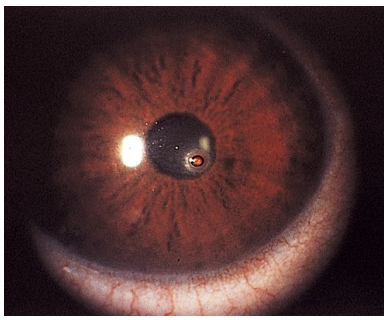


Alkali chemical burn



• Corneal & conjunctival foreign bodies:

- History of trauma
- Foreign body sensation
- Tearing

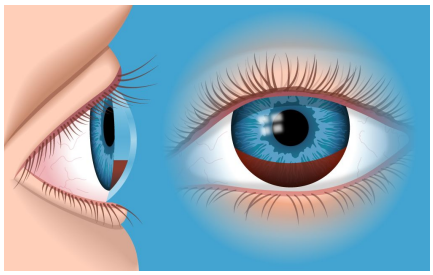


Management:

1. Instill topical anesthetic.
2. Remove foreign body.
3. Instill topical antibiotic.
4. Treat corneal abrasion.

*the foreign body could be hidden under the lid!

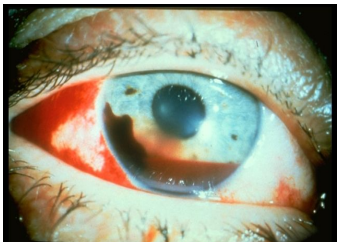
• Hyphema:



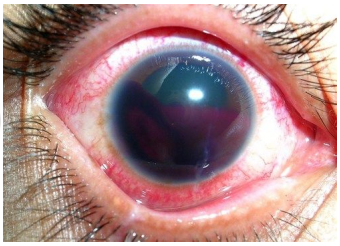
The presence of blood within the aqueous fluid of the anterior chamber.
Can occur with blunt or penetrating injury, in fact the most common cause of hyphema is "trauma".
doctors call it "8 Ball hyphema[3] " when it is filled with blood.

- Can lead to **high intraocular pressure.**
- Detailed history (Sickle cell[4]) to help in the treatment.

Management:



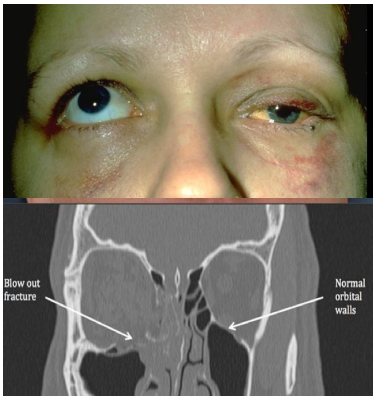
- Bed rest 2-3 days to prevent re-bleeding "no movement".
- Topical steroid to reduce inflammation
- Topical cycloplegic "Atropine" to fix the iris (pupil dilation) to prevent clotting & dislodging of the clot which cause re-bleeding.
- Anti-fibrinolysis agents (Tranexamic acid)
- Surgical evacuation "if no supentenouse resolve" usually we wait for 5 days, but in sicklers the time window is shorter we wait only for 2 days then we do surgery.



When do we take the pt to the OR?

- if increase IOP, stays more than five days and not responding to treatment.
- If total wait for 3 days if not responding and pressure more than 30 mmHg do Surgical evacuation,
- Not total wait for 5 days if pressure less than 30 mmHg.

• Orbital wall fracture:



An orbital fracture is a traumatic injury to the bone of the eye socket. These injuries are usually the result of blunt force trauma to the eye (*Blowout fracture*)
Assess ocular motility, sensation over cheek and lip
Palpate for bony abnormality (Enophthalmos; eye sinking inside)

When evaluating orbital fractures, focus on the following exam findings(from ophthobook):

1. **Vision, color:** Make sure the optic nerve isn't involved.
2. **Extraocular movements:** Usually decreased from swelling or muscle contusion, but make sure there isn't any gross muscle entrapment. If concerned, you can perform forced ductions. This involves pulling on the eye with forceps to see if the eye is mobile.
3. **Proptosis:** Measure the degree of proptosis or enophthalmos using the Hertel exophthalmometer (a fancy ruler).
4. **Palpate:** Feel along the orbital rim for step-off fractures and subcutaneous emphysema (air crepitus).
5. **Sensation:** Check sensation of the V1 and V2 sensation on the forehead and cheek. V2 runs along the orbital floor and can be damaged with floor fractures.

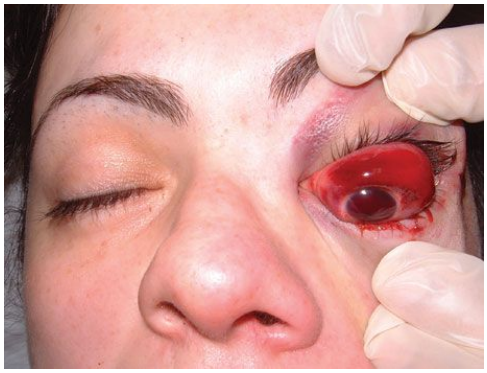
---

[3] "Eight-Ball" Hyphema. This hyphema completely fills the anterior chamber

[4] Moderate increase of IOP in sickle cell hemoglobinopathy patients may produced rapid deterioration of visual function, because sickling can lead to obstruction of the central retinal artery and profound irreversible visual loss.

- **Ruptured globe:** take the pt immediately to the OR

occurs when the integrity of the outer membranes of the eye is disrupted by blunt or penetrating trauma, the weakest part of the eye is insertion of the muscles and lamina cribrosa.

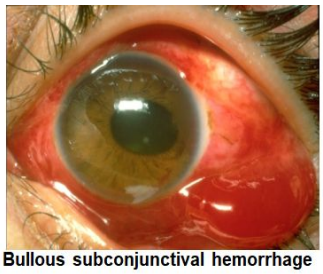


Etiology:

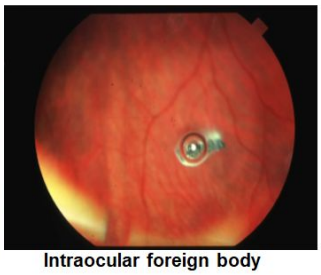
- Severe blunt trauma hit by a fist or tense ball
- Sharp object

Suspect a ruptured globe if:

- Bullous subconjunctival hemorrhage Take him to OR explore the area and suture if you leave it you'll still have infection.
- Uveal prolapse (Iris or ciliary body)
- Irregular pupil
- Hyphema
- Vitreous hemorrhage
- Lens opacity
- Lowered intraocular pressure
- Intraocular foreign body (if you have a foreign body first thing to do is take the pt t the OR to suture eye, then deal with the foreign body in another surgery you don't need to take it out immediately.)



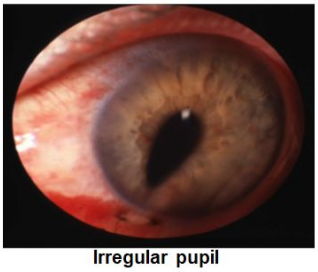
Bullous subconjunctival hemorrhage



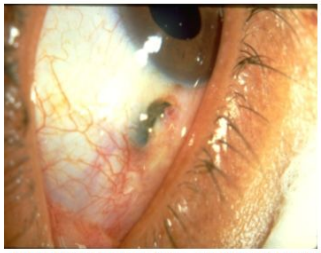
Intraocular foreign body

IF suspect a ruptured globe:

1. Stop examination.
2. Shield the eye.
3. Give tetanus prophylaxis.
4. Refer immediately to ophthalmologist.

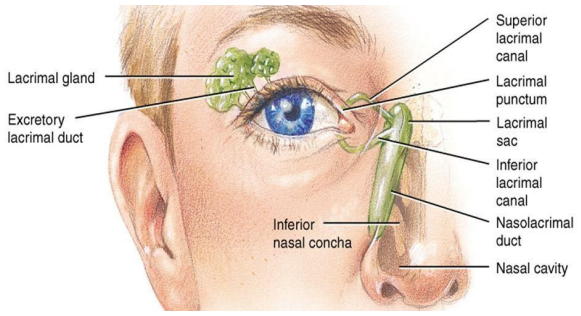


Irregular pupil



Uveal prolapse (Iris or ciliary body

- **Lid Laceration:**

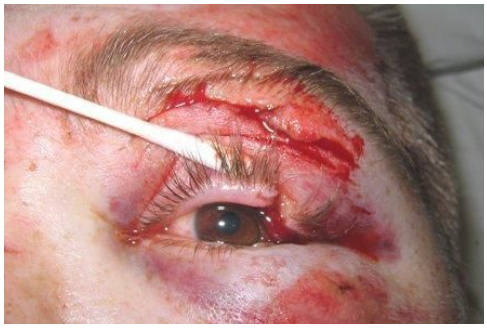


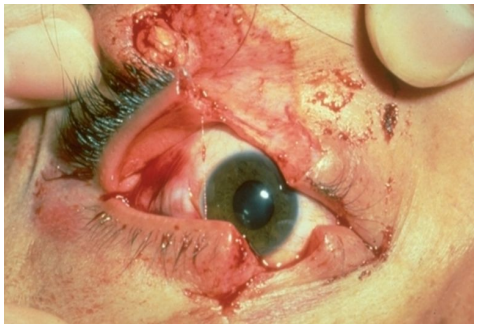
*it is not considered emergency unless it involves the canal

- Can result from sharp or blunt trauma
- Rule out associated ocular injury

غالبا العين ما راح تتأثر لكن الي يتأثر هي العضلات الي حواليها so we do surgery to realign the normal anatomy

Treatment: surgery (approximate the lids and close them following normal anatomy), If approximation is not following the normal anatomy: patient will have problems (the lids will be deformed, tearing won't be appropriate and the eye will be prone to infections).





Red Eye

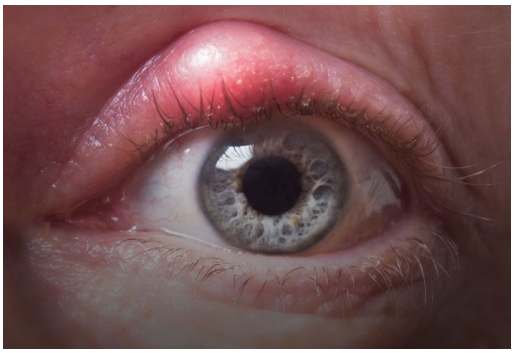
Differential diagnosis of RED eye

- ➔ **Conjunctival**
  - ◆ Blepharoconjunctivitis
  - ◆ Bacterial conjunctivitis
  - ◆ Viral conjunctivitis
  - ◆ Chlamydial conjunctivitis
  - ◆ Allergic conjunctivitis
  - ◆ Toxic/chemical reaction
  - ◆ Dry eye
  - ◆ Pinguecula/pterygium[5]
- ➔ **Lid diseases**
  - ◆ Chalazion[6]
  - ◆ Sty [7]
  - ◆ Abnormal lid function
- ➔ **Corneal disease**
  - ◆ Abrasion
  - ◆ Ulcer
- ➔ **Foreign body**
- ➔ **Dacryoadenitis**
- ➔ **Dacryocystitis**
- ➔ **Masquerade syndrome**
- ➔ **Carotid and dural fistula**
- ➔ **Acute angle glaucoma**
- ➔ **Episcleritis/scleritis**
- ➔ **Subconjunctival hemorrhage**
- ➔ **Factitious**

- **Blepharitis:**

(blef-uh-RYE-tis) is inflammation of the eyelids, usually involves the part of the eyelid where the eyelashes grow and affects both eyelids. Commonly occurs when tiny oil glands located near the base of the eyelashes become clogged.

Inflammation lid margin, Frequently associated with styes (Chronic inflammation of the lid margins), and Meibomian gland dysfunction, Adults > Children

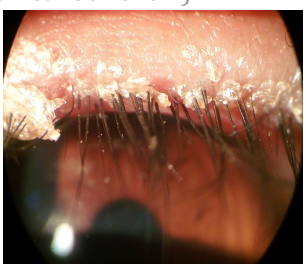


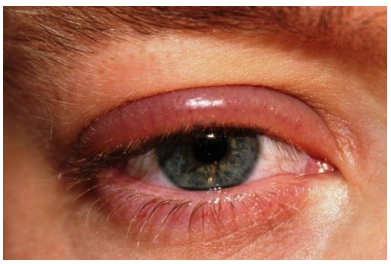
Symptoms & signs:

- Itchiness
- Soreness
- Worse symptoms in the morning
- Crusting of the lid margins in anterior blepharitis and redness in both (crust over eye lashes).
- Debris in the form of collarettes around the eyelashes (cylindrical dandruff).

Treatment:

- Lid hygiene (avoid mascara and eyeliner)
- Topical antibiotics
- Lubricants
- Warm compression





* these are the mainstay of the treatment will resolve if Not responding do surgery (eye incision and curettage)

[5] Pinguecula is a growth that looks like a yellow spot or bump on the conjunctiva. It often appears on the side of the eye near your nose. A pinguecula is a deposit of protein, fat, or calcium. Pterygium is a growth of fleshy tissue that may start as a pinguecula.

[6] A benign, painless bump or nodule inside the upper or lower eyelid. Chalazia (plural for chalazion) result from healed internal styes that no longer are infectious. These cyst-like nodules form around an oil gland (meibomian) within the eyelid, resulting in red, swollen eyelids.

[7] Also named hordeolum is an acute focal infection (usually staphylococcal) involving either the glands of Zeis (external hordeola, or styes) or, less frequently, the meibomian glands (internal hordeola).

Anterior blepharitis:
Inflammation of the lid margin is concentrated in the lash line and accompanied by squamous debris around the eyelashes.
The conjunctiva becomes injected. It is sometimes associated with a chronic staphylococcal infection. In severe disease the cornea is affected (blepharokeratitis). Small infiltrates or ulcers may form in the peripheral cornea (marginal keratitis) due to an immune complex response to staphylococcal exotoxins.

signs:

- Redness and scaling of the lid margins
- Some lash bases may be ulcerated – a sign of staphylococcal infection.
- Debris in the form of a collarette around the eyelashes (cylindrical dandruff). This may indicate an infestation of the lash roots by Demodex folliculorum .
- Reduction in the number of eyelashes.

Posterior blepharitis (or meibomian gland dysfunction)**:**
the meibomian glands are usually obstructed by squamous debris.

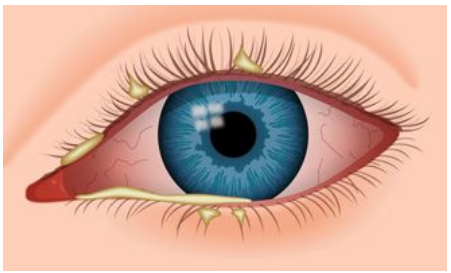
Signs:

- Obstruction and plugging of the meibomian orifices.
- Thickened, cloudy, expressed meibomian secretions.
- Injection of the lid margin and conjunctiva.
- Tear film abnormalities and punctate keratitis.

Both forms of blepharitis are strongly associated with seborrhoeic dermatitis, atopic eczema and acne rosacea. In rosacea there is hyperaemia and telangiectasia of the facial skin and a rhinophyma (a bulbous irregular swelling of the nose with hypertrophy of the sebaceous glands).

● Bacterial Conjunctivitis:

Both adults and children



Signs & symptoms:

- Redness
- Tearing
- Foreign body sensation
- Burning
- Stinging
- Photophobia
- Mucopurulent or purulent discharge
- Papillae
- lid and conjunctival edema.

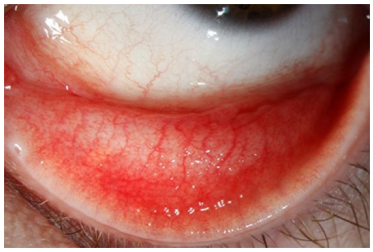
Organism: *Streptococcus pneumoniae, Staphylococcus aureus, Haemophilus influenzae, and epidermis.*

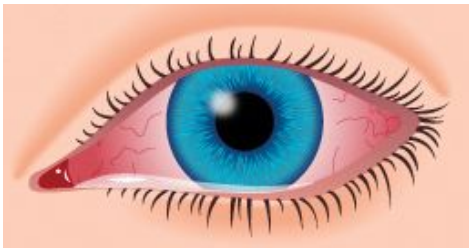
Management:

- Conjunctival swab for culture. ما ناخذ مسحه من اول مره الا بحالة طفل
- Topical broad spectrum "Fluoroquinolones:(ofloxacin, levofloxacin)"

● Viral Conjunctivitis:

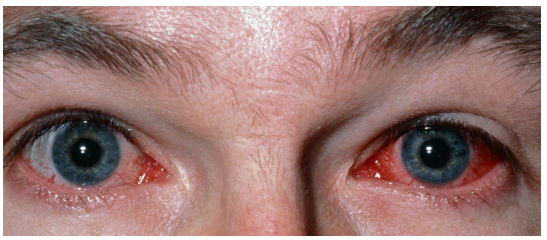
Hx of contact with someone with red eye.
"Contagious"





Signs & symptoms:

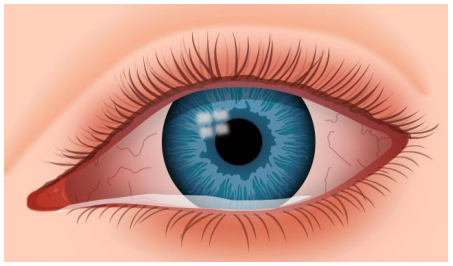
- Acute, watery red eye
- Soreness
- Foreign body sensation
- Photophobia
- Conjunctivitis is often intensely hyperaemic
- Follicles
- Haemorrhage
- Inflammation membranes
- Preauricular lymph node



Etiology: The most common cause is an adenoviral infection "URTI"
Treatment: no specific therapy but Cold compression are helpful

• Allergic Conjunctivitis:

Encompasses a spectrum of clinical condition "atopy", all associated with the hallmark symptom of **itching**
There is often a history of **rhinitis**, **sinusitis**, **asthma** and family history of **atopy**[8]

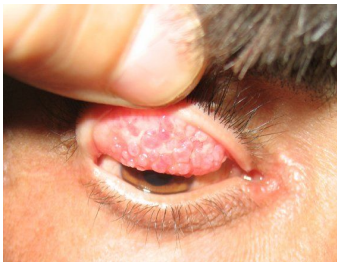


Signs & symptoms:

- Mild red eyes
- Itching
- Watery discharge
- Chemosis
- Papillary hypertrophy and giant papillae

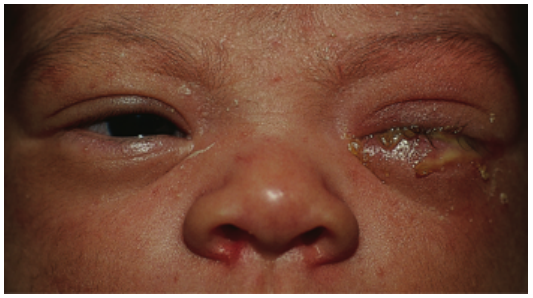
Treatment:

- Cold compress and eye rest
- Antihistamine
- Nonsteroidals
- Mast cells stabilizers
- Topical steroids (use it with caution to avoid reaching the steroid side effects)
  they'll love it and keep on using it resulting in glaucoma and cataract. (avoid it)



• Chlamydial Conjunctivitis:

Usually occur in sexually active individuals with or without an associated infection
Common in kids known as Ophthalmia neonatorum.



Signs & symptoms:

- Usually unilateral
- Tearing
- Foreign body sensation
- Lid crusting
- Mucopurulent discharge
- Follicles
- Non-tender preauricular node

Treatment: Oral tetracycline or azithromycin
If baby got infection during delivery, even the mother should be treated.

Now let's recap the differences b/w the types of conjunctivitis:

Bacterial conjunctivitis	Viral conjunctivitis	Allergic conjunctivitis	Chlamydial conjunctivitis
Mucopurulent discharge + Papillae	Watery discharge + Follicles + tender palpable Preauricular LN	Watery discharge + Papillae + nasal congestion + sneezing	Unilateral + Mucopurulent discharge + Follicles + Non tender palpable Preauricular LN

• Dry Eye:

Symptoms:

- Burning or foreign body sensation
- Trearing
- Usually bilateral

Etiology:

- Idiopathic
- Weather.
- Collagen vascular disease: Rheumatoid arthritis, sjogren syndrome
- Conjunctival scarring
- Infiltration of the lacrimal gland
- Vitamin A deficiency
- Drugs: Isotretinoin (Roaccutane) Excessive Vitamin A

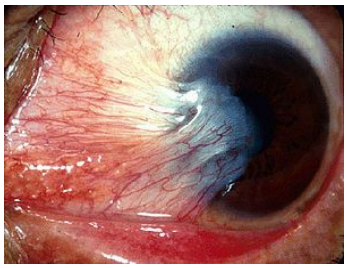
Treatment: Artificial drops

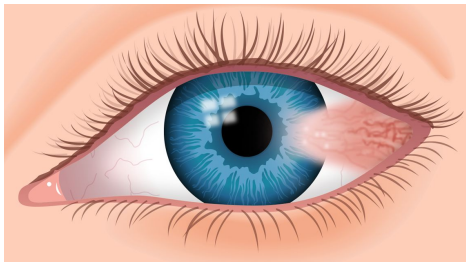
[8] Atopy refers to the genetic tendency to develop allergic diseases such as allergic rhinitis, asthma and atopic dermatitis (eczema)

• Pterygium لحمية العين:

Extension of conjunctiva, secondary to sun exposure prevention with sunglasses and lubricating with eye drops.

Fibrovascular membrane invading the cornea

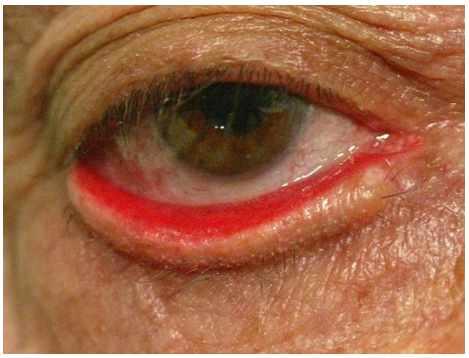




Indications of surgery:

- If affecting the central vision by involving central axis
- Suspicion of malignancy (squamous cell carcinoma)
- If it causes Astigmatism
- Cosmetic (if large)

• Lid Ectropion:



Ectropion is an eversion of the eyelid away from the globe.

causes:

- age - related orbicularis muscle laxity.
- Scarring of the periorbital skin.
- Seventh nerve palsy.

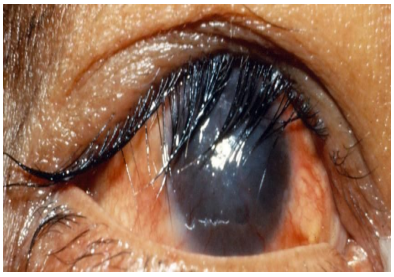
Symptoms:

- Dryness
- Redness
- excessive tearing

Treatment: Surgery by suturing the eyelid (Blepharoplasty)

The malposition of the lids everts the puncta and prevents drainage of the tears, leading to epiphora. It also exposes the conjunctiva and lower globe to dehydration . Ectropion causes an irritable eye. Surgical treatment is an effective treatment.

• Trichiasis:



Eyelashes growing toward the eye (Secondary to trachoma[9])

It is distinct from entropion (all lid inside يلف كل الجفن على جوا).

The lashes rub against the cornea and can cause irritation > abrasion > Ulcer.

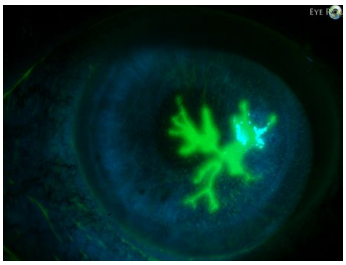
Treatment: adjust it by electrolysis

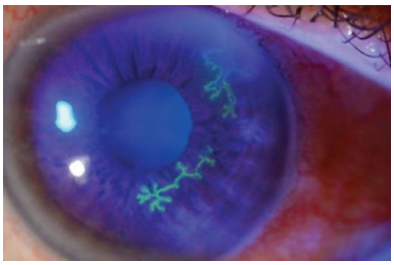
Surgery is needed in case of **Entropion**

• Infectious Keratitis: AKA corneal ulcer

Keratitis is infection of the cornea that can lead to corneal ulcer.

• HSV Dendrites (Herpes Keratitis):





Symptomes:

- Very red
- Swollen
- Painful

Management:

- **Fluorescein** staining to confirm (**dendritic pattern** seen on exam)
- Antiviral "Acyclovir"

*__NEVER__ **use steroids** for herpes keratitis, it makes the condition worse.

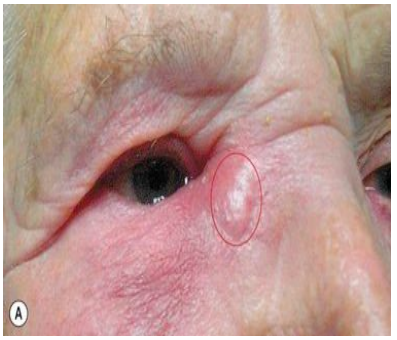
[9] Trachoma is an chlamydia trachomatis infection causes a roughening of the inner surface of the eyelids.

• Nasolacrimal Obstruction:

Can lead to Dacryocystitis:

Closed obstruction of the drainage system predisposes to infection of the sac (Dacryocystitis)

*if the lacrimal GLAND get affected we call it: Dacryoadenitis



Symptomes:

- Pain
- Redness
- Swelling over the innermost aspect of the lower eyelid
- Tearing
- Discharge

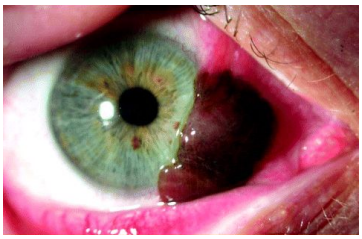
Organisms: *Staph aureus, streptococcus,* and *diphtheroids*

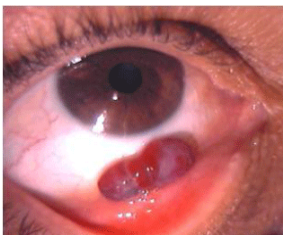
Treatment:

1. Systemic antibiotic until infection subside
2. Surgical drainage to open the lacrimal sac "Dacryorhinostomy"

lacrimal obstruction > open surgically.

Dacryocystitis > Antibiotic then later open surgically.





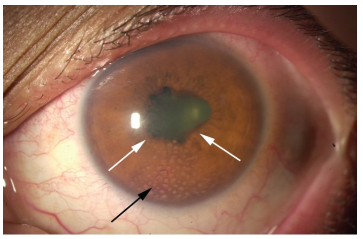
• Conjunctival Tumor:

Melanoma and can be associated with Masquerade syndrome.

• Iritis:

Associated with TB

WBC attached to the back of the cornea called **"Keratic precipitates"** النقط البيض



• Episcleritis:

This inflammation of the superficial layer of the sclera

Symptoms:

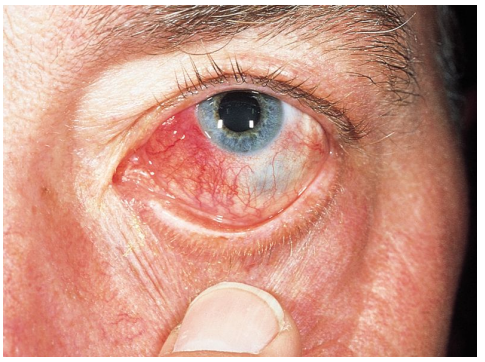
- Localized(sectorial) or diffuse redness
- Asymptomatic or mild pain/discomfort
- Usually self-limited.

Etiology: Mostly idiopathic, but Sometimes associated with gout (if so check uric acid)

Treatment: Topical or systemic NSAIDs.

• Scleritis:

This is a more severe condition than episcleritis, and may be associated with the collagen vascular diseases, most commonly rheumatoid arthritis, but also polyarteritis nodosa and SLE. It is a cause of intense ocular pain. Both inflammatory areas and ischaemic areas of the sclera may occur.



Symptoms:

- Severe Pain and tenderness (unlike episcleritis)
- Tearing
- Photophobia
- Localized or diffuse redness and swelling
- Nodules

Etiology: 30 to 60% associated with systemic disease e.g. RA

Complications: Can result in scleral necrosis (scleromalacia perforance)

scleral thinning, sometimes with perforation, keratitis, and uveitis.

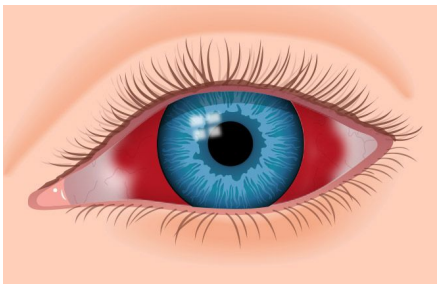
Treatment: Steroids (may need **systemic**)

Test: phenylephrine > cause vasoconstriction:

bleached > conjunctivitis.

not > scleritis.

- **Subconjunctival Hemorrhage:**



Symptoms:
- Usually asymptomatic
- Blood underneath the conjunctiva, often in a sector of the eye

Etiology:
- Idiopathic
- Valsalva (coughing or straining)
- Traumatic
- Hypertension
- Bleeding disorder

Treatment: mostly it's self limiting, but most importantly is to identify the cause, it could be the earliest sign of leukemia **(No need for treatment it is caused by trauma but if you suspect lymphoma, leukemia do CBC)**

Red Eye Treatment Algorithm

History
- Trauma
- Contact lens wearer
- Severe pain/photophobia
- Significant vision changes
- History of prior ocular disease

Exam
- Abnormal pupil
- Ocular tenderness
- White corneal opacity
- Increased intraocular pressure

YES ??! ➡ refer urgently to ophthalmologist

Is it Conjunctivitis?

History:
- Itching? → Allergy, Viral
- Exposure to person with red eye → Viral
- URTI → Viral
- Past history of conjunctivitis → Allergy
- Discharge with morning crust → Allergy, chlamydia
- Exposure to drug → Allergy

Signs:
- Discharge → Bacterial, chlamydia(depends on the nature of discharge)
- Lid and conjunctival edema → Bacterial
- Conjunctival redness
- Preauricular lymph node → Viral
- Facial or eyelid vesicles→ HSV

Summary from 435 team
Hyphema is defined as the presence of blood within the aqueous fluid of the anterior chamber. The most common cause of hyphema is "trauma". it Can lead to high intraocular pressure and usually treated with-Bed rest , Topical steroid and Topical cycloplegic . **Corneal abrasions** result from a disruption or loss of cells in the top layer of the cornea, called the corneal epithelium. the patient come with foreign body sensation, photophobia and severe pain. **Alkaline chemical injury** to the eyes is worse because it'll penetrate. Immediate irrigation is very important as management . **Inflammation of the uveal tissue** (iris, ciliary body, or choroid), retina, blood vessels, optic disc, and vitreous can be involved. and we treat it first with topical steroids . **Orbital cellulitis** More serious than preseptal cellulitis because it may go to the brain and lead to death. May be a consequence of preseptal cellulitis. **Endophthalmitis:** (extreme ophthalmic emergency) treated with Intravitreal antibiotics injection plus topical antibiotics.

Viral conjunctivitis	Bacterial conjunctivitis
Bilateral	Unilateral
Watery discharge	purulent , thick discharge
Easily transmissible	Poorly transmissible
Normal vision	Normal vision
Itchy	Not itchy
Preauricular adenopathy	No adenopathy
No specific treatment	Topical antibiotic

	Conjunctivitis	Uveitis	Glaucoma	Abrasion
Presentation	Itchy eye, dicharge	Autoimmune disease	Pain	Trauma
Eye finding	Normal pupils	Photophobia	Fixed midpoint pupil	Feels like sand in my eye
Most accurate test	Clinical diagnosis	Slit lamp examination	Tonometry	Fluorescein stain
Best initial therapy	Topical antibiotics	Topical steroids	Acetazolamide, mannitol, pilocarpine, laser trabeculoplasty	No specific therapy; patch not clearly beneficial

MCQs

30 years old girl came to uveitis clinic complaining of pain and fat keratic precipitate. -redness. Examination showed mutton What is the most common diagnosis? TB

Which conjunctivitis is least likely to occur bilaterally?

A. allergic
B. Viral
C. bacterial
D. vernal

The correct answer is (c) bacterial. Allergies are likely to affect both eyes and present with itching and watering. Vernal is a type of seasonal allergy you see in young boys. Viral conjunctivitis usually starts in one eye, but hops to the other eye as it is very contagious. Bacterial conjunctivitis can occur bilaterally, but of the available choices is most likely to occur in just one eye

A young 23-year-old black man presents with a hyphema in the right eye after blunt injury. All of the following are acceptable initial treatments except?

A. sleep with the head elevated
B. prednisolone steroid eye drops
C. cyclopentolate dilating drops
D. carbonic anhydrase inhibitor pressure drops

The correct answer is (d). For patients with hyphema (blood in the eye) advise them to avoid straining and sleep with their heads elevated to allow the blood to settle. Use steroids to decrease the inflammation and a medium-acting cycloplegic to dilate the eye for comfort and to keep the inflamed iris from "sticking" to the underlying lens. If the pressure is high, you can use pressure drops, but we avoid CAIs in African Americans as it induces RBC sickling in sickle-cell patients. You can get a sickle prep if you are suspicious for this disease.

A 7-year-old boy presents with a grossly swollen eyelid. His mother can't think of anything that set this off. What finding is most characteristic of a dangerous orbital cellulitis?

A. chemosis
B. warmth and erythema of the eyelid
C. physically taut-feeling eyelid
D. proptosis

The correct answer is (d). With any eyelid cellulitis, you must determine if the infection is pre-septal or post-septal (i.e., orbital cellulitis). While chemosis is certainly seen with orbital infection, a proptotic bulging eye is even more indicative of orbital infection. Other signs include decreased eye-movement, pain with eyemovement, and decreased vision

What location for a retinal detachment would be most amenable to treatment by pneumatic retinopexy?

A. inferior rhegamatogenous detachment
B. superior tractional retinal detachment
C. superior rhegamatogenous detachment
D. traumautic macular hole

This question covers several concepts. Rhegamatogenous detachments are the classic detachment occuring from a break in the retina. A pneumatic retinopexy is the technique of injecting a gas bubble into the eye that floats and tamponades the break. Gas bubbles require careful head-positioning and work best for superior breaks (patients can't stand on their heads for weeks for inferior breaks). The correct answer is therefore (c).

A 27-year-old contact lens wearer presents to the ER complaining of ocular irritation. On exam he has a small 2mm corneal abrasion. You should

A. treat with erythromycin ointment
B. treat with ciprofloxacin drops
C. bandage contact lens for comfort and speed reepithelialization
D. patch the eye and follow-up in 72 hours

You need to be concerned for pseudomonas infection in any contact lens wearer. Erythromycin is great stuff, but these higher risk patients should get something stronger like a fluoroquinolone (cipro). A bandage contact lens can help with painful abrasions, but I'd avoid one in this patient as the abrasion isn't big, and you typically don't patch ulcers. Patching can also be used to help with lubrication and comfort, but I never patch a potential infection, as bacteria like to grow in dark warm environments. If you decide to patch, you need to see your patient daily to make sure nothing is brewing under that patch. The most appropriate answer is (b).

10. A woman presents to you complaining of a red, watering eye for the past two days with stinging and some photophobia. Her vision has dropped slightly to 20/30. She has a history of diabetes and is taking drops for glaucoma, but is otherwise healthy. The most likely cause of her redness is:

A. angle-closure glaucoma
B. viral conjunctivitis
C. diabetic retinopathy
D. papilledema

This woman probably has a history of POAG (primary open angle glaucoma) if she is on drops. If she were to have an acute angle closure, her eye would be very painful and the vision would have gotten much worse from corneal edema. Diabetic retinopathy is usually a background finding of leaky vessels in the retina and doesn't create this picture. She merits a full eye exam, but her symptoms are consistent with "pink eye," with viral conjunctivitis being the most common cause in an adult. The correct answer is therefore (b).

A patient presents after MVA with a fracture of the orbital floor. What would be the indication for surgery in the near future?

A. double vision that worsens with upgaze
B. chemosis and moderate proptosis
C. restricted forced ductions
D. decreased extraocular movement

Floor fractures are very common and these patients always look impressively bad on exam, with marked swelling and subconjunctival bleeding. They can have decreased EOMs and proptosis from this swelling alone, which shouldn't concern you. More worrisome is entrapment of the inferior rectus muscle in the orbital floor – this entrapment can only be determined by forced ductions … grab the limbus with forceps and tug on the eye to see if movement is restricted. The correct answer is (c).

Which orbital bone is most likely to fracture with blunt trauma to the eye?

A. zygomatic
B. maxillary
C. ethmoid
D. sphenoid

The orbital floor, which is formed by the maxillary bone, is the most commonly fractured wall of the orbit. Orbital fat will commonly herniate through this bone and muscle can get stuck if the break acts like a trapdoor. The ethmoidal lamina papyracea is also often broken because it is the thinnest, but this occurs less often because of extensive bolstering. The lateral zygomatic component of the orbit is rarely broken, nor the more posterior sphenoid. The correct answer is (b)

A 32-year-old white man with a history of type-1 diabetes presents to you complaining of decreased vision. He has not seen an eye doctor in years. On exam, you find numerous dot-blot hemorrhages, hard exudates, and several areas of abnormal vasculature in the retina. Pan-retinal photocoagulation might be done in this patient to:

A. kill ischemic retina
B. tamponade retinal tears
C. ablate peripheral blood vessels
D. seal off leaking blood vessels

PRP is performed to kill areas of peripheral ischemic retina. By doing so, less VEGF is produced, leading to cessation and regression of neovascularization. While it is true that we sacrifice some of the peripheral retina with PRP, it is worth it to save important central vision. Lasers can be used to help peg down retinal tears and to help with leaking vessels … but this is called "focal laser therapy." The correct answer here is (a).

Which of the following is a risk factor for retinal detachment?

A. black race
B. male sex
C. presbyopia
D. myopia

The correct answer is (d) myopia. Myopic (near-sighted) eyes are large eyes with a stretched-out retina that is more likely to tear at the periphery. Neither blacks nor males are at higher risk of RD. Presbyopic lens hardening occurs with age and doesn't have anything to do with the retina

A 57-year-old man complains of flashing lights and a shade of darkness over the inferior nasal quadrant in one eye. On exam you find the pressure a little lower on the affected eye and a questionable Schaffer's sign. What condition would lead you to immediate treatment/surgery?

A. macula-off rhegmatogenous retinal detachment
B. epi-retinal membrane involving the macula
C. dense vitreous hemorrhage in the inferior nasal quadrant
D. mid-peripheral horseshoe tear with sub-retinal fluid

Schaffer's sign is when you see pigment behind the lens on slit-lamp exam, and occurs when a tear of the retina allows the underlying pigment to release into the vitreous chamber. A macula-off retinal detachment is unfortunate, but isn't an immediate emergency. It certainly needs to be repaired, but can wait for a few days if necessary, as the damage to the detached macular photoreceptors has already occurred. Epi-retinal membranes are common and aren't an emergency unless actively creating a tractional detachment. Vitreous hemorrhage is not an emergency either, assuming there isn't a detachment behind that blood on your ultrasound. Smaller retinal tears, however, need to be treated early to make sure they don't progress and peel off the macula. The answer is (d).

Oral doxycycline helps blepharitis patients by:

A. antibiotic tear secretion
B. changing lipid viscosity
C. inhibiting cytokine release
D. improved lacrimal gland excretion

Doxycycline changes the lipid viscosity of the meibomian gland secretions, improving oil secretion from the gland into the tear film. This superficial lipid layer is needed to keep the tears from evaporating too quickly. The correct answer is (b).

A man calls the office complaining of eye pain after splashing bleach in his eye. You should instruct him to:

A. patch the eye and immediately go to the office
B. irrigate the eye for 15 minutes and then go to the office
C. immediately apply lubricating ointment and then go to the office
D. immediately wash the eye with contact saline solution and go to the office if he notices any change in vision

The final visual outcome for a chemical burn is going to depend upon how quickly the chemical is washed out of the eye, so have your patient wash out their eye immediately! Chemical injury is one of the few eye problems that you treat prior to checking vision. The correct answer here is (b).

What antibiotic would you use in a newborn with suspected chlamydial conjunctivitis?

A. Ciprofloxacin drops
B. Erythromycin drops
C. Oral Doxycyline
D. Erythromycin drops and oral erythromycin

Chlamydia is one cause of conjunctivitis you should suspect in the newborn. Treatment involves topical drops such as erythromycin and systemic coverage because of concurrent respiratory infections these kids can develop (chlamydia infects mucous membranes and can cause pneumonitis). You don't use doxy in children (especially under the age of eight). Fluoroquinolones might work, but we don't use them in children because of theoretical bone suppression. The correct answer is (d).

OphthoBook

1. You have a contact lens wearer with a small corneal abrasion. He is in excruciating pain and requests that you pressure-patch his eye for comfort. Will this speed up healing?
Patching may speed healing by keeping the eye immobile and lubricated but you should never patch an abrasion that might fester an infection. Thus, you don't patch contact lens wearers as you don't want a pseudomonas infection brewing under that patch! If you decide to patch a patient, you should really follow them closely to make sure they don't develop an ulcer.

2. What's the easiest way to see a corneal abrasion? How often do you need to follow simple, non-infected abrasions?
Abrasions are easiest seen with fluorescein under the slit-lamp microscope, though large abrasions can be detected with only a handlight as the edges of the abrasion create a circular shadow on the iris underneath. You'll want to measure the epithelial defect and see the patient often (sometimes daily), until it heals to make sure they don't become infected.

3. What is the Seidel test?
This is a method to see if a laceration has penetrated completely through the cornea. Basically, you're using fluorescein to look for leaking aqueous fluid.

4. What findings would prompt you to take a patient with an orbital floor fracture to surgery?
If the patient has muscle entrapment or significant enophthalmos. Most patients have some degree of EOM restriction from soft-tissue swelling. Entrapment causing reflexive bradycardia would also push you toward surgery.

5. What portion of the eyelid do you worry about with lid lacerations?
If the laceration is medial (near the nose) it could involve the tear drainage pathway. These canalicular tears are more complicated to repair.

6. A patient accidentally splashes a large amount of bleach-based cleaner in her eye. What should she do?
Wash it out immediately - the faster, the better!!!! If an ambulance picks her up, have the EMTs irrigate in route, and alert the ER to irrigate her eyes as soon as she hits the door.

7. What is the best way to test the pressure in an eye with a likely open globe injury: with slit-lamp applanation or with the hand-held tonopen?
If you suspect open globe, you don't want to be mashing on the eye, so neither of these is correct. This is a trick question … hahahahaha! Seriously, though, don't push on the eye.

8. How often should a patient with a hyphema be seen and why?
These patients need to be seen almost daily for the first week to check for pressure. This is especially important on post-trauma days 3 – 5 as this is when clots begin to retract and rebleed.

9. An African American presents with hyphema after trauma. What additional workup might you consider? Are there any medications you would avoid?
You may consider getting basic coagulation labs and a sickle prep. Avoid CAIs as these promote acidosis and can worsen sickling of blood in the anterior chamber and worsen glaucoma.

Ocular Pharmacology & Toxicology

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

Objectives:

➢ Not given.

Done by: Khawla AlAmmari, Ola AlNuhayer.
Revised by: Rawan Aldhuwayhi, Munerah alOmari.
Resources: Slides + Notes + 435 Team (F2)

Summary LINK

(done by Reema Alnasser from last year)

Key Points LINK

(في حال قررتوا تسحبون عالمحاضرة مروا عليه اضعف الإيمان*^)

General Pharmacological Principles

Pharmacodynamics:

- **Mechanism of action:** it's the biological and therapeutic effect of the drug.
- Most drugs act by binding to regulatory macromolecule, usually neurotransmitters or enzymes or hormone receptors.
- If the drug is working at the **receptor level**, it can be **agonist or antagonist.**
- If the drug is working at the **enzyme level**, it can be **activator or inhibitor.**

Pharmacodynamics:

It is the absorption, distribution, metabolism, and excretion of the drug : how the drug reach particular area and how it will be execrated. A drug can be delivered to ocular tissue as:

Loca-lly

1/ Eye Drops

We prefer local on systemic b/c of more effect on target tissue and less side effects

- Most commonly used, best way, can use it during day time.

زبده الكلام التي تحت انه قاعد يقول لك لاتحسبين كل مازدتي كميه القطرات كلما زاد الامتصاص لا, العين لها سعه امتصاصيه معينه 7-10 µl مهما زدتي عنها كميه القطرات بعدها مرح تمتص.

- one drop = 50 µl, more than third of the drug will wash out, so 1 drop is more than enough.
- volume of conjunctival cul-de-sac(the fornix of conjunctiva that act as reservoir of drug) **7-10 µl**
- Measures to increase drop absorption, so increase effect: ايش نسوي عشان نزيد السعه الامتصاصيه (نبهوا عليها اهاليكم الي ياخذون عده قطرات بنفس الوقت)
  - Wait 5-10 minutes between drops
  - Compress lacrimal sac, that will decrease systemic effect.
  - Keep lids closed for 5 minutes after instillation(blinking sucks the fluid from the ocular surface and drain it to the nasal cavity that's why we till the patient to close his eyes) increase local effect and decrease systemic effect

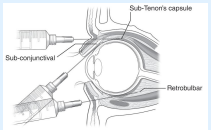
Once you open the bottle, if it preserved like in fridge you can use it till expiry date , it it outside the fridge then you can use for 1 month only

2/ Ointments

- Increase the contact time of ocular medication to ocular surface thus better effect. More viscous = stays for longer time
- It has the disadvantage of **vision blurring** (Thick! advise pt to put it before going to sleep)
- The drug has to be high lipid soluble with some water solubility to have the maximum effect as ointment.

Note: eye drops and ointments are more likely to affect anterior segment of the eye (cornea , conjunctiva, anterior chamber , the iris, lens and posterior chamber) but not any further, so if posterior segment of the eye is affected we need to use injection around the eye or directly to the eye.

3/ Peri-ocular injection



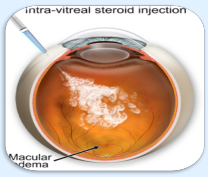
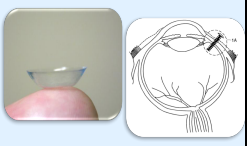
- Reach behind iris-lens diaphragm better than topical application.
- e.g. subconjunctival, subtenon (capsule surround the sclera and behind conjunctiva), peribulbar (around the globe → extraconal), or retrobulbar (behind the globe → intraconal)
- This route bypass the conjunctival and corneal epithelium: good for drugs with low lipid solubility (e.g. penicillins).
- Also steroid (reduce inflammation) and **local anesthetics*** can be applied this way
- Use short needle or you will puncture the glop.

***Notes about Local anesthesia for eye operation:**

1/ Topical eye drops → It numbs the surface (Anesthesia) but it doesn't block cranial nerves → the eye can move (no Akinesia) "this is good for cooperative patients". In uncooperative patients we need Anesthesia with Akinesia → retrobulbar or peribulbar block

2/ Retrobulbar (Intraconal): depositing local anesthetic inside the muscle cone (which contains many vital structures: nerves / vessels and it is a confined area hemorrhage can increase the pressure in that area and damage these vital structures). It can lead to direct injury to the vital structures or retrobulbar hemorrhage (which can lead to central retinal artery occlusion and optic nerve atrophy. Emergent surgical intervention to decompress the orbit is needed)

3/ Peribulbar (Extraconal) block: Safer and widely used compared to retrobulbar.

	4/ Intraocular injections 	Intracameral(anterior chamber)or intravitreal delivers the medication directly into the eye. **Eamples:** ○ Intracameral acetylcholine (miochol) during cataract surgery. During cataract surgery, we put a lens inside the eye. How can we access the area behind the iris? We dilate the pupil → access the lens → aspirate it → put a lens. In order to secure the lens in position, we inject acetylcholine into the eye (parasympathomimetics) → constricts the pupil and luck the lens. ○ Intravitreal antibiotics in cases of endophthalmitis a true ocular emergency that happens when an organism reaches the vitreous cavity and releases endotoxins that destroy the ocular tissue. Giving an antibiotic ASAP is essential. ○ Intravitreal steroid in macular edema (diabetic patients) ○ Intravitreal anti-VEGF(anti - vascular endothelial growth factor) for Diabetic retenopathy. the most recent method of treating diabetic retinopathy.
	5/ Sustained-release devices 	These are devices that deliver an adequate supply of the medication at a steady-state level, These devices increase patients compliance by decreasing the frequency of administration. **Eamples:** 1. Ocusert delivering pilocarpine. 2. Timoptic XE delivering timolol. 3. Ganciclovir sustained-release intraocular device. Antiviral (CMV retinitis in cases of immunocompromised patients). We implant it surgically. 4. Collagen shields. Dissolve in 2-3 hrs while the contact lens will not dissolve
Syst-emic	Oral or IV. (When do we use them? posterior segment or bilateral involvement of the eye / Autoimmune disease or TB Infection "the disease is outside the eye"/ or if there was a disease in the eye and I don't want it to spread) **Factor influencing systemic drug penetration into ocular tissue:** 1. **Lipid solubility of the drug:** more penetration with high lipid solubility , Major factor : more lipid binding less effect. Lipid solubility is favorable in case of systemic medications 2. **Protein binding:** more effect with low protein binding. More protein binding = will not go to ocular tissue 3. **Eye inflammation:** more penetration with ocular inflammation.so, when the eye is inflamed, we decrease the dose.	

Factors influencing local drug penetration into ocular tissue EXAM

1/ Drug concentration	The higher the concentration the better the penetration e.g. pilocarpine 1-4% but limited by reflex tearing.If you increase the concentration of a solution → more strong & more **irritant** to the ocular tissue → release of tears. So they are testing different amounts of concentrations and they reach certain concentration beyond which the irritation will be less.
2/ Viscosity	Higher viscosity increases drug penetration by: • Increasing the contact time with the cornea. • Altering corneal epithelium. More penetration to the ocular tissue.
3/ Lipid solubility	The higher lipid solubility the more the penetration (because of the lipid rich environment of the epithelial cell membranes). (more lipophilic = more diffusion to the ocular tissue = more effect of the medication).
4/ Surfactants	The preservatives used in ocular preparations alter cell membrane in the **cornea** and increase drug permeability e.g. benzalkonium and thimerosal. Simply, surfactants are preservatives (compound added to medications to make these medications stable for certain period of time). There are some medications with or without preservatives. Preservatives alter (loosen) the junction between the cell membranes, so there will be more diffusion of the medication to the ocular tissue "damage to corneal epithelium". Sometimes scraping of cornea is done to increase penetration "same idea"

5/ pH	The normal tear pH is 7.4(a little bit alkaline=alkaloid شبه قلوي) and if the drug pH is much different, this will cause **reflex tearing**. Both acidic or alkaline are not desirable because both will be identified by the eye as irritants.
6/ Drug tonicity	When an alkaloid drug is put in relatively alkaloid medium, the proportion of the uncharged form will increase, thus more penetration. The non-ionized form of the medication is the active form. So if you want a non-ionized drug, you should make it more ALKALINE (~7.5, not so far from 7.4)

Ocular Pharmacotherapeutics

Before starting you need to know the autonomic nervous system effect on the eye:

- Sympathetic NS → (1) pupil dilation "contraction of pupillary dilator or radial muscle" (2) decrease production of aqueous humor (3) retraction of the eye "contraction of muller muscle"
- Parasympathetic NS → (1) pupil constriction "contraction of pupillary constrictor or circular muscle" (2) increase production of aqueous humor. (3) Accommodation "contraction of ciliary muscle → suspensory ligament will relax → the lense will be rounded → optical power of the lense will increase "this help in near object vision, it will bring the image on the retina"

Accommodation is accompanied by 2 things: 1) convergence. 2) miosis. All these brings the image on the retina. As lense accommodation increase the refractive power, the convergence of the eyes will keep the image in the center of fovea, and miosis increase the depth of focus of the eye by blocking the light scattered by the periphery of the cornea. See the video here.

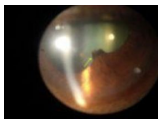
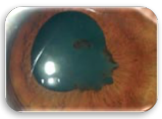
Note: anti-glaucoma medication. Patient takes it for life.

Cholinergic agonist

Directly acting agonists:	**Indirectly acting (anticholinesterases):** More potent with longer duration of action (act by binding to cholinesterase, the inhibition of this enzyme could be reversible or irreversible)	
[pilocarpine, acetylcholine (miochol), carbachol (miostat)]	**Reversible inhibitors** [Physostigmine used in Myasthenia Gravis]. Less potent, not commonly used	**Irreversible inhibitors** [e.g.phospholine iodide].
To Induce miosis, for glaucoma. **Mechanism of action:** • Miosis by contraction of the iris sphincter muscle. • Accommodation by circular ciliary muscle contraction. A change in lens refractive power in order to see the nearer objects. • **Increases aqueous outflow** (inside eye to outside) through the trabecular meshwork by longitudinal ciliary muscle contraction. Contraction of the longitudinal ciliary muscle > **the spaces in trabecular meshwork will open** > more fluid will go to the circulation. • Note that in people with hyperopia "the eye is small" this mechanism can lead to iridolenticular apposition → glaucoma	**uses:** • glaucoma We use it to treat glaucoma as it increase the aqueous outflow (drainage to the circulation = less fluid in the eye = less pressure). • lice infestation of lashes.يسبب نيوروتوكسستي للقمل ويموتون	• Used in **accommodative esotropia**.  - Esotropia = Eyes in - Exotropia = Eyes out These children are usually hyperopia which means that the image is formed behind the retina. Accommodation increases the refractive power of the eye and brings the image from behind the retina to the retina. - When someone accommodates or changes his refractive power 2 things happen: 1) miosis 2) conversion. (eyes will go in). So, when these children try to accommodate, the 2 things (miosis & conversion) happen. This explains why they have esotropia. **- We use this medication to induce accomodation without actively having the child to accommodate himself.** (they have strabismus when focusing in typically farsightedness)

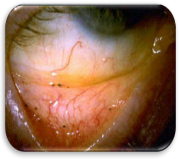
Side effects: **Local**: diminished vision (**myopia** with long use. Why? 1. Optical power is increased and 2. the contraction of longitudinal ciliary muscle will bring the lense forward → focal point will be in front of retina), headache (continuous contraction of the ciliary muscle), cataract, miotic cysts, and rarely retinal detachment. **Systemic:** diarrhea, lacrimation, salivation, perspiration, bronchospasm, nausea, vomiting and urinary urgency.	**Side effect**: CNS side effects.	**Side effect**: Iris cyst Anterior subcapsular cataract Can causes apnea if used with succinylcholine or procaine. Use atracurium as an alternative. **Contraindications** (special): angle closure glaucoma
Contraindications: asthma, Parkinsonism		

Cholinergic antagonist

Cholinergic antagonists	
Examples	Tropicamide (6 hrs), cyclopentolate, homatropine, atropine (10-14 DAYS)
Cause	Mydriasis (by paralyzing the sphincter muscle) with cycloplegia (by paralyzing the ciliary muscle (so there will be loss of accommodation, warn the patient that they won't be able to read for a few hours ضروري يمكن يكون عنده شغله تحتاج تركيز!)
Uses	Fundoscopy, cycloplegic refraction (procedure to measure the refractive error by temporarily paralyzing the muscles that aid in the accommodation because if there is **accomodation** the result would be wrong glass prescription.Thus, measurement will be precise.), anterior uveitis (inflammation → adhesion. We give it to decrease the contact between the iris and the lens → decrease adhesions), Cycloplegic drop help manage pain when there is inflammation in the eye, the ciliary body may spasm, causing pain.   Pic: Adhesion of the iris to the lense AKA posterior synechiae
Side effects	• **local:** allergic reaction, blurred vision especially in bright light • **Systemic:** nausea, vomiting, pallor, vasomotor collapse, constipation, urinary retention, and confusion ➔ Specially in children they might cause flushing, fever, tachycardia, or delirium ➔ Treatment by DC (direct cholinergic) or physostigmine as an antidote

Adrenergic agonists

<table>
<tr><th>Non-selective agonists</th><th colspan="2">Selective agonists</th></tr>
<tr><td>( ,  ,  , ) E.g. epinephrine, dipivefrin (prodrug of epinephrine)</td><td>Alpha-1 agonists (e.g. phenylephrine) work on muscle</td><td>Alpha-2 agonists (e.g. brimonidine, apraclonidine) decreases the pressure</td></tr>
<tr><td>Used in glaucoma in the past

<u>Side effects:</u>
• Headache, arrhythmia, increased blood pressure,
• cystoid macular edema in aphakic eyes. phakic=lens, pseudophakic= artificial lens, Aphakic=no lense زمان لما كانوا يشيلوا الكتراكت ماكانوا يحطون عدسات مكانها ماكنوا مكتشفين انه ممكن نتركب العدسه داخل العين , طيب فلو هذول الاشخاص استخدموا ابنيفرن مرح يصير فيه حاجز على طول بتتدخل القطره للريتينا و الماكيو لا مكونه سيست
• conjunctival adrenochrome (pigments/black dots in conjunctival fornix in pt use adrenergic drugs chronic users of epinephrine)”pic”
Pic : notice the small dots if present, you have to ask about these drug, the dots are dangerous because it is a pigmentation lesion.  </td><td>• Used to induce Mydriasis (<u>without</u> cycloplegia) for fundus evaluation, they do not have effect on the ciliary muscle,
➔ Q)WHAT IS THE MEDICATION THAT CAUSES MYDRIASIS WITHOUT CYCLOPLEGIA?يجيك سؤال وحده عندها اختبار بعد العياده وانتي تبين تسوينلها فوندس اقزامنيشن وش افضل قطرات تحطينا بحيث ماتأثر عالقراءه
• decongestant it is a vasoconstrictor

<u>Side effect</u>:
• Can cause significant <u>increase blood pressure</u> especially in infant and susceptible adults
• Rebound congestion
• Induce acute angle-closure glaucoma in patients with narrow angles</td><td>• <u>Uses:</u> glaucoma treatment [treatment of the <u>open</u> angel not the closure angle] and prophylaxis after glaucoma laser procedures.
• <u>Mechanism:</u> decrease aqueous production, and increase uveoscleral drainage (drainage 90% by canal of schlemm, 10%outflow uveoscleral) most of the drainage happen through trabecular meshwork (conventional). This medication affects only the uveoscleral one.

<u>Side effect</u>:
• Local: allergic reaction, mydriasis, Conjunctival blanching (pale), lid retraction (it activates sympathetic which innervates muller muscle).
In the exam: pic & asking which eye is using agonist? The more opened eye.
• Systemic: oral dryness, headache, fatigue, drowsiness, orthostatic hypotension, vasovagal attacks</td></tr>
<tr><td colspan="2"><u>Contraindications</u>:
• <u>closed</u> angle glaucoma can cause crowdening of the angle
• cardiac patient.</td><td><u>Contraindications</u>:
• infants because of their CNS side effects
• MAO inhibitors users tendency to increase BP
*MAO: monoamine oxidase inhibitors for depression</td></tr>
</table>

★ فإذاً عمرنا مانستخدم adrenergic agonist مع ال angle glaucoma closed !! لأنها تصغر الزاويه اكثر فتزيد it dilates the pupil and draws the peripheral iris toward the angle!!!! الأمور سواءًا

اتفقنا لاتنسونها طول حياتكم ^.^

Adrenergic antagonists

Alpha adrenergic antagonists	Beta-adrenergic antagonists (the most effective initial treatment of open glaucoma)
E.g. thymoxamine, dapiprazole	• **Nonselective:** timolol, levobunolol, metipranolol, carteolol • **Selective:** betaxolol (beta 1 "cardioselective") **(Good for asthmatic)**
Uses: to reverse pupil dilation produced by phenylephrine (better not to be used because of the risk of retinal detachment) **Not widely used**	• **Uses**: glaucoma (commonly used to treat glaucoma because of their action on reducing the formation of aqueous by ciliary body) • **Mechanism**: reduce the formation of aqueous humor by the ciliary body. **Side effects**: bronchospasm (less with betaxolol) (non- selective:exacerbates bronchial asthma, COPD) , cardiac impairment

Carbonic anhydrase inhibitors

Examples	**Acetazolamide**, methazolamide, dichlorphenamide, dorzolamide, brinzolamide.
Mechanism	**aqueous suppression** (carbonic anhydrase have a role in producing aqueous humor)
Uses	glaucoma(if not respond to other meds), cystoid macular edema(previous mcqs), **pseudotumor cerebri (= idiopathic intracranial hypertension)** Increased ICP without the presence of a tumor, we use it to decrease production of CSF. CSF→ ↑ ICP→resembling tumorلوحظ انه يحصل بالنساء السمينات يقل عندهم الدرينج
Side effects	myopia, **paresthesia** circumoral numbness and **peripheral** numbness, anorexia, GI upset, headache, altered taste and smell , Na and K depletion, metabolic acidosis normal anion gap, **renal stone,** bone marrow suppression *"aplastic anemia"*.
Contraindications	sulfa allergy it's sulfa derivative, digitalis users lethal hypokalemia, pregnancy

Osmotic agents

(Used to suppress IOP as fast as possible in Acute attacks): Dehydrate vitreous body which reduce IOP *significantly* loading the circulation with high concentration of fluid > less conc. In the vitreous > the water will go from lesser to higher concentration, we are basically dehydrating the vitreous.

Examples are:

1. Glycerol 50% syrup (cause nausea, hyperglycemia) oral, caution in uncontrolled DM
2. Mannitol 20% IV (**cause fluid overload, avoid in heart failure and renal impairment!!!**) (evaluate CVS before use)

Use in case of acute angle closure glaucoma to reduce IOP rapidly.

Prostaglandin analogue

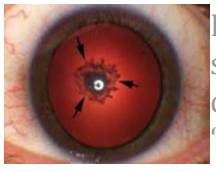
★ *CASE*:patient has bronchial asthma and high blood pressure presented with open angle glaucoma. the most appropriate drug to treat glucoma?Prostaglandins Analogue

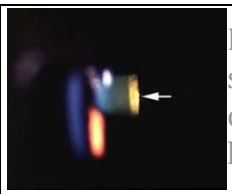
- **E.g.** latanoprost, bimatoprost, travoprost, unoprostone.
- **Uses**: glaucoma.
- **Mechanism**: increase **uveoscleral** aqueous outflow.

Side effects: darkening of the iris **(heterochromia iridis)**الصوره جت بساك سابق, lengthening and thickening of eyelashes ;p عطني واحد بروستاقلاندين بليز, intraocular inflammation because it's an inflammatory mediator, macular edema.

Anti-inflammatory it's very important to know the side effect of corticosteroid

	Corticosteroids		NSAIDS
	Topical	**Systemic**	
Example	• Fluorometholone • Remixolone • Prednisolone • Dexamethasone • hydrocortisone	• Prednisolone • cortisone	• Ketorolac, • Diclofenac • flurbiprofen
uses	• Postoperatively • Anterior uveitis • Severe allergic conjunctivitis we don't use it as 1st line, only if other medications failed. • Vernal keratoconjunctivitis (severe form of ocular allergy) • Prevention and suppression of corneal graft rejection • Episcleritis • Scleritis	• Posterior uveitis • Optic neuritis • Temporal arteritis with anterior ischemic optic neuropathy.	• Postoperatively • Mild allergic conjunctivitis • Episcleritis • Mild uveitis • Cystoid macular edema • Preoperatively to prevent miosis during surgery "to inhibit prostaglandin which is known to constrict the pupil"
Mechanism	inhibition of arachidonic acid release from phospholipids by **inhibiting phospholipase A2** (very potent)		**Inactivation of cyclooxygenase enzyme** (prevent formation of PG which causes miosis, so we use it in cataract surgery)
Side Effects	• Susceptibility to infections • **Glaucoma** • **Cataract** • Ptosis • Mydriasis • Scleral melting • Skin atrophy على كثر البلاوي الي تسويها بالعين بس انتبهوا انها ماتسبب اوبتك نيورايتس فلايلخبطونكم بالخيارات :) • The most serious side effect of topical steroids is increased IOP (STEROID INDUCED GLAUCOMA) asymptomatic, permanent damage.	**Local: posterior subcapsular cataract**, glaucoma, central serous retinopathy **Systemic:** suppression of pituitary-adrenal axis, hyperglycemia, osteoporosis, peptic ulcer, psychosis	• Stinging.


Posterior subcapsular cataract "retroillumination"


Posterior subcapsular cataract "slit lamp"

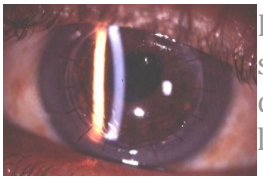
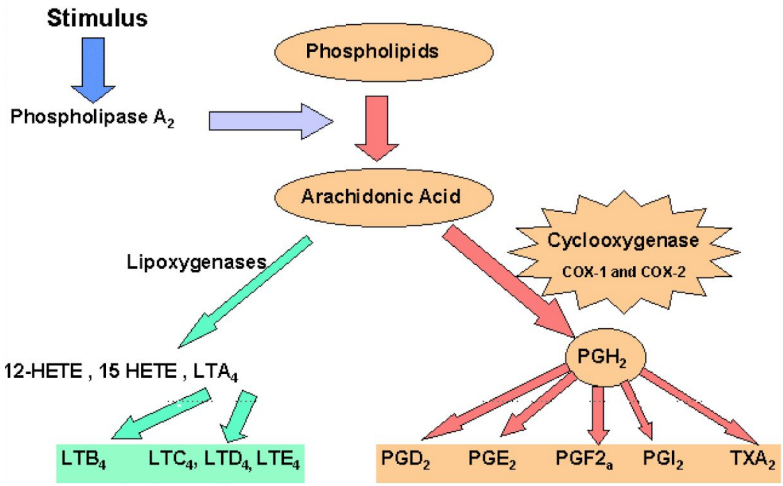

Posterior subcapsular cataract "slit lamp"

Figure 2 : Biosynthesis of eicosanoids



Anti Allergic

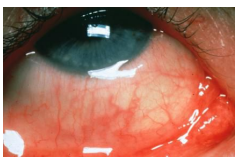
★ **Remember:** the use of antiAllergic should be (**temporary**): antihistamine or steroids (only prescribed in serious situations and for a short period because of the serious side effects).

Type	Example	Mechanism and Uses	Side Effect
Antihistamines	Pheniramine levocabastine	Work by **blocking histamine** that is produced by the body in response to allergens or irritants	Drowsiness. bradycardia and overdose may lead to sleep disorders.
Decongestants	naphazoline phenylephrine tetrahydrozoline	used to relieve redness, puffiness, and itchy/watering eyes due to colds, allergies, or eye irritations	Stinging. Redness. widened pupils, or blurred vision.
Mast cell stabilizers	cromolyn, lodoxamide pemirolast nedocromil olopatadine	They **block a calcium channel** essential for mast cell degranulation, stabilizing the cell and thereby **preventing the release of histamine** and related mediators.	-
NSAID	Ketorolac	-	Stevens Johnson syndrome.
Corticosteroids	Fluorometholone remixolone prednisolone	-	Posterior subcapsular cataract. Glaucoma. Papilledema. Predisposition to fungal infections. لو جاكم سؤال وحده عندها الرجي بالكونجكتافا وطاحت على دواء يخففها وصارت كل شوي كل شوي تستخدمه لين صارلها high IOPايش هو الدواء ياترى :) ماغيره كورتيكوستيرويد

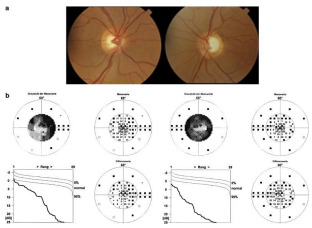
Tips:

✓ Avoidance of allergens, cold compress(نستخدم ثلج عشان نسوي كونستركشن للفيزيلس فماعاد يوصل دم ولا انفلاماتوري سيلز للمنطقه ونفتك منها ومن غثاها:), lubrications.

✓ Drug combinations(if needed)



Chemosis (روحوا ابحثوا وش تعني يورهوم وورك:)



cupping "visual field defect" seen in advanced glaucoma

EXTRA PAGE Taken from Lecture Note book:

للأمانه ترتيب السلايدز (الترتيب السابق) لأدويه الجلوكوما موعاجبني ابد :) هذا ترتيب ثاني حسب نوع الجلوكوما

Chronic Open angle glaucoma

Medical treatment:
In chronic open angle glaucoma the **prostaglandin analogues** are becoming the first-line treatment. They act by increasing the passage of aqueous through the uveoscleral pathway. Topical adrenergic **beta-blockers** may further reduce the pressure by suppressing aqueous secretion. Non-selective beta-blockers carry the risk of precipitating asthma through their beta-2 blocking action, following systemic absorption, or they may exacerbate an existing heart block through their beta-1 action. **Pilocarpine** may occasionally be used in the treatment of chronic open angle glaucoma.
If intraocular pressure remains elevated the choice lies between:

- **adding additional medical treatment**
- **laser treatment**
- **surgical drainage procedures**

Primary angle Closure glaucoma

The acute and dramatic rise in pressure seen in angle closure glaucoma must be **urgently** countered to prevent permanent damage to the vision.

- **Medical treatment(initially):** **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 gradient across the iris. These measures often break the attack and lower intraocular pressure.
- **Subsequent management requires that a small hole (iridotomy or iridectomy) be made in the peripheral iris to prevent further attacks.** This provides an alternative pathway for fluid to flow from the posterior to the anterior chamber, bypassing the pupil and thus reducing the pressure gradient across the iris. This can be done with a YAG laser or surgically.

Congenital glaucoma

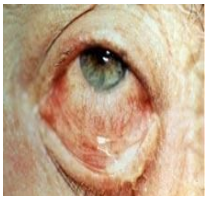
Congenital glaucoma is usually treated surgically:
An incision is made into the trabecular meshwork (goniotomy) to increase aqueous drainage, or a direct passage between Schlemm's canal and the anterior chamber is created (trabeculotomy).

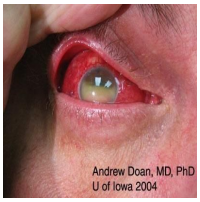
Secondary glaucoma

Treatment broadly follows the lines of the primary disease. In secondary glaucoma it is important to treat any underlying cause, e.g. uveitis, which may be responsible for the glaucoma

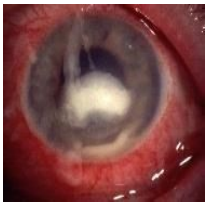
Antibiotics

Examples	Penicillins, Tetracyclines,Fluoroquinolones,Cephalosporins,Chloramphenicol,Vancomycin Sulfonamides,Aminoglycosides,Macrolides.
Uses موضع أسئله لاتسلكون لها تراها اعاده لدرس الاكيولر اميرجنسي	• Used **topically** in prophylaxis (pre and postoperatively) and treatment of ocular bacterial infections. • Used **orally** for the treatment of preseptal cellulitis e.g. amoxicillin with clavulanate, cefaclor. If there is an inflammation or infection in front of orbital septum > preseptal cellulitis > we use oral antibiotics. If the inflammation or infection is behind this septum > orbital cellulitis > a true ophthalmic emergency that can progress to meningitis or encephalitis or cavernous sinus thrombosis or periosteal abscess, that's why we treat it aggressively with **IV** antibiotics + admit the patient. • Used **intravenously** for the treatment of orbital cellulitis e.g. gentamicin, cephalosporin, vancomycin, flagyl. • Can be injected **intravitreally** for the treatment of endophthalmitis.a true ophthalmic emergency that has to be treated by injecting the antibiotic directly to the eye, otherwise nothing can help! Hypopyon (collection of pus in the anterior chamber) is a sign of endophthalmitis. • **Trachoma** can be treated by topical **and** systemic tetracycline or erythromycin, or systemic azithromycin. Caused by chlamydia trachomatis • **Bacterial keratitis** (bacterial corneal ulcers) can be treated by topical fortified penicillins, cephalosporins, aminoglycosides, vancomycin, or fluoroquinolones **"hourly".** Keratitis = cornea, there will be opacity. It can affect the vision permanently. Treat it with TOPICAL Abx EVERY HOUR even if the pt is sleeping. • **Bacterial conjunctivitis** is usually self-limited but topical erythromycin, aminoglycosides, fluoroquinolones, or chloramphenicol can be used. Broad spectrum Abx


Ectropion



Endophthalmitis


Corneal Ulcer

Antifungal

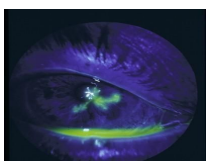
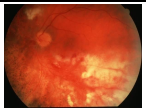
Uses: fungal keratitis, fungal endophthalmitis.

1/ Polyenes: E.g. amphotericin B, natamycin.
Damage cell membrane of susceptible fungi.
Side effect: nephrotoxicity.

2/ Imidazoles: E.g. miconazole, ketoconazole.
Increase fungal cell membrane permeability.

3/ Flucytosine:
Act by inhibiting DNA synthesis

Antiviral

Acyclovir	• interact with viral thymidine. • Kinase (selective). • Used in herpetic keratitis. **This picture could come in the exam > Stained eye showing dendritic shape ulcer > herpetic keratitis in fluorescein stain .** 
Trifluridine	• **More** corneal penetration. • Can treat herpetic iritis.
Ganciclovir	• Used intravenously for **CMV Retinitis.** Immunocompromised pts. 

Ocular diagnostic drugs ادري انكم بديتوا تطفون لما وصلتوا هالنقطة بس ركزوا بكل حرف فيها جزئيه سهله مره بس يحبووون يسألون عنها(:

Fluorescein dye:

it goes to the area that doesn't have epithelium, so if the surface epithelium is deficient in some area,it will be stained by Fluorescein. Available as drops or strips

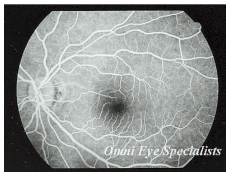
Uses: stain corneal abrasions, applanation tonometry to measure IOP, detecting wound leak, nasolacrimal duct obstruction If a patient came to you with tearing in the right eye, but the Fluorescein in the eye and check it after 5 minutes, after that, if you find more Fluorescein in the eye it means that the Fluorescein did not go to the nasal cavity means that there is some blockage in the nasolacrimal duct. That's how we diagnose NLD obstruction., fluorescein angiography I.V. > retinal circulation.

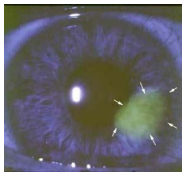
Caution!

- ★ Stains soft contact lens.so before staining Ask if the patient is wearing any contact lens as fluorescein might stain it permanently
- ★ Fluorescein drops can be contaminated by Pseudomonas sp.

Normal Fluorescein angiography

In the normal macula, the capillary-free zone is seen as dark due to blockage of choroidal fluorescence retinal pigment epithelial cells





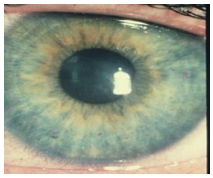
Abrasion stain

Dendritic ulcer staining with fluorescein

Rose Bengal Stain:

Stains devitalized epithelium.

Uses: severe dry eye **(use it if u suspect sjogren's syndrome)**, herpetic keratitis



Local anesthesia

1/ Topical: E.g. proparacaine, tetracaine.

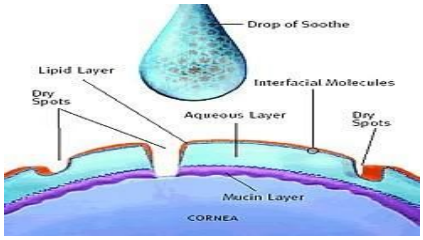
- **Uses:** applanation tonometry, gonioscopy viewing the angle of the eye, removal of corneal foreign bodies, removal of sutures, examination of patients who cannot open eyes because of pain.
- **Adverse effects:** toxic to corneal epithelium, allergic reaction rarely.

2/ Orbital infiltration: Peribulbar or retrobulbar.

- Cause anesthesia and akinesia for intraocular surgery. E.g. lidocaine, bupivacaine.

Lubricants

- Drops or ointments.
- Polyvinyl alcohol, cellulose, methylcellulose.
- Preserved or preservative free. Less irritation

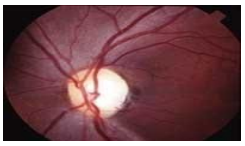
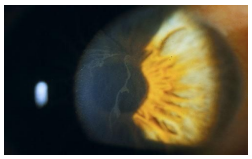
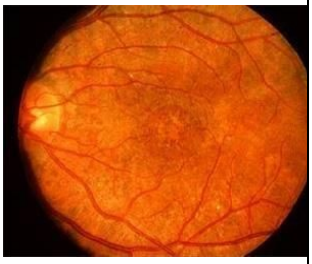


Ocular toxicology

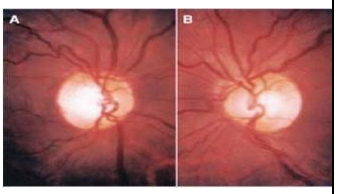
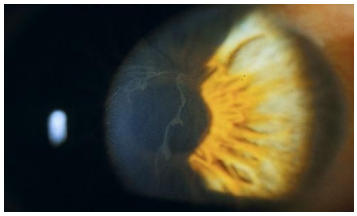
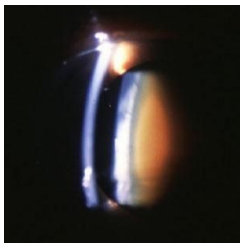
Complications of topical administration

- Mechanical injury from the bottle e.g. corneal abrasion.
- Pigmentation: epinephrine adrenochrome.
- Ocular damage: e.g. topical anesthetics, benzalkonium.
- Hypersensitivity: e.g. atropine, neomycin, gentamicin.
- Systemic effect: topical phenylephrine can increase BP.

Drugs associated with ocular toxicity

Drug	Effect
Amiodarone	• A cardiac arrhythmia drug. • **Causes optic neuropathy** (mild decreased vision, visual field defects, bilateral optic disc swelling)  • Also causes **corneal vortex keratopathy (corneal verticillata)** which is whorl-shaped pigmented deposits in the corneal epithelium. 
Digitalis	• A cardiac failure drug. • Causes **chromatopsia (objects appear yellow)** with overdose. abnormal perception of color. If the object appears yellow > xanthopsia. :) اموت اذا ماسألوا عنها  
Chlorpromazine	• A psychiatric drug • Causes corneal punctate epithelial opacities, lens surface opacities • Rarely symptomatic • Reversible with drug discontinuation.
Thioridazine	• A psychiatric drug • Causes a pigmentary retinopathy after high dosage • **SALT AND PEPPER APPEARANCE** 

Diphenylhydantoin	• An epilepsy drug
	• Causes dosage-related cerebellar-vestibular effects:
	  ○ Horizontal nystagmus in lateral gaze
	  ○ Diplopia, ophthalmoplegia
	  ○ Vertigo, ataxia
	• Reversible with the discontinuation of the drug

Topiramate	• A drug for epilepsy • Causes **acute angle-closure glaucoma** (acute eye pain, redness, blurred vision, halos). • Treatment of this type of acute angle-closure glaucoma is by cycloplegia and topical steroids (rather than iridectomy) with the discontinuation of the drug
Ethambutol	• An anti-TB drug • Causes a **dose-related optic neuropathy** • Usually reversible but occasionally permanent visual damage might occur 
Chloroquine	• E.g. chloroquine, hydroxychloroquine • Used in malaria, rheumatoid arthritis, SLE • Cause vortex keratopathy (corneal verticillata) which is usually asymptomatic but can present with glare and photophobia • Also cause retinopathy **(bull's eye maculopathy)** • **vortex keratopathy could be caused by BOTH amiodarone and Chloroquine.**  
HMG-CoA REDUCTASE INHIBITORS (STATINS):	• E.g. pravastatin, lovastatin, simvastatin, fluvastatin, atorvastatin, rosuvastatin • Cholesterol lowering agents. • Can cause cataract in high doses especially if used with erythromycin  
Methanol	Optic atrophy and blindness (Patient presents with history of alcohol drinking).
Contraceptive pills	Pseudotumor cerebri (papilledema), and dryness (CL intolerance)
Chloramphenicol and streptomycin	Optic atrophy
Hypervitaminosis A	Yellow skin and conjunctiva, pseudotumor cerebri (papilledema), retinal hemorrhage.
Hypovitaminosis A	Night blindness (nyctalopia), keratomalacia.

Agents that can cause Toxic Optic Neuropathy

- **Methanol (IMP!!!)** Can cause bilateral blindness
- Ethylene glycol (antifreeze)
- Chloramphenicol
- Isoniazid
- Carbon monoxide
- Lead
- Mercury
- Ethambutol
- Digitalis
- Chloroquine
- Streptomycin
- Thallium (alopecia, skin rash,severe vision loss)
- Malnutrition with vitamin B-1 deficiency
- Amiodarone
- Quinine
- Methotrexate
- Pernicious anemia (vitamin B12 malabsorption phenomenon)
- Vincristine and methotrexate (chemotherapy medicines)
- Sulfonamides
- Melatonin with Zoloft (sertraline, Pfizer) in a high-protein diet
- Radiation unshielded (exposure to >3,000 rads).

Refractive Errors

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

Objectives:

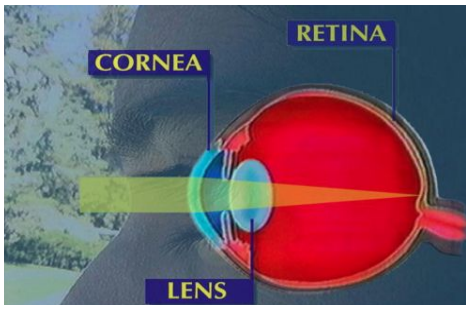
➢ Not given.

Done by: Ruba Alselaimy, Munira Alhussaini.
Edited & revised by: Lamya Alsaghan.
Resources: Slides + Notes + Lecture Notes of Ophthalmology + 434 / 435 Team.

Last 2 pages are extras from Group A!

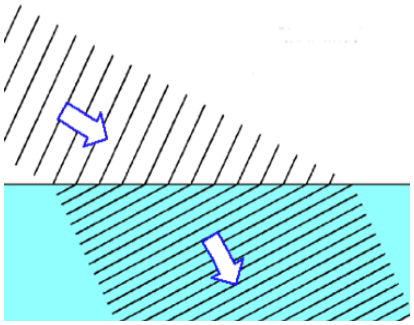
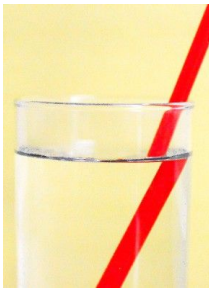
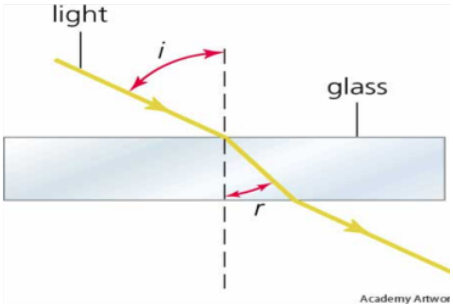
Physiology

• Refraction



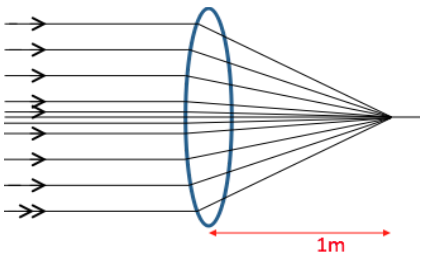
- Simply light enters the eye by going through the cornea and lens. These two organs modify light rays by a phenomenon called **refraction (انكسار الضوء)** in order to have a single point -not two- in the fovea as it should be to see clearly.

- When light waves travel from a medium with a given refractive index to a medium with another refractive index. At the boundary between the two media, the wave phase **velocity is altered, it changes direction**.
- The amount of bend depends on the refractive index of the media and the angle of incidence.

		
Important: light refracts towards central line from low to high density. **For example**, when light moves from air (lower density) to water (higher density), light waves will slow down then it will refract towards the medial vertical line (central line).	Light passes two different media and the straw appears to be broken due to refraction but it isn't.	- Light passes through the glass from: * Low to high → **TOWARDS** central line * High to low → **AWAY** from central line - Light rays (picture: yellow) are parallel to each other. متوازيين

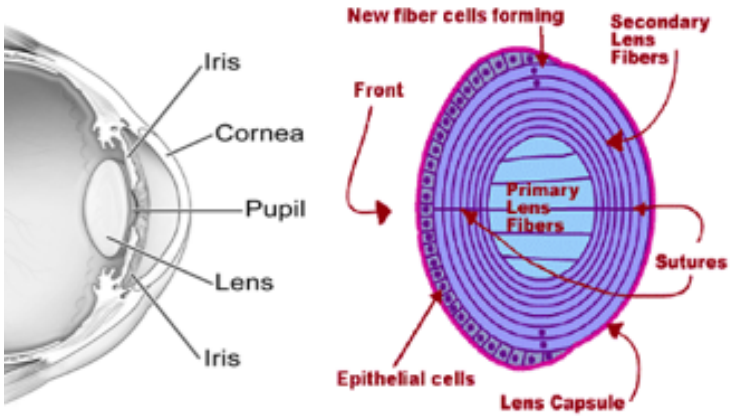
- Unit of refraction:

- **Diopter = 1 / focal length of a lens.**
- The power of the lens is measured by the diopter (D).
- The eye requires about **60 diopters** of power to focus the light from a distant object (6 meters or more) precisely onto the retina.

When looking at a far object, light rays are coming parallel (≥ 6 meters). It will enter through the lens and then it will refract in a single focal point at 1 meter. This is due to the power of lens, which is 1 diopter. **Example:** If we used another lens, rather than having a focal point at 1 meter it will be at 25 cm distance. What is the power of the lens? **4 diopter.** How? diopter = 1/focal length (m) | 25 cm → 0.25 m | diopter = 1/0.25 = 4 "ليش نقول الكلام هذا؟ لأننا نتعامل مع العين و العين قوتها 60D (مجموع الـ cornea & lens)"	

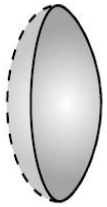
- The refracting surface of the cornea and lens are spherically convex.
- Cornea has a higher refractive index than air; the lens has a higher refractive index than the aqueous and vitreous humours that surround it. The velocity of light is reduced in a dense medium so that light is refracted towards the normal. When passing from the air to the cornea, or from the aqueous to the lens, the rays converge.

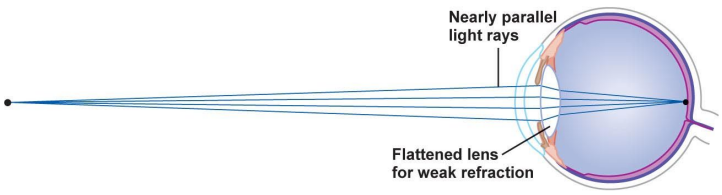
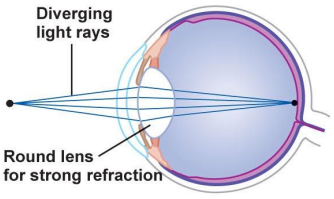
• The eye's optical system: very important

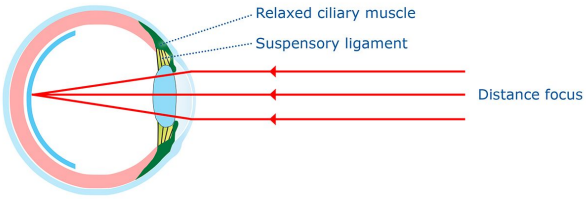
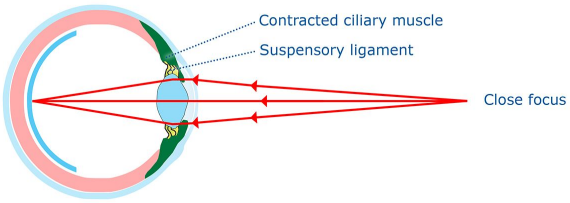
Cornea	Crystalline Lens
Main refracting surface.	**Double purpose:** balancing eye's refractive power and providing a focusing mechanism.
Provides **40 diopters**, or 75% of the total refracting power of the eye. (2/3 the power of the eye)	Provides **20 diopters** (القوة البؤرية) of refractive power. (1/3 the power of the eye)
Fixed	**Changes** (depends on how far the object is from the eye)
 In children the power of cornea = 32D. It reaches the maximum power at the age of 18 = 40D. That's why it's NOT recommended to do any refractive surgery before age of 18. At age 40 they will have presbyopia. At age 60 = 0D.	

• Accommodation "theories of eye accommodation LINK"

- **Helmholtz theory:** When the eye look at close object → contraction of ciliary muscle → decrease tension in zonule fibers (relaxation) → elasticity of lens capsule mold lens into spherical shape anterior-posterior curvature (diameter) increase → greater dioptric power → divergent rays are focused on retina.
- Contraction of ciliary muscle is supplied by parasympathetic third nerve.
- After prolonged reading there might be ciliary spasm associated headache.



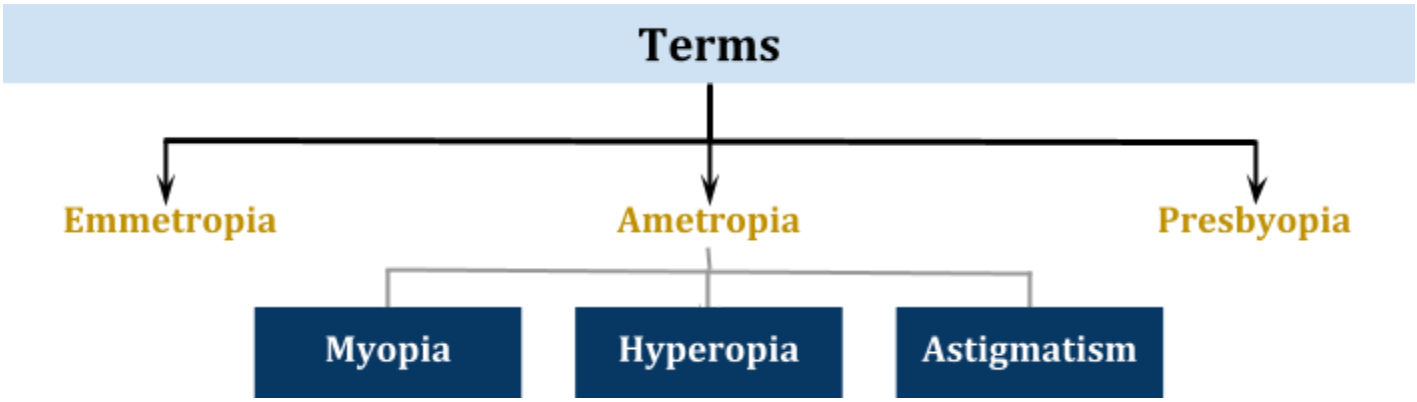
Viewing a DISTANT object (≥ 6 meters)	**Viewing a NEAR object** (< 6 meters)
	
Relaxed situation: Light rays are coming parallel Accommodation is very relaxed Eye power = 60D (40D + 20D)	* **Cornea:** will not change. * **Lens:** the closer the object the more diopter needed to maintain a point at the fovea. * If this mechanism is disrupted, as you get closer to the object the focal point will be behind the fovea (no accommodation) and vision will be unclear. More diopters are needed! (example: at 30 cm, lens goes from 20D -relaxed- to a higher diopter 23/25/28D) Refractive power can increase up to 15D more, like in children (with time it becomes less)

Accommodation	
 Looking at a far object. No accommodation.	 **1. Change of lens shape.** Notice how the shape of the lens changes in relation to different distances (in near object the rays are not parallel, so we need a more powerful lens to maintain the point in the fovea so the lens becomes more thick) **2. Miosis:** to allow as much light rays as possible to enter. **3. Convergence:** contraction of medial recti.

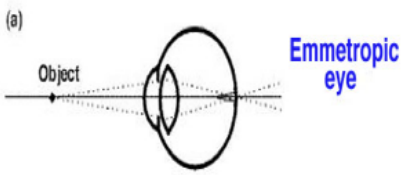
- **Axial length** (AL)**:**
  - Normal **axial length is 22.5 mml** (it's measured from the tip of the cornea to the surface of the retina).
  - if the axial length is **longer** = the picture will be in **front** of the retina "Myopia".
  - If the axial length is **shorter** = the picture will be **behind** the retina "Hyperopia".

Refractive Errors

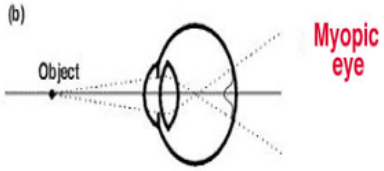
- Facts: 75% of avoidable blindness is due to: uncorrected refractive error, cataract, trachoma.
- A mismatch between the **refractive power (RP)** & the focusing distance of the eye. AL & RP should match!
- Inability to see clearly is often caused by refractive errors.



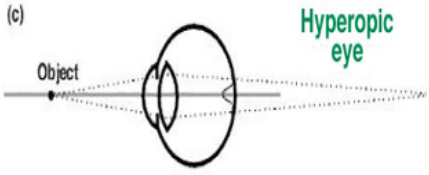
- **Emmetropia (normal)** ما يلبس النظارات و لا يعرفها
- **Ametropia = refractive error,** three types of refractive errors:
  - Myopia (nearsightedness)
  - Hyperopia (farsightedness)
  - Astigmatism.



Point at the retina
Matching between axial length (AL) & refractive power (RP)



Point in front of the retina
* high RP & normal AL **OR**
* normal RP & long AL



Point behind the retina
* low RP & normal AL **OR**
* normal RP & short AL

Emmetropia

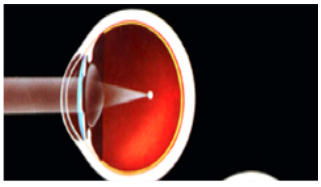
- Adequate correlation **OR** matching between **axial length** (distance from cornea to the fovea) and **refractive power of the eye** (cornea and the lens).
- Rays of light from a distant object are brought to a pinpoint sharp focus on the retina (without accommodation). fovea لما يكون قوة العين وطولها متطابقين معناته النقطة بتسقط على الـ
- All refractive errors are some deviation from emmetropia.

Ametropia

Myopia

- More common than hyperopia and astigmatism.
- Most prevalent among Asians (80-90%), African Americans (25%), and Caucasians (13%) varies according to race.
- **Etiology:** not clear, **genetic** (family history is important), acquired (excessive accommodation, near objects, aging)
- **Average age of onset:** 6/8/10 (preschool/ school age) to 20 years (normally stops at 20)

- Rays of light from a distant objects converge **in front of the retina**, causing a blurred image on the retina.
- The myopes can see close objects clearly, commonly known as "short-sightedness" قصر النظر: القريب أوضح من البعيد
- Myopia can be: **essential** (↑ RP, ↑ AL) OR **secondary** (other causes)

Causes of myopia: refractive & axial

1. Refractive myopia القوة كبيرة ولكن حجم العين طبيعي

a) Change in lens nucleus or shape:
- **Cataract** (ex. senile cataract: high density/thick/hard, low elasticity → high refractive power = induced myopia)
"senile cataract الـ جديدة بسبب myopia يبدأ تصير عنده أرقام 60/70 عمره لما يوصل لكن النظارة ما يعرف عمره طول"
- **Spherophakia:** congenital anomaly where the lens is spherical.
- **Diabetes**. How it affects the lens? Through a process called **"osmosis"** due to variabilities in blood sugar.
* Diabetics have both myopia and hyperopia depending on the level of the blood sugar.
* High blood sugar → high sugar in aqueous humour → shrink of lens → **HYPEROPIA**.
* Low blood sugar → fluid shift into lens → globular shape → **MYOPIA**
* Undiagnosed diabetics might end up with a brain injury. Uncontrolled (fluctuations in blood sugar) lead to **blurry vision.**
"لما تتابعي patient ويقولك أنا عندي زغللة نظر مستمرة تعرفي أنه مو controlled الـ blood sugar لأن فيه fluctuation عالي"

b) Lens repositioning:
- **Ciliary muscle shift:** treatments like miotics. Contraction of ciliary muscles persistently → induced myopia
- **Lens movement** e.g. anterior lens dislocation. Trauma → lens moves forward → image will be in front → myopia

c) Ciliary muscle tone: **Excessive accommodation**, e.g. medical students. Reading a lot at a near distance.

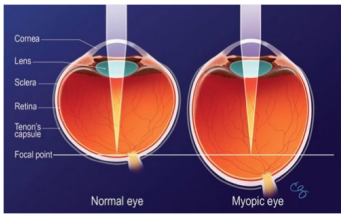
d) Increase corneal power: Keratoconus (cone shaped cornea), Congenital glaucoma (big eye + protrusion of cornea)


Keratoconus


Congenital Glaucoma

2. Axial myopia

- Excessive long globe. القوة طبيعية ولكن حجم العين كبير
- **More common ★**
- Causes: (what causes big eye)
a) Congenital glaucoma,
b) Posterior staphyloma (bulging of posterior part of eye).



Myopia Forms

Benign myopia (school age myopia)	**Progressive OR Malignant myopia**
- Commonest type ★ - Onset 8-12 years, myopia increases until the child stops growing in height. - Generally tapers off at about 18 to **20 years of age**.	- Also called degenerative or pathological myopia. - Myopia increases rapidly each year and is associated with, fluidity of vitreous and chorioretinal change. "الأرقام مستمرة في النزول"

Symptoms

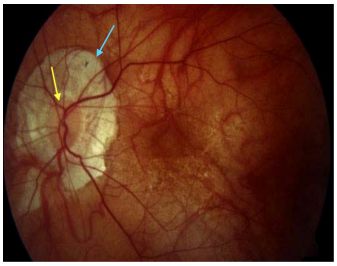
Headache (due to eyestrain), blurred distance vision, squint in an attempt to improve uncorrected visual acuity when gazing into the distance. | **Children:** strabismus & amblyopia.

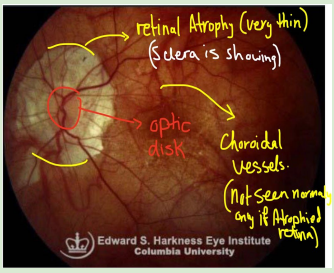
Morphologic eye changes (axial myopia)

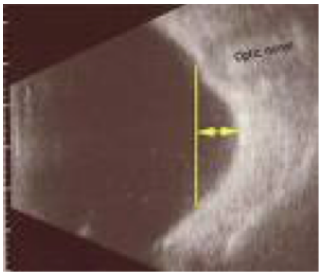
Changes occur with axial myopia specifically. The following changes occur with increase in length (from front to back):
1. Deep anterior chamber. | **2. Atrophy of ciliary muscle.** due to excessive stretching
3. Vitreous may collapse prematurely leading to opacification. The eye is bigger (more space) and the vitreous (gel-like substance) will stay the same. The space will be partially filled with vitreous and the rest with aqueous humour (watery) causing them to mix in a process called "liquefaction" (less density) **[important]**
4. Fundus changes: loss of pigment in RPE , large disc and white crescent-shaped area on temporal side , RPE atrophy in macular area , posterior staphyloma , retinal degeneration → hole → increase risk of retinal detachment & vision loss.
"لو قلنا أن أرضية الغرفة هي الـ retina وجينا بعدنا الجدار ، وش حيصير في الأرضية؟ فيه أماكن ما راح يكون فيها coverage في الـ retina، في البداية لو كان موكيت ممكن يجي معاك بس بعدين حينقطع بيصير حاجة اسمها retinal break الـ retina ما عاد تقدر أنها تغطي حجم العين الكبيرة فيصير فيه breaks والجل liquified فيدخل مع الـ breaks هذي، لما يدخل وش يسوي في الـ retina ؟ يدفها/يفصلها، فيصير عندنا حاجة اسمها retinal detachment أو انفصال الشبكية"

One of the most important risk factors of retinal detachment is high myopia. وممكن توصل للعمى

Posterior Staphyloma (bulging)







Yellow arrow: optic nerve with distorted margins because of vitreous | Blue arrow: retinal margins. | White area: The retina (which is transparent) and choroid (red in color) are very thin and the sclera (white) is showing (behind the retina). | **US:** shows bulging at the back of the eye because the pressure of the eye pushes the weak structures. (eye is getting bigger and the walls weaker)

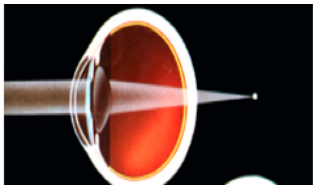
Correction of myopia

Concave lenses (negative)

Hyperopia

- Parallel rays converge at a focal point posterior to the retina. myopia نعكس كل الأشياء اللي كانت بالـ
- **Etiology:** not clear, **inherited**. trauma may cause dislocation of the lens.

- Rays of light from a distant object now focus **behind the retina**.
- Must accommodate when gazing into distance to bring focal point on to the retina. However, this reduces their accommodative reserve when they want to view close objects. So, their distance vision is generally better than near vision, hence the term "long-sightedness" "الأشياء البعيدة أوضح من القريبة لكن حتى البعيدة مو واضح مره"
- Can be: **essential** (↓ RP, ↓ AL) OR **secondary** (other causes)

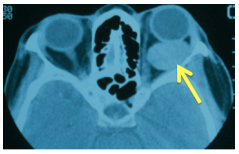
Causes of hyperopia: refractive & axial

1. Refractive hyperopia

- Decreased refractive power of the eye (insufficient):
a) Absent (a*phakia*: absent *lens*) or posteriorly repositioned lens (image at the back). can be congenital or due to trauma.
b) Weak accommodation: trauma (it affects muscles or zonules), marijuana. (causes weak accommodation)

2. Axial hyperopia

- Decreased effective axial length (excessive short globe)
- More common (retina is pushed forward)
- Causes: Tumor, orbital mass.
العين ماهي صغيرة فعليًا ولكن في حاجة ثانية دفت العين والـ retina

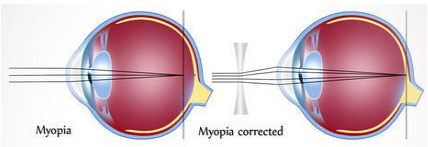
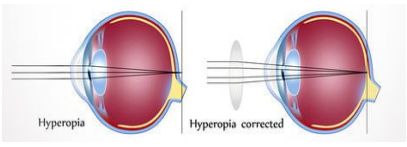

Cavernous Hemangioma

Symptoms

Eye pain / strain, headache in frontal region (especially after reading, they require more accommodation), visual acuity at near tends to blur (blurry vision) relatively early ''inability to read fine print'' | Pediatrics: strabismus & amblyopia.

Correction of hyperopia

Convex lenses (positive)

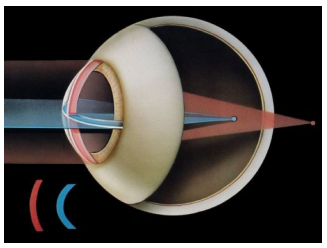
	Myopia	Hyperopia
Other name	Nearsightedness / Short-sightedness	Farsightedness / Long-sightedness
Focal point	Single point in front of the retina	Single point behind the retina
Refractive power	Increased	Decreased
Axial length	Increased (long eye)	Decreased (short eye)
Correction	Concave lens	Convex lens
Picture		

Astigmatism

- **اللابُؤَرِيّة:** ما عندها بؤرة وحدة /الانحراف (misleading لأن الانحراف ممكن يعني حول وهذا مو حول)
- Cornea is usually shaped like half a football. In these eyes there will be no astigmatism. you may describe it to the patient as " your eye is shaped as a rugby ball instead of a football"
- In astigmatism parallel rays come to focus in ≥ 2 focal lines rather than a single point.
- **Etiology:** hereditary.
- **Cause:** refractive media is not spherical → refract differently along one meridian than along meridian perpendicular to it → focal.

الكرة أقطارها متساوية ← single point focus ← طبيعي No astigmatism

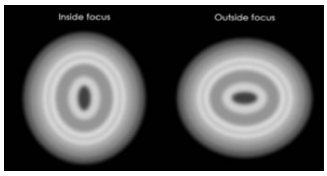
الكرة أقطارها غير متساوية جزء flat وجزء حاد ← two focal points ← غير طبيعي astigmatism



2 Meridians:
Blue: flat
Red: perpendicular
= 2 focal points

Classification

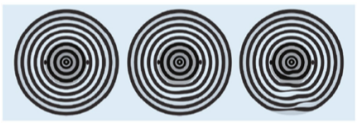
Regular astigmatism 2 focal points.



"لازم أتعامل مع two points، كيف أعالجها؟ **(1)** أشكلهم كـ one بعدين **(2)** أحطها على الـ retina، أما الـ myopia و hyperopia بس مجرد أحطها على الـ retina"

Irregular astigmatism ≥ 2 focal lines

"اللي على اليسار شكلها منتظم بس اليمين الـ cornea مضروبة بسكين (eye injury) وخيطناها لكن باقي المنطقة متعوجة (هي اللي تشتت الأضواء في أكثر من نقطة) وباقي الـ cornea تكون clear، هنا أنا أتعامل مع عشرة نقاط (أكثر من اثنين) فأصعب علاجها! فنجي لطريقة ثانية للـ treatment، أجيب حاجة وأضعها في القرنية فأخلي شكلها منتظم غصب عليها، ما ينفع معها glasses ولا ينفع معها شيء"



Types فيه افتراضات كثيرة

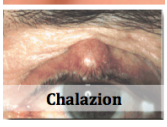
1. **Simple Myopic Astigmatism:** one before the retina, and one on the retina.
2. **Simple Hyperopic Astigmatism:** one on the retina and another behind the retina.
3. **Compound Myopic Astigmatism:** both are before the retina but at two different locations.
4. **Compound Hyperopic Astigmatism :** both behind the retina but at different virtual locations.
5. **Mixed Astigmatism:** one is before the retina and the other is behind the retina.

Causes

Corneal causes (majority)

- Simple corneal astigmatism. بس جزء بسيط متضرر الباقي منتظم
- Keratoconus (causes myopic astigmatism) very common in our community → Disease of the collagen of the cornea → astigmatism عالي الدرجة

- Masses e.g. lid tumor يضغط من فوق ويتغير شكلها شيل الـ tumor بيروح الوزن ويترفع الـ lid وترجع الـ cornea للوضع الطبيعي
- Ptosis: لما يكون الجفن مرتخي على الـ cornea يضغطها شوي فـ induce astigmatism، و مهم عند الأطفال، طفل عنده congenital ptosis يصير عنده astigmatism ويسوي له amblyopia.

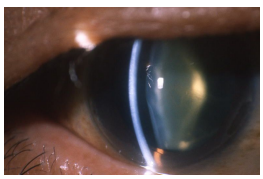


Lenticular causes

"واحد جاه بوكس وصار اختلال في شكل الـ lens"

- Lens dislocation. part of the zonules are cut.
جزء من العدسة مرتخي فتصير غير متساوية

- Lenticonus. ↓



Symptoms

Asthenopic symptoms (headache, eye pain), blurred vision, **distortion of vision** مع العالي الدرجة، يا عين أو عينين, head tilting يحاول يبعد عن الميلان اللي يزعجه and turning, amblyopia (with uncorrected astigmatism > 1.5 Diopters)

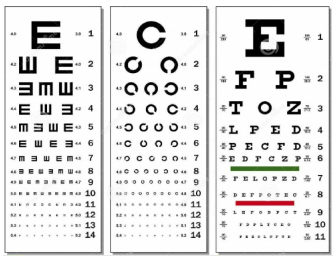
Presbyopia

- Physiological loss of accommodation in advancing age
- Deposit of insoluble proteins in the lens with advancing age → elasticity of lens progressively decrease → decrease accommodation.
- **Around 40 years of age, accommodation become less than** 3D → reading is possible at 40-50 cm → difficulty reading fine print, headache, visual fatigue.
- Lens is not as flexible because of decreased elasticity (gets dry) + zonules relaxes.
- Correction of Presbyopia: **convex lenses** for reading.

"المريض يستخدم النظارة للقراءة لكن يناظرك بدونها، تلقى طول عمره ما يعرف النظارة، ولما وصل الأربعين بدأ يلاحظ الأشياء القريبة موب واضحة فيبدأ يبعّد عشان توضح لكن بعدين مهما يبعّد ما توضح، فيضطر يستخدم نظارة عشان يقدر يقرأ لكن البعيد كويس"

Testing Vision

- **Visual acuity** حدة البصر "refraction لا تلخبطون بينها وبين الـ"



- Vital sign of the eye and first thing to do in the clinic with Intraocular pressure (IOP)
- **Central visual acuity:** display of different sized targets shown at a standard distance from the eye: Allen's & Snellen chart. "أشكال أو حروف، تتدرج بالحجم من كبير إلى صغير بقياس معين ومسافة معينة"
- Patient should be sitting & examine each eye alone (cover the other eye). Always start by showing large letters (assuming everyone is blind) and go smaller till normal.
- VA: 20/20 (American), 6/6 (British) | 20/20 ft = 6/6 m. **What does 20/20 mean?**

The patient can see at 20 feet what the normal population can see at 20 feet. Just like vital signs we have ranges from the normal population. **Examples:** 20/200: the patient can see at 20 ft what the normal population can see at 200 ft (far away from the normal). 20/40: the patient can see at 20 ft what the normal population can see at 40 ft (not that far away from the normal)

- **Testing poor vision**

First: If the patient is unable to read the largest letter < 20/200 → Move the patient closer e.g. 5/200.

"حتى الحرف الكبير ما يشوفه فأول شيء أسويه أقرب المريض، يعني مثلًا بدل ما أخليها 20ft أخليها 10ft وأجي أقول واللهي he can see الحرف الكبير at 10/200 فـ(he can see at 10 ft what the normal can see at 200 ft) بعد كذا ممكن حتى الحروف كلها ما يشوفها مهما قربت، نجي إلى ↓ "

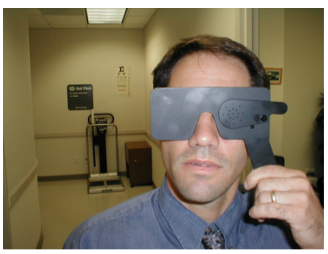
Second: If patient cannot read:

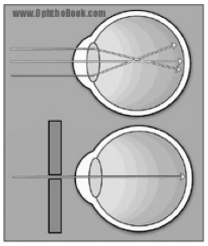
1. Count fingers (CF) examine each eye alone and write down the distance ex. CF at 1 foot or 2 feet. Not ↓
2. Hand motion (HM) Not ↓
3. Light perception (LP) with a torch examine all quadrants: up, down, right, left (sometimes all quadrants are affected except one). You have to document that.
4. No light perception (NLP) complete blindness ما يشوف تمامًا

- **Pinhole testing** simple but very important. Routinely done to all patients with decreased VA.

"**مثال:** مريض زار العيادة، أول ما جاء قسنا الـ VA حقته، لقينا حدة البصر 20/100 معناته أنه decreased، و (he is far away from normal)، طيب ليش؟ هل عنده refractive error ولا lens disease زي الـ cataract ولا retinal ولا optic nerve؟ we don't know نجي هنا نستخدم هذي الـ tool"

"زي البلاستيك يغطي عين والعين الثانية فيها فتحات صغيرة، نحط قدامه الـ Snellen chart ونقوله ركز على وحده من الفتحات الصغيرة و نقيس الـ visual acuity مرة ثانية (pinhole will cause muscle spasm). إذا تحسن لـ 20/20 فعنده refractive error بس ما ندري وش نوعه (corrects for about three diopters) وإذا ما تحسن فهو ماهو refractive error (شيء ثاني). كيف يتحسن النظر من فتحة صغيرة وأوصل 20/20؟ لاحظوا في الصورة الـ المريض عنده myopia لما أجي أحط له الـ pinhole بتسمح بدخول الـ central ray فقط وتروح على الـ fovea أما بقية الـ rays هي اللي مسببة مشاكل و confusion فـ we eliminate them. الفتحة هذي 1.2mm فـ (it has to be 1.2) لو زادت أو نقصت بتغير القياسات وما بتوصل 20/20، زي اللي يصغرون فتحة عينهم لما يناظرون التلفزيون يعملون pinholing عشان يتحسن نظرهم"





- **Measuring refraction** [video](#) **(1:05 to 1:31)**

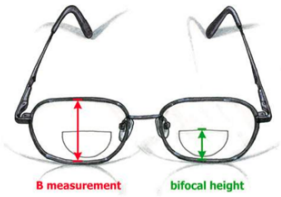

"الآن لقينا بالفعل المريض عنده refractive error، ما ندري عنده myopia ولا hyperopia ولا astigmatism، لازم نفرق هو جاي عندي ويبي أعطيه prescription، نبي نعرف النوع والدرجة in diopter، عشان نفصل له نظارة. فاستخدم الـ **retinoscopy** "

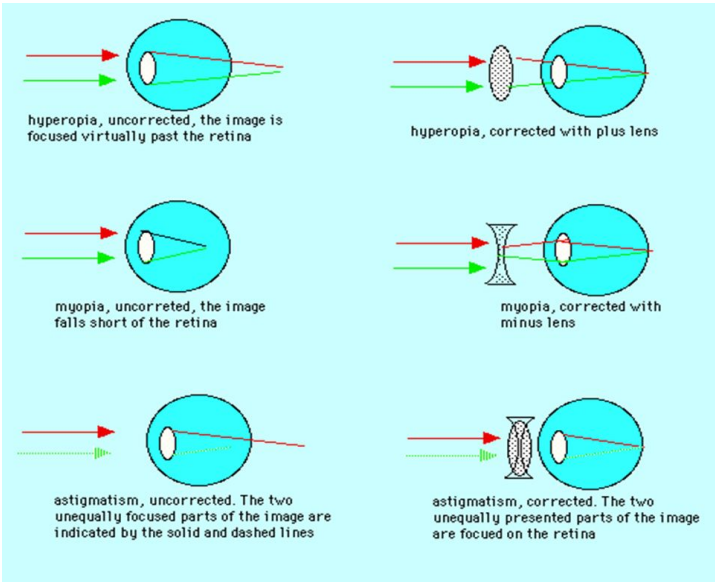
Correction of Refractive Errors

- **Spectacle lenses "Glasses":** most common
  - Monofocal lenses لها قوة وحدة: spherical lenses for myopia and hyperopia, cylindrical lenses for astigmatism
  - Multifocal lenses لها قوتين: for patients with: presbyopia + myopia/hyperopia.

First picture: Hyperopia
إما حجم العين صغير أو القوة ضعيفة، نحتاج عدسة قوية (+) | شكلها Biconvex
Second picture: Myopia
إما حجم العين كبير أو القوة قوية، نحتاج عدسة (-) نصعب عليها الأمور ونبعد الـ rays زيادة لأنها قوية | شكلها Biconcave
Third picture: Regular astigmatism corrected with cylindrical lens: has both (plus +, minus -)
باقي التفاصيل العميقة و الـ advanced يدرسونها الـ optometrists

Multifocal lenses
Has 2 lenses (powers) One for far vision (up) and the other for near vision (down).





To summarize the Tx: (1) Cylindrical lens → astigmatism +/- myopia or hyperopia **(2)** Biconcave lens → myopia (-) **(3)** Biconvex lens → to correct hyperopia (+)

- **Contact lenses.** common
- **Keratorefractive surgery:** last option,options: cornea (change shape) or intraocular to get rid of glasses

1. Cornea نقلل من سماكتها بطريقة محسوبة**: LASIK** ليزك (Laser-Assisted In-Situ Keratomileusis), **INTRALASIK**, **LASEK** لازيك (Laser Epithelial Keratomileusis). We remove part of the stroma (central part) using laser by evaporating tissue from corneal surface. **Video1** | **Video2** (not the same as presented by the doctor)

2. Intraocular: PRK (Photorefractive Keratectomy): another lens نشيل العدسة الطبيعية ونزرع بدالها
"أو نزرع عدسة أمام العدسة الطبيعية (نربط العدسة بالـ iris) بدل ما أحط العدسة (نظارة أو عدسة لاصقة) أمام العين أحطها في العين"

Clinical Scenario

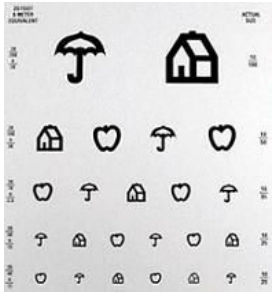
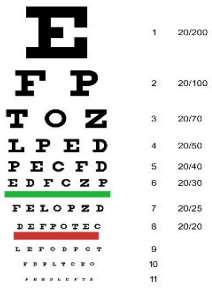
Scenario: 14 year-old boy complaining of difficulty seeing the blackboard (common presentation)
- Visual acuity (VA): 20/200 (first thing to do)
- PH (Pin hole):
  - **Improve: (1)** to **20/20** → Refractive errors | **(2)** to **20/80** → both. (RE & other cause)
  - **No** improvement → Other causes: cataract, optic nerve disease..etc

Extra from 435 team

- The refractive index of a medium is defined as the ratio of the phase velocity of a wave light in a reference medium to its velocity in the medium itself.
- To have a clear picture in the retina & to be seen in the brain, there should be a clear cornea, clear anterior chamber, and clear lens, clear vitreous cavity then the picture should be focused on the retina with normal refractive index.
- The retina is responsible for the perception of light. It converts light rays into impulses; sent through the optic nerve to your brain, where they are recognized as images.
- Power of accommodation is {(15 - age)/4} of the lens. accommodation is strong in children.
- All three types of ametropia can be corrected by spectacle lenses . These diverge the rays in myopia, converge the rays in hypermetropia, and correct for the non - spherical shape of the cornea in astigmatism.
- Japanese tend to have myopia more due to their crowded narrow surroundings which requires excessive accommodation.
- Symptoms of hyperopia: Accommodative esotropia: because accommodation is linked to convergence leading to easotropia..
- It should be noted that in hypermetropia, accommodative effort will bring distant objects into focus by increasing the power of the lens. This will use up the accommodative reserve for near objects.
- Astigmatism is a common and generally treatable imperfection in the curvature (mismatched curves) of the eye that causes blurred distance and near vision. It's the worst in the quality of vision.
- In astigmatism, surface of cornea is not homogenous. Usually it is congenital.
- Regular astigmatism: (2 meridians) power and orientation of principal meridians are constant. The principal meridians are 90 degrees apart (perpendicular to each other). With the rule astigmatism, Against the rule astigmatism, Oblique astigmatism.
- Irregular astigmatism: (different meridians > 2) power and orientation of principal meridians change across the pupil. The principal meridians are not perpendicular.
- **Anisometropia:** (not mentioned by the doctor or slides)

* Anisometropia is the condition in which the two eyes have unequal refractive power. Generally a difference in power of two diopters or more is the accepted threshold to label the condition anisometropia. the image of an object in one eye differs in size or shape from the image of the same object in the other eye.
* More than 3 diopters difference if not detected in pediatrics and corrected it can cause unilateral amblyopia "in the weaker eye".
* Individuals can tolerate up to 2-3 Diopters of anisometropia before becoming symptomatic.
* If the difference between 2 eyes: (D=diopter), > 3D → glasses | > 3 but < 7 → contact lenses | > 7D → surgery.

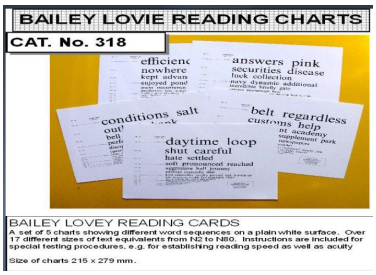
Causes: Correction of a refractive error Anisometropia, Antimetropia (being myopic (nearsighted) in one eye and hyperopic (farsighted) in the other.), Meridional aniseikonia occurs when these refractive differences only occur in one meridian (see astigmatism), Refractive surgery.

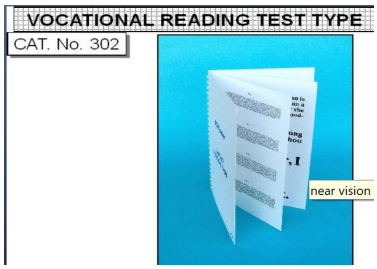
Allen chart (age 3 – 6 years)	**Snellen chart** (more than 6 years)
	

- **Notes for testing vision & VA:**

* In the first 2 months of life: do light objection test (if the baby objecting or closing the eye in response to light it means he/she is seeing)
* From 2 months – 3 years: do follow and fixate test. At this age, babies will start to follow the objects, so bring a toy in front of them and do the test. (If following the toy → good vision). OR you can do (central= seeing centrally. Steady= no nystagmus. Maintained= baby is following object & after blinking he/she continues following the same object)
* The vision maturation is acquired skill for the brain, so babies when they're first born they will be legally blind.

* The axial length of the eye will grow quickly in the first 6 months. So if anything stops the growing they will have amblyopia (lazy eye) E.g: vitreous hemorrhage, congenital cataract.
*** Legal blindness:** if the vision in the best eye is w/ best correction and providing less than 20/200, this is considered legal blindness. (patient needs assistance). The criteria used to determine eligibility for government disability benefits and which do not necessarily indicate a person's ability to function. In the US, the criteria for legal blindness are: * Visual acuity of 20/200 or worse in the better eye with corrective lenses. * Visual field restriction to 20 degrees diameter or less (tunnel vision) in the better eye. Note that the definition of legal blindness differs from country to country.
*** Testing near visual acuity:** It is done at a standard working distance ~ 30-40 cm. A variety of charts are available:





* - To assess the effect of pathology on VA. You must eliminate the effect of refractive error. This is achieved by measuring: the patient's best spectacle correction or viewing the test chart through a pinhole.
- Contact lenses:
* Higher quality of optical image and less influence on the size of retinal image than spectacle lenses
* Indication:cosmetic,athletic activities,occupational,irregular corneal astigmatism,high anisometropia & corneal disease
* Disadvantages: careful daily cleaning and disinfection.
* Complications: infectious keratitis, giant papillary conjunctivitis, corneal vascularization, and severe chronic conjunctivitis.
- Refractive surgery – flattens corneal surface (more successful because it's easier to flatten than to make it more convex) for myopia or increases its curvature in Hyperopia.
- Improves unaided visual acuity but may have complications.
- Intraocular surgery: for high power للي نقصهم مرة عالي, نشيل العدسة بكبرها ونحط جديدة أو نزرع عدسة قدام العدسة الطبيعية

- Give best optical correction for aphakia; avoid significant magnification and distortion caused by spectacle lenses.
- Clear lens extraction(with or without IOL)
- Phakic IOL (intraocular lenses): lenses made of plastic or silicone that are implanted into the eye permanently to reduce a person's need for glasses or contact lenses.
- One of the side effects of intraocular lens procedure => loss of accommodation.

	Photorefractive keratectomy (PRK)	Laser-assisted-in-situ Keratomileusis (LASIK)
Flap	No flap. We just remove the epithelium apply laser then the epithelium will grow.	Thin flap.
Advantage	Safer on the long run.	Immediate 20/20 vision, no pain, good visual rehabilitation, can correct high numbers (up to-80)
Disadvantage	Severe pain for 1 week, blurred vision for 2-3 weeks	Severe trauma, the flap can fall down

Chronic Visual Loss

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

Notes:

- Don't skip any picture it is very important for exam
- Yellow highlights are Exam Question.
- Doctor notes are very helpful for better understanding.
- In exam it is a MUST to have picture of:
  - Glaucoma "cupping"
  - Diabetic Retinopathy DR
  - Pallor of Disc

Done by: Afnan AlMalki.
Revised by: Rawan Aldhuwayhi
Resources: Slides + 435 team + Notes + ophthobook.

INTRODUCTION TO CHRONIC VISUAL LOSS

Definition: Slowly progressive painless visual loss.

Vision: So how can we assess the vision?
- Quantity: VA (Visual acuity)
- Quality: VF (Visual Field), clarity of vision, color vision

لما نتكلم عن البصر و الرؤية بشكل عام هو مو بس شيء نقيسه بالأرقام هذا مفهوم خاطئ فيه ناس نظرهم بالأرقام سليم كامل سته على سته لكن عندهم مشاكل في نوعية النظر مثلا اللي عندهم عمى الألوان أو الاشخاص اللي يشوفون الشكل ويقدرون يتعرفون عليه مثلا يعرفون ان هذا مربع بس عليه غباش او مثلا يقدرون يشوفون الصورة بالوسط بس حوافها مايشون زين فمهم جدا نقيس النظر من وجهين النوع والكم

Causes:

Always be systematic

1. **Refractive** the most common
2. Cornea
3. Lens
4. Vitreous
5. Retina
6. Optic Nerve
7. Neurologic

Now let's discuss each of them one by one

1.REFRACTIVE

Occurrence:
- Mostly in young patients

Causes:
- Myopia, hyperopia or astigmatism.
- Amblyopia: كسل العين
  - → Patient who developed refractive error earlier in life and they don't treat it they might develop Amblyopia. Simply: brain tends to ignore the weak eye, brain will be confused, thus can't fuse images

Myopia: image will be anterior to Retina. So, why they are wearing negative glasses? it will bring the image to the Retina. What is the cause of myopia? check Refractive error lecture
Hyperopia: image will be posterior to Retina. Opposite to myopia
Astigmatisms: اللابؤرية في هذه الحالة بدل أن يتجمع الضوء الداخل إلى العين في نقطة واحدة واقعة على الشبكية فإنه يتشتت ويتجمع في عدة نقاط مسببا تشويشًا غير منتظم للرؤية ، سبب هذه الحالة عدم تساوي تحدب القرنية

Signs:
- Normal exam (however Sometimes patient with astigmatisms have **corneal scar** in examination)
- Refraction needed to show errors (everything will be normal except for visual acuity)

Treatment:
- Glasses, Contact Lenses, Refractive surgery (lesser or PRK)
  - NB: lenticular causes needs cataract surgery.

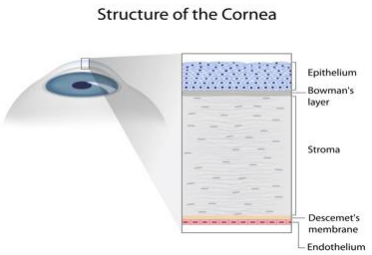
2.CORNEA

"Cornea is Avascular tissue"

Cause:

- Scar: trauma, infection
- Hereditary: corneal dystrophies, keratoconus القرنية المخروطية

Why there is scar whenever you have trauma or infection? Because the collagen fibber intercalated with each other instead of being parallel it will cross each other causing scaring.
We suppress the scarring event by **Steroid** اغلب عمليات العيون يعطونهم ستيرويد بعد العملية لان الجراحة بحد ذاتها تعتبر تروما!=عشان كذا



Signs:

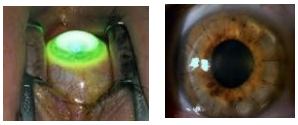
- corneal scar, bulging corneal, stromal opacities. Might have some conjunctival injection with chronicity

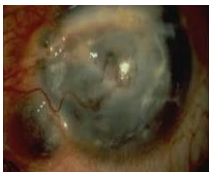
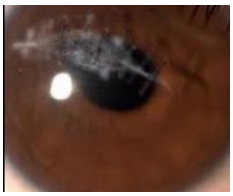
Treatment:

- Refraction, Contact lens (soft or hard), corneal cross linking, keratoplasty زراعة القرنية

You will treat the underlying conditions. If you have patient with corneal dystrophy or significant corneal scar→ simply replaces the scars corneal tissue with another tissue = keratoplasty "multiple options"
- Penetrating keratoplasty: ناخذ القرنية كاملة ونزرعها
- Lamellar keratoplasty: Corneal Stroma and epithelium transplant and leave the Descemet's

وظيفة الاندوثيليال سيل تعمل زي المضخه تعمل انها تخلي الستروما نظيفة مافيها فلويد عشان تكون القرنية شفافة خالية من الشوائب
تخيلوها نفس قزاز السيارة لما يجي مطر مانشوف ونحاول ننظفه بالمساحة عشان تتضح الرؤية *الدكتور قال لاتدخلون بتفاصيل العمليات*

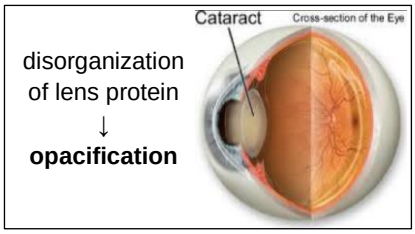


Keratoconus • What is the sign? Munson's sign is a V-shaped indentation observed in the lower eyelid when the patient's gaze is directed downwards بس خلي يطالع تحت وبتشوف ذا الشكل Develop In advance keratoconus • Can patient with keratoconus develop scar? Yes, the cornea is coning anteriorly so once its cones anteriorly you know that you are having a layer within the cornea called Descemet's membranes once this break the aqueous will go within the corneal stoma and will accumulate there→with chronicity it will end up with scaring	
Chronic inflammatory process →chronic vascularization→scaring = Stromal opacities • Usually the cornea is Avascular tissue so once you have vascularity reaching the cornea you will have leakage of inflammatory mediators→scaring and lipid deposit	
Corneal scar • Penetrating globe injury →underwent primary repair→suturing the wound → ended up by corneal scar • this patient has trauma penetrating globe injury the scaring isn't the issue here you need to maintain the integrity of globe ○ لأن فيه احتمال لو ماتخيط زين ممكن يفتح وندخل في مشاكل الانفكشن مثل الاندوفثلمايتس اللي ممكن تنتهي بفقدان البصر	

3.LENS "CATARACT" الماء الأبيض

Pathophysiology:

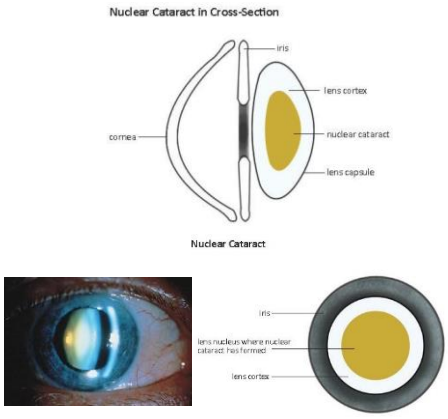
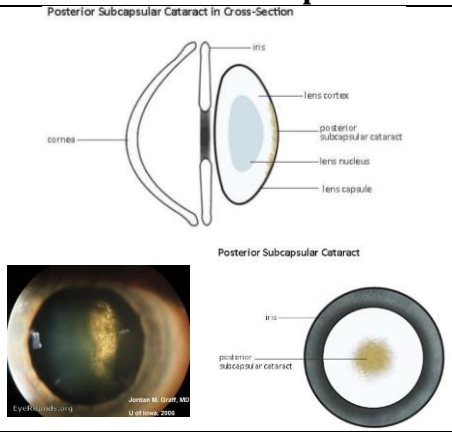
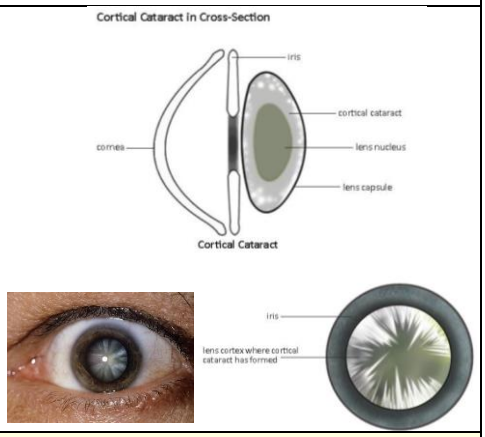
- At **age of 40 or more** the lens will loss its elasticity "less accommodation" because of disorganization of lens protein →**opacification** they will start to wear eyeglasses or complaints of loss of near vision
- Why do we have development of cataract, gray hair and wrinkling of skin start at the age of 40? Same embryological origin.



Cause:

- Age related the most common
- Metabolic DM
- Traumatic
- Congenital: Unilateral or bilateral You need to rule out another pediatric syndrome
- Drugs: **Steroid**(The problem not only develop posterior sub capsule cataract they will develop Glaucoma as well)
- Inflammation Uveitis
- Ocular:Patient with **Retinitis pigmentosa**

Clinical Classification:

<table>
<tr><th colspan="3">According to The Maturity</th></tr>
<tr><th>Immature</th><th>Mature</th><th>Hyper-Mature</th></tr>
<tr><td>• you can see the posterior pole "fundus"</td><td>• you will never see the posterior pole "fundus"
• The whole lens is opaque white
• The anterior chamber is shallow(narrow) →Risk of glaucoma, so do a prophylaxis which is iridotomy.</td><td>• The lens protein starts to leak through the capsule
• It is advance part of mature cataract
• causing open angle glaucoma</td></tr>
<tr><th colspan="3">According the Anatomic location *IMPORTANT*</th></tr>
<tr><th>Nuclear</th><th>Sub capsular</th><th>Cortical</th></tr>
<tr><td> 
</td><td> 
</td><td> 
</td></tr>
<tr><th colspan="3">According to the Age</th></tr>
<tr><th>Congenital</th><th>Infantile</th><th>Pre-senile & Senile</th></tr>
</table>

- Focus only in the first two classification!

Clinically:

- Painless loss of vision gradual onset
- Visual acuity: worsening of existing myopia, correction of hyperopia
  - → Especially patient with **nuclear sclerosis**, why? Because refractive power of lens will increase, and the image will become more anterior to Retina→worsen of myopia and correction of hyperopia بالمختصر المايوب مشكلتهم الصورة قدام الشبكية فبتزيد الصورة بتتقدم اما الهايبروب مشكلتهم الصورة ورا الشبكية فلما تتقدم الصورة بيكون ذا الحل لهم تقدمت ورجعت للشبكية مكانها الطبيعي
- Loss of contrast sensitivity in low light مايقدرون يفرقون بين درجات اللون الواحد مثلا الأزرق يشوفونه لون واحد مايقدرون يميزون السماوي الكحلي
- Glare in bright light (scatter of light): Especially in posterior subcapsular cataract. most of the time this is the complain of patient who drive at the night.

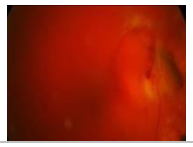
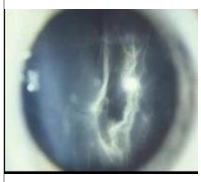
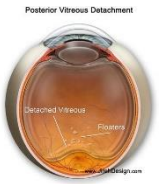
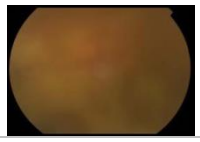
Treatment:

Medical Treatment isn't effective! So Simply you have a cataract you need to remove it by extraction

- **Congenital:** lens aspiration ± IOL (intraocular lens) remove the optical lens→replace it with new one and keep the capsule
- **Acquired:**
  - ECCE(Extracapsular cataract extraction) + PCIOL(posterior chamber intraocular lens): "very severe cataract"
  - or Phaco (Phacoemulsification) + PCIOL
    Phaco is modified ECCE: small opening and putting a foldable lens.

4.VITREOUS

Causes:

○ **Vitreous hemorrhages:** → Causes: trauma, PDR(Proliferative diabetic Retinopathy)DM is the most common cause, uveitis, PR	
○ **Vitreous condensation, opacification:** → Causes: Chronic inflammation, Other cause is posterior vitreous detachment (PVD),the vitreous is a jelly like transparent structure once it detached from its insertion it will shrink and cause condensation. sometimes it will affect the vision.	 
○ **Vitritis:** → Causes: uveitis (Pic: Vitreous haze because of inflammation)	

Treatment:

- Treat the underlying causes
- Most of the time you will do vitrectomy= removal of vitreous

5.GLAUCOMA الماء الأزرق أو السويرق

The significance: video

- **Second** leading cause of blindness! Early diagnosis is crucial to prevent loss of vision
- Why it is important? Because it's common, cause irreversible blindness and Silent disease

ليه اسمه الماء الأزرق هل لأن العين زرقاء؟ ممكن لكن هذا فقط اذا كانت من الولادة الأصح بأن المرضى المصابين يرون حلقات زرقاء على الضوء

Signs:

- **High IOP** + Characteristic **optic nerve head changes** + visual field loss secondary to nerve fiber layer loss + Gonioscopy: open or closed angle "will be discussed next page"

Symptoms:

- Initially Asymptomatic.usually pts will come in later stages when they lose their sight or incidentally by follow up. مهم تفهمون ان ارتفاع ضغط العين ممكن يجي بألم شديد جدا بالعين وحواليها ومعه صداع وممكن يجي بدون أعراض وللان محد يعرف السبب بس لو فيه ألم بالعكس جدا مهم لي كطبيب بيكون منذر لي عشان انتبه وابدا علاج لكن المصيبة اللي مايحسون بشيء أبدا!
- Usually detected on routine examination
- Glaucoma starts with **peripheral (navigational) vision involvement.** No central vision problem that why Most of the patient comes late!

NORMAL VISION | EARLY GLAUCOMA | ADVANCED GLAUCOMA

Risk factors:

- Age, **Family history,** IOP , Medication "Steroids" , DM / HTN and **Myopia**
  - IOP is the single factor to be controlled. Normal is 10-21mmHg

Aqueous humerus: video

Active secretion:
1- Na/K ATPase.
2- Cl secretion.
3- Carbonic anhydrase.

Passive secretion:
1- Ultrafiltration.
2- Diffusion

Optic Nerve Head (ONH) complex evaluation:

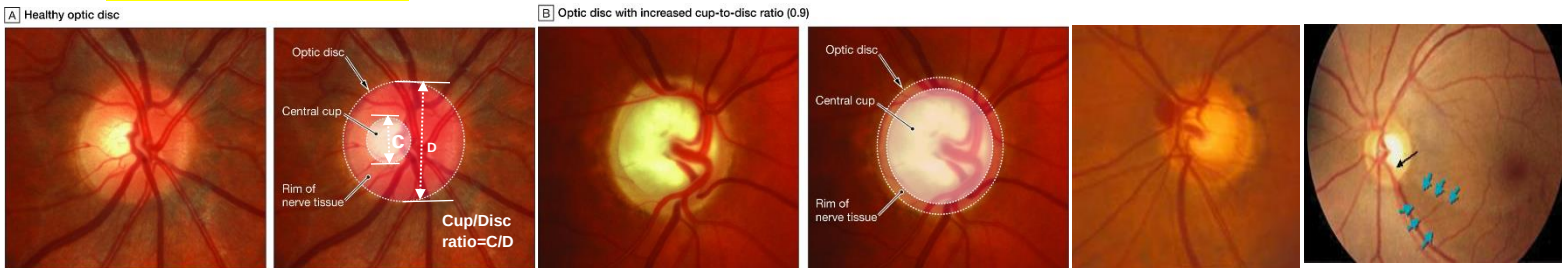
In clinic do detailed optic nerve exam because you might have:

- glaucomatous optic nerve head damage
- Anomalous disc
- Disc pallor because of CNS or DM

What to evaluate:

- Disc margin and disc diameter
- **Cup/disc ratio:**
  - How to estimate it? Take the vertical ratio if more than 0.3 we should worry
  - normal value is 0.3, bigger cup=more nerve tissue loss! Cause for large cups: glaucoma
- Neuroretinal rim: area of axon
- Disc size
- PPA: peripapillary atrophy
- NFL defect (nerve fiber layer)
- Optic disc hemorrhage

Empty central part = cup بجيب لكم بالاختبار صورة للجلكوما ركزوا بالفايندينق



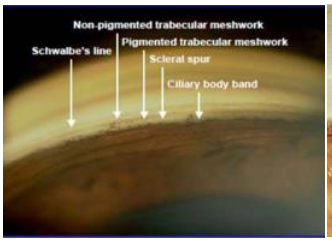
Normal optic disc | Notice: increase cupping, thinning of neuroretinal rim | Optic disc hemorrhage | Blue arrows: wedge shape NFL loss

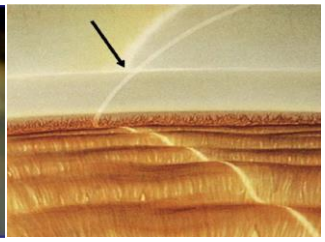
Gonioscopy or zeiss gonioscopy lens:

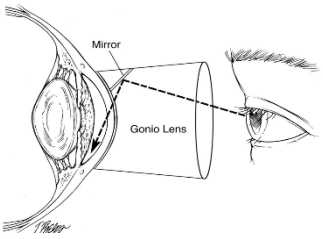
Why we use gonioscopy We need to check the angel between the iris and cornea **to classified Glaucoma** either open angel or closed.

Norma Angle structures: "احفظو ها"

- Schwalbe's line (**SL**)
- Trabicular meshwork (**TM**)
  - Non pigmented
  - Pigmented
- Sclera spur (**SS**)
- Ciliary body

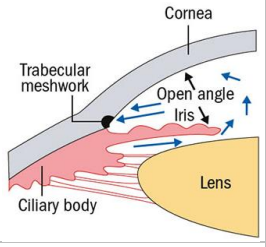
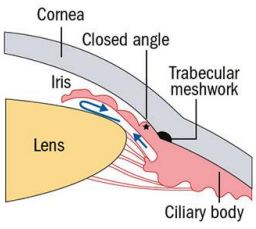
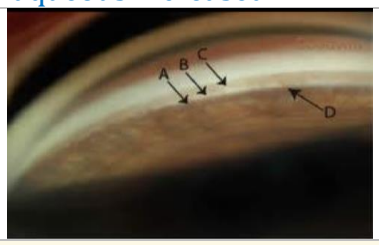
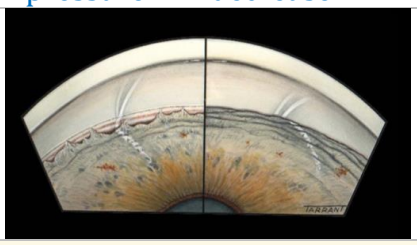






Classification of glaucoma:

It is important to know the classification because different management

According to the angle: It can be: open, closed or combined mechanism	
Open = Iris Not covering TM	**Closed = Iris covering TM**
In open angel you can see the angel structure, The problem with the pores in the TM التيبركولر ميشورك شبكه فيها فتحات يطلع من الاكويس فلويد , بالاوين انقل جلوكوما مشكلتنا مع الفتحات انها انسدت فماعاد تلطع السوائل, ايش ممكن السبب ممكن مع التقدم بالعمر اومع استخدام الستيرويد بيصير فيه ترسب لماده امينوقلوقلايكانن فتسد البورز 	You will not able to see the angel structures unless you press the lens the structures will  Mcq's or SAQ:in **peripheral anterior synechiae** there will be adhesion between iris and angle
Laser Rx: SLT= selective lesser trabeculoplasty→ burns TM→ capability of drainage of aqueous increased.	**Laser Rx**: PI = peripheral iridotomy→laser opening behind the iris → aqueous goes behind the iris→pressure will decrease.
	
According to the etiology:	
Primary	**Secondary**
No detectable reason and often bilateral	Predisposing factor and often unilateral

Treatment:

Treatment is aimed at reducing intraocular pressure by 3 modalities available

1. Antiglaucoma medications
2. Laser treatment: discussed above according to the angle
3. If no improvement do surgery, Surgical treatment either:
   a. incisional: trabeculoplasty, iridotomy, Canaloplasty and Ahmad implant
   b. Non incisional

6.MACULAR DEGENERATION(MD)

AGE RELATED MACULAR DEGENERATION

Introduction:

- Impaired **central** vision, Peripheral vision preserved. Opposite to glaucoma
- Leading cause of **legal blindness**[1] in developed world.
- Classical presentation of those patient: Many RTA Or he will till you I cannot park my car

Symptom: احفظوها زين

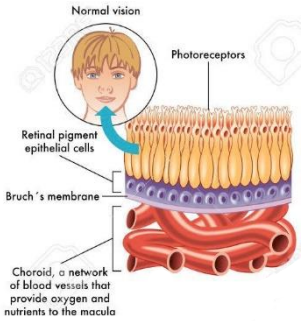
- Metamorphopsia: distorted vision الرؤيه مطعوجة كأني أشوف اللي قدامي مكسر
- Micropsia: reduction of size of objects
- Macropsia: enlargement of size of objects
- Scotoma: visual field loss

Etiology:

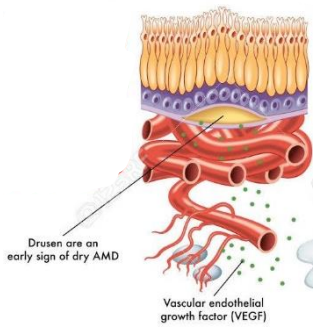
- **Multifactorial**: age, smoking, vascular disease, UV light, diet, and FHx.

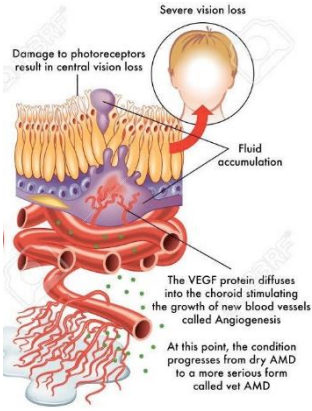
Pathogenesis:

- **Macular involvement:** أول شي نتعرف على ابطال القصة-الطبقات الي بتتأثر-
  - ✓ Outer retinal layer "photoreceptors"
  - ✓ Retinal pigment epithelium (RPE)
  - ✓ Bruch's membrane
  - ✓ Choriocapillaris



1	**Drusens** Drusens(yellowish discoloration):lipid products from photoreceptor outer segments, found under retina. ▪ **EXPLANATION:** photoreceptors will secret lipids ''Drusen" →accumulates under bruch's membrane, once the RPE can't clear them it will accumulate in larger amount → irregularity in RPE→ raising the choroid and the RPE→ could lead to: ✓ separation of photoreceptors→small object "micropsia" ✓ or Accumulations of Photoreceptors→Macropsia ○ With chronicity of disease the photoreceptors will Atrophied→start to loss vision ○ When there is area atrophied, area small objects and area large objects these called Metamorphopsia
2	**Neovascularization** MCQ: With ischemia new vessels from choroid grow into the subretinal space forming subretinal neovascular membrane or preaching the Bruch's membrane -any new blood vessels in abnormal place are ABNORMAL-
3	**Hemorrhage** -the new vessels are very fragile can bleed easily- ▪ New vessels is bad Either it continues to vitreous causing **vitreous hemorrhages** or it will bleed under the RPE causing sub **RPE hemorrhage**s or it will bleed under the internal limiting membrane and causes **subhyaloid hemorrhage**. ▪ Hemorrhage into subretinal space or even through the retina into the vitreous (significant loss of vision).

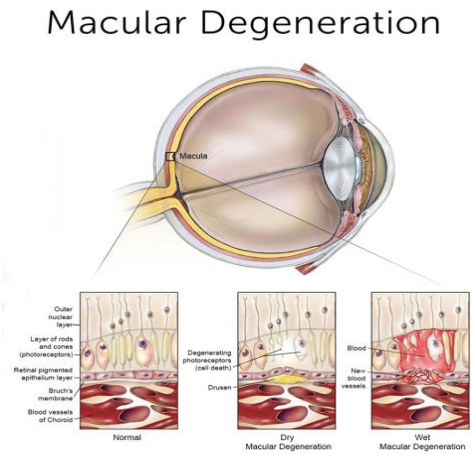


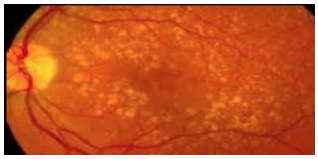
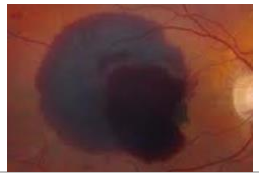
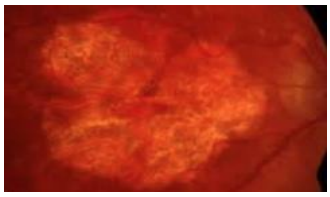
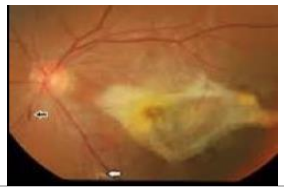


باختصار تجمعت الدهون تحت منعت التغذية و الدم صار فيه اسكيميا جاء الكوروويد فزع وسوا اوعية جديدة اللي احنا ما نبغاها لانها بسرعة بتنقطع من اتفه شيء وتدخلنا في مشاكل النزيف بالشبكية وفقدان البصربالذات لو جاء النزيف في الفيتروس

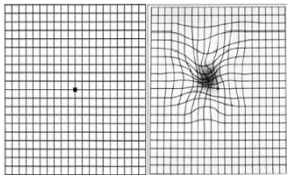
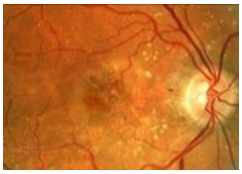
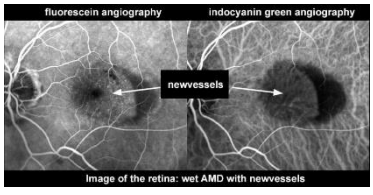
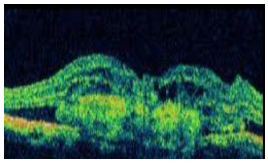
[1] When we call someone legally blind? If the Central visual acuity of 20/200 or less

Types:



Atrophic = dry	Exudative = wet
Most benign, often asymptomatic. Gradual over years	Rapidly progressive (weeks)
Signs: • Drusen • Geographic atrophy • Photoreceptor degeneration • scotoma when light adapting	**Signs:** • Choroidal (subretinal) neovascularization • Preretinal hemorrhage • Elevation of retina • Subretinal fibrosis • Metamorphopsia • Central scotoma[2] pic→ 
Drusen With time will causes RPE atrophy 	**Preretinal hemorrhage 2 hemorrhages**: ○ grayish: under RPE ○ Bright:above RBE الدكتور يقول لو جتكم بالاختبار الي تحلها صح تستاهل ميه على طول:) 
Geographic atrophy: Once you lost your photoreceptors that lead to this Disappearance 	**Subretinal fibrosis:** The yellowish structures is fibrosis replaced the photoreceptors لكم ان تتخيلو اذا تبدلت الفوتوريسبتور بالفايبروزيز ايش ممكن يصير اكيد بيروح النظر فيها 
What's the difference between dry and wet age-related macular degeneration? Dry ARMD is when you have drusen and macular RPE atrophy. Wet ARMD implies choroidal neovascularization that has grown up through Bruch's membrane and bleed into the retina. "Wet" essentially means "bloody" in this instance.	

Diagnosis:

Visual acuity	Amsler grid	Ophthalmoscopy	Fluorescein angiography	ICG (Indocyan Green)	OCT(Optical Coher Tomography)
	 If the patient saw wavy lines, then the macula is abnormal	 Pic:discret scattered yellowish subretinal drusens "dry MD"	 inject IV fluorescein\or ICG to visualize the retinal vessels.		 OCT shows subretinal fibrosis and hemorrhages

[2] Irreversible visual loss , same as glaucoma but MD less common

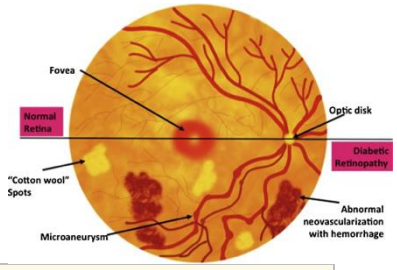
Treatment:

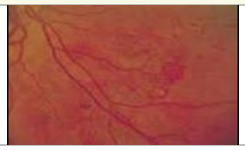
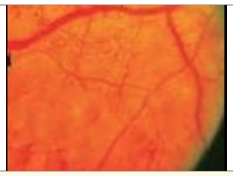
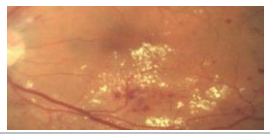
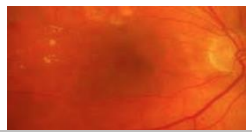
- If dry:
  - **Lifestyle**: Stop smoking, reduce UV exposure, Zinc & antioxidants
  - Low-vision aid
  - Monitoring with Amsler chart
- If advanced, you cannot do anything there is already fibrous tissue:
  - Observation
  - Laser photocoagulation, for neovascular membrane especially for the wet type.
  - Anti - VEGF agents. Wet type
  - You don't need to know this: Verteporfin photodynamic therapy (PDT): injection of photosensitizer into systemic circulation followed immediately by laser targeting new vessels in macular area

7.DIABTIC RETINOPATHY ★

Pathophysiology:

- **Microangiopathy**[3] which involves precapillary arterioles, capillaries and postcapillary venule. Have 2 mechanisms:
  1. Microvascular occlusion
  2. Microvascular leakage



1.Microvascular occlusion	
A. **Thick capillary basement membrane.** Mean lumen is smaller. Picture show: Abnormal blood vessels like fan new vascularization "NV"	
B. **Capillary endothelial cell damage** [4][5] **→Retinal ischemia**[6] **→AV shunt and NV**	
C. Change in blood cell	
2.Microvascular leakage	
Loss of pericytes between endothelial cells→ leakage into retina →exudates and edema	
Exudate of lipid in Retina 	Retinal Edema 

Risk factor:

Duration, Pregnancy, Nephropathy, Poor metabolic control , Smoking, HTN, Obesity and hyperlipidemia

[3] Microangiopathy= small blood vessels. Most if it presents in Retina, kidney, heart ,and the brain عشان كذا اغلب مضاعفات السكر تلقونها هنا
[4] Mean lumen narrower and narrower
[5] increased viscosity of RBC > clot formation > lumen closed > ischemia
[6] **Abnormal new vascularization because of vascular endothelial growth factor from ischemic tissue, this new vessels is fragile and easily bleed and causes fibrosis**

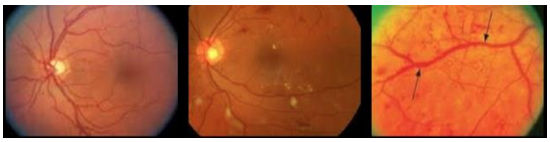
Clinical classification: بجيب منها في الاختبار

Non proliferative diabetic retinopathy :

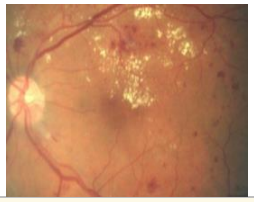
1. Non proliferative DR: mild , moderate and severe.

Pic show macular Edema and the arrow show ischemia

- Asymptomatic
- Decreased visual acuity : Clinical significant macular edema & macular Ischemia



Hemorrhagic spots



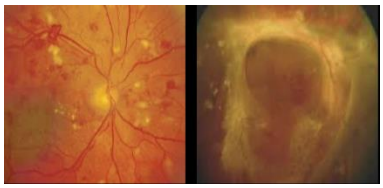
florican angiography for a very ischemic Retina "you cannot see the small blood vessels"

Proliferative diabetic retinopathy :

2. Proliferative DR : early and advance

Treat as soon as possible!

- Symptomatic
- Can causes macular edema and or ischemia



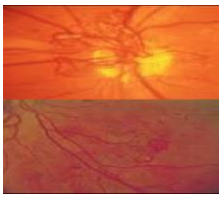
A. Neovascularization

a. **NVD: neovascularization of the disc** pic above

واضحة جدا تشوفون اوعية كثير متشابكة وتكون حول الديسك

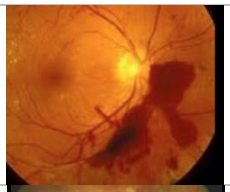
b. **NVE: neovascularization elsewhere** pic below

تشوفون اوعية كثير متشابكة بعيد عن الديسك



B. Fragile (intra-retinal or vitreous hemorrhage)

tractional retinal detachment[7]



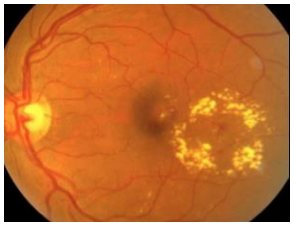
C. Associated with fibrous proliferation TRD

Fibrous tissue

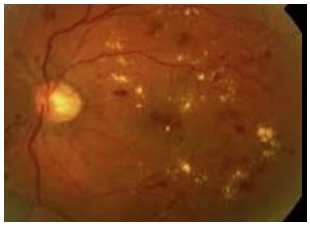
Diabetic Macular Edema:

- Retinal edema threatening or involving the macula
- Evaluate: location of retinal thickening relative to the fovea and the presence and location of exudates

FOCAL Clinical significant macular edema: ring pattern and leaking in center.
Treat. focal lesser



Diffuse hemorrhages and exudate all over no specific pattern
Treat. Grid lesser



[7] Recall the types of RD:
- **Rhegmatogenous** RD: you have break ثقب صغير يدخل من خلاله الفلويد ويفصل الريتنا
- **Tractional** RD"in "DR": you have fibrous tissue once its contract it will pulling the Retina
- **Exudative** RD: inflamed choroid layer -> accumulation of fluids under Retina -> detachment

Treatment:

- Laser
- intravitreal steroid injection
- intravitreal anti-VEGF injection
- Pars plana vitrectomy

8. RETINITIS PIGMENTOSA(RP) العشى الليلي-

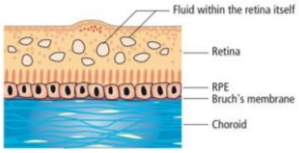
General characteristic:

- Group of genetic disorders affect the retina ability to respond to light يشوف بالنهار افضل
- **Symptoms** Slow loss of vision:
  - **Nyctalopia**:loss of night vision.
  - Tunnel vision[8] "loss of peripheral vision"
  - blindness
- Most are legally blind (Central visual field of less than 20 degrees) by 40s.
- **X linked recessive:** males: more often and more severe, females: carry the genes and experience vision loss less frequently
- Target photoreceptors and Associated with pigmentary changes in the RPE, which may be primary or secondary to the photoreceptor loss.

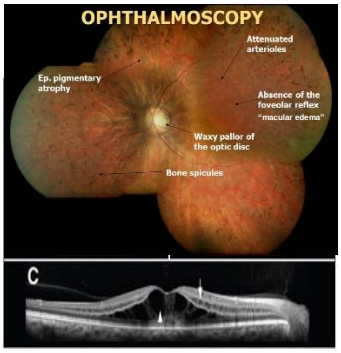
Rods important for night vision and cons in day vision but it will affect both of them

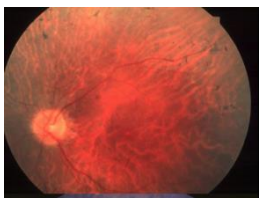
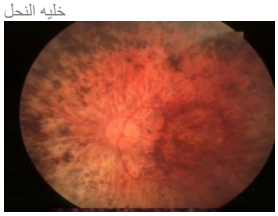
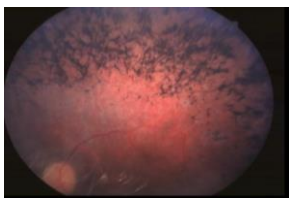
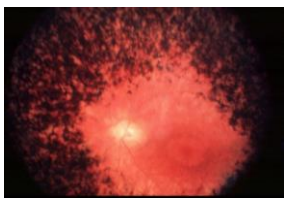
Signs:

- Visual acuity varies from :20/20 to no light perception at all.
- +-APD (afferent pupillary defect)
- PSCC (posterior sub capsular cataract)
- RPE hyperpigmentation **(bone spicules)** alternate with atrophic regions.
- **Attenuation of the arterioles** "thinning".
- **Waxy pallor** of the optic nerve head.
- cystoid macular edema (severe cases of RP) Extra pic→.



EXTRS PICTURES



RP stages and signs			
Early stage	**2nd stage**	**3rd stage**	**4th stage severe**
faint black spots in periphery " bone Spicules"	• bone spicules Increased more and more • Start to loss photoreceptors • Waxy disc appearance نفس لون الشمع حق خليه النحل	• more loss of photoreceptor • More clumping of RPE	• Severe photoreceptors loss • Tunnel vision • Severe RPE loss and clumping • Bone spicules • Waxy pallor
			

[8] Glaucoma have high IOP but in RP not

Investigation:

- VF test tunnel
- Color testing (mild blue-yellow axis color defects)
- Dark adaptation study (reduced contrast sensitivity relative to VA)
- Genetic subtyping
- OCT (CME) **Very important to check for cystoid macular edema**
- FFA florican angiography
- ERG Electroretinography is an eye test that detects function of the retina including photoreceptors
- EOG Electrooculography to record eye movement

Systemic Associated:*ارجعولها* محمه للي بيتخصص منكم بيديا او نيورو او حتى ميدسن

- **Hearing loss and RP**:
  - Usher syndrome (MOST IMP THING FOR US) Usher syndrome is a condition characterized by partial or total hearing loss and RP.
  - Alport syndrome is a genetic condition characterized by RP, kidney disease, and hearing loss.
  - Refsum disease is an autosomal recessive neurological disease that results in the over-accumulation of phytanic acid in cells and tissues.

- **Kearns-Sayre syndrome**:
  - External ophthalmoplegia مايقدر يحرك عينه
  - Lid ptosis
  - Heart block
  - Pigmentary retinopathy

- **Abetalipoproteinemia** is a disorder that interferes with the normal absorption of fat and fat-soluble vitamins (↓vitamin A →RP)
- **Mucopolysaccharidoses**
- **Bardet-Biedl syndrome** genetic disorder characterized principally by obesity, RP, pigmentosa, hypogonadism, and kidney failure in some cases
- **Neuronal ceroid lipofuscinosis** lysosomal storage disorders characterized by dementia , RP, and epilepsy .

Treatment:

Unfortunately, nothing can be done to prevent the progression of the disease, associated ocular problems can be treated:

- CAI: CME "Carbonic anhydrase inhibitor work will in patient with RP and CME"
- Vitamins
- Cataract: surgery
- Low vision aids
- Gene therapy!! خز عبلات

- THE END -

Extra
What's the difference between a PCO and a PSC cataract? • PCO: posterior capsular opacification. This is an "after cataract" that forms on the back surface of the posterior capsule after successful cataract surgery. This opacity can be cleared with a YAG laser. • PSC: posterior subcapsular cataract. This is a cataract that forms on the back portion of the lens. These tend to occur more often in diabetics and those on steroids, and tend to be visually significant because of their posterior position.
What drops are given after a cataract surgery? Usually an antibiotic, such as ciprofloxacin or vigamox. Also, a steroid is given to decrease inflammation.
What retinal findings do you see with glaucoma? You see increased cupping of the optic disk, usually in a vertical pattern that goes against the ISNT rule. You can sometimes see hemorrhages at the disk and "undermining" of the blood vessels as they exit the disk.
What's the difference between open-angle and closed-angle glaucoma? How about chronic versus acute glaucoma? • Open angle is a common, chronic condition where aqueous drainage is impaired. • Closed-angle glaucoma is caused by acute closure of the iridocorneal angle leading to blockage of ALL aqueous drainage – an ophthalmologic emergency that can quickly lead to blindness.
What are the retinal signs of diabetic retinopathy. How do they compare to, say, hypertensive retinopath?. With diabetic retinopathy you typically see a lot of dot-blot hemorrhages, cotton-wool spots, and hard exudates. Hypertension usually has more flame hemorrhages and vascular changes such as arterial-venous nicking and copper/silver wiring.
How do we categorize diabetic retinopathy? As either NPDR (nonproliferative diabetic retinopathy) or PDR (proliferative diabetic retinopathy) depending upon the presence of neovascularization.

Neuro-ophthalmology

[**Color index:** Important | Notes: **F1**, **F2/A** | Extra] **EDITING FILE**

Objectives:

➢ Not given.

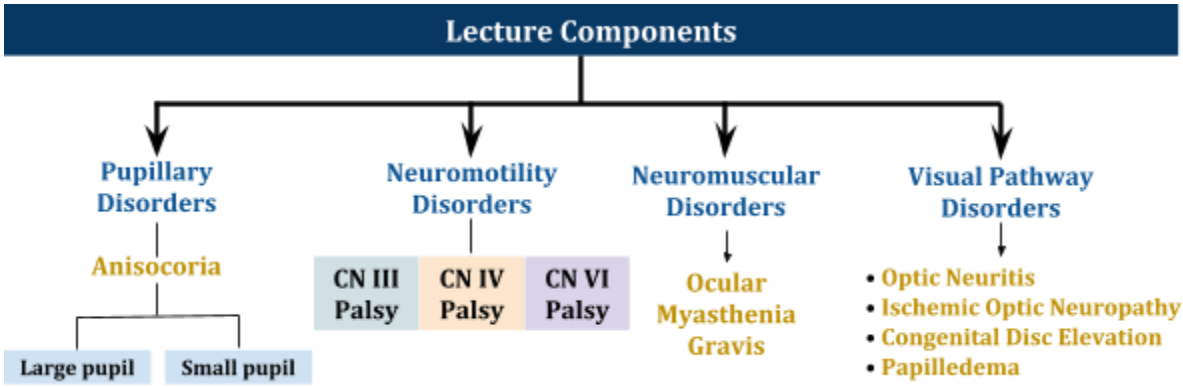
Done by: Luluh Alzeghayer, Samar AlOtaibi,
Edited & revised by: Lamya Alsaghan, Munerah AlOmari.
Resources: Slides (old version) + Notes + Lecture Notes of Ophthalmology + OphthoBook.

The secretory did not send the updated student version slides from Dr. Daniah Alshowaier despite repeated demands!

Last pages are extras!

Introduction

– **Neuro-ophthalmology** deals with visual problems that affect the optic nerve and its connection to the brain.
– Our eyes simply receive visual information and we actually see with our brain. In turn, the brain controls the position and focus of the eyes, directing our visual attention.

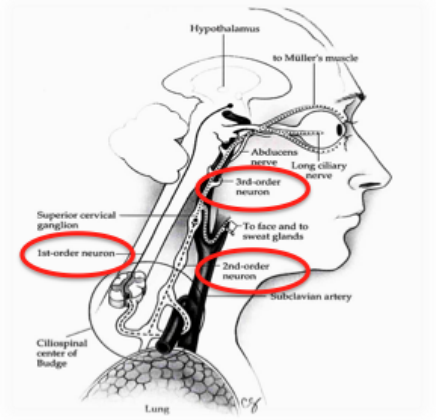


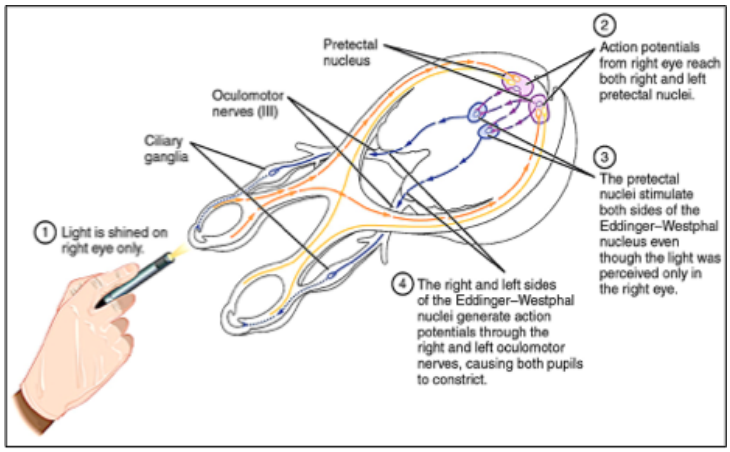
Pupillary Disorders

• Anatomy & physiology

– Pupil size is controlled by a balance between:
  ○ **Parasympathetic** with illumination (miosis: pupil constriction by sphincter muscles)
  ○ **Sympathetic** innervation in the dark (mydriasis: pupil dilation by iris dilator muscles.)
– Pupil (reacts) constricts to: Light | **(2)** Near stimuli: triad of: accommodation, convergence & miosis. This is why on examination we ask the patient to look away to eliminate the near reflex.

Sympathetic (adrenergic) pathway	Parasympathetic (cholinergic) pathway
very important – **The first-order neuron** descends from the **hypothalamus** to the first synapse, which is located in the cervical spinal cord (levels C8–T2, also called ciliospinal nucleus of Budge). **– The second-order neuron** travels through the brachial plexus, over the lung apex (that is why a tumor in the apex of the lung "Pancoast tumor" lead to horner's syndrome -or any cut of the sympathetic pathway-). It then ascends to the superior cervical ganglion located near the angle of the mandible and the **bifurcation of the common carotid artery**. **– The third-order neuron** then ascends within the **adventitia of the internal carotid artery,** through the cavernous sinus, where it is in close relation to the sixth cranial nerve. The oculosympathetic pathway then joins the ophthalmic (V1) division of the fifth cranial nerve (trigeminal nerve). In the orbit & the eye, the oculosympathetic fibers innervate the: ○ **Iris dilator muscle**: group of muscles in the peripheral 2/3 of the iris ○ **Müller's muscle**: a small smooth muscle in the eyelids responsible for a minor (around 2-3 mm) portion of the upper **lid elevation** (main eyelid muscle elevator is ***Levator palpebrae superioris***, supplied by 3rd nerve) ○ **Lower lid retractors**. Some patients might tell you they feel that their eyes are getting smaller and that is actually because the lowers lids are going back. This is called **[REVERSE PTOSIS].**	– Stimulation starts when you shine the light and that is important when you do the pupillary reflex. – When you generate action potential with the light stimulation, it will go back to the **Pretectal nucleus** then to the **Edinger-westphal nucleus bilaterally [important:** both nuclei will be innervated with the same amount that is coming from one eye (this is why shining light at one eye will constrict both pupils)**]** from there joining the parasympathetic fibers of the oculomotor nerve and it will go back to the eye causing miosis. Parasympathetic fibers are divided into **superior** and **inferior** division. Inferior division go to ciliary ganglia (parasympathetic ganglia) and finally reaching ***Sphincter pupillae*** **muscle to constrict the pupil**.





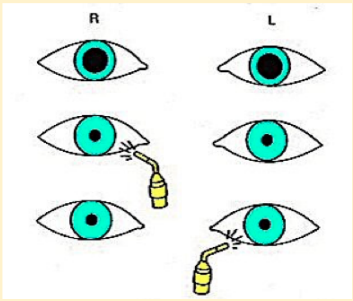
- **Examination of the pupil**

OSCE: pupil examination is done either with an ophthalmoscope or a penlight. Check this video

– Best conducted in dim light room using a bright light.
– The patient should be relaxed and fixing on a distant object to relieve/eliminate the near reflex.
– The **size, shape, position** of each pupil should be noted in **light & dark** conditions. equally round & symmetrical.
– Check light reflex looking for a relative afferent pupillary defect (RAPD).

1) Test light reflex: when shining light to one eye both pupils will constrict because it will stimulate both pretectal nuclei. When moving to the other eye normal reaction is either the same size or slightly constricted but never dilated. If there is dilation = relative afferent pupillary defect (RAPD): Pupils are equal and of normal size, but the response to light from affected side is reduced, while the near reflex is intact.

~ **Illuminated** eye → **direct** (reflex) constriction response.
~ **Other eye → consensual/ indirect** (reflex) response.

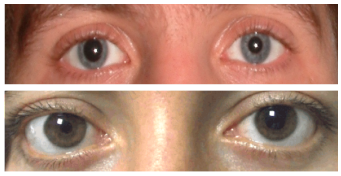


2) Swinging light reflex: looking for **(RAPD)** to assess the optic nerve function. Pupil **constricts (C) → dilates (D)** a fraction as the light passes over the nose → constricts again.

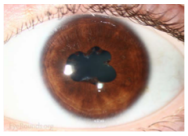
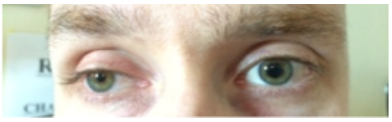
~ C → C → C → C = two normal eyes.
~ C → D → C → D = "**Marcus gunn pupil**" There will be constriction when shining light on the good eye. But, when crossing to the bad eye, both eyes seem to dilate a little. The bad eye still senses light and constricts but not as well.

+ Test near vision: accommodation → constriction.

- **Anisocoria** it is not the same as afferent pupillary defect (RAPD)! It is not related to the optic nerve



– **Anisocoria:** difference in pupil size. Always ask yourself which is affected? is it the large or small pupil? In order to answer that you need to examine the patient in **light & dark (dim light)** conditions, and measure in millimeter (mm)
– Small pupil does not dilate as well as the large pupil in dim light → **small pupil is abnormal.**
– Larger pupil does not constrict as well as the small pupil in response to a light stimulus → **large pupil is abnormal.**
– **Normal physiological anisocoria:** 20% of patients will have some difference between the size of pupils.
  - Difference should be **less than 1 mm** and **same amount in the dark and light** (important to examine the patient in both conditions).
  - **Hallmark: Intermittency or variability,** which is a reassuring sign. (with pediatric patients the mother says "sometime it's the left eye sometimes I can't differentiate, sometimes it goes to the other side")
  - With every patient take full history and do examination to rule out **sympathetic & parasympathetic lesions.**

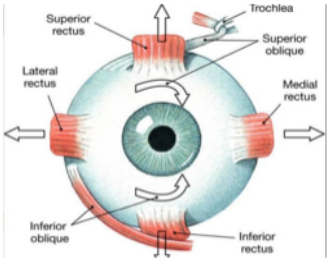
Large pupil is abnormal rule out by hx	**Small pupil is abnormal**
– Previous ocular surgery. – Ocular trauma. – Use of medications, like cycloplegics e.g. atropine, cyclopentolate. Sometimes we prescribe a drug to a child, the mother uses the drops, then touches her eyes. – Third nerve palsy. Fixed dilated fixed pupil, not responding to light (usually associated with severe ptosis, sometimes it covers the whole eye) will be discussed in details	– Previous ocular surgery. – Ocular trauma or inflammation. – Use of medication: ex.pilocarpine/ glaucoma medications **Picture:** small irregular pupil due to adhesions between the iris and the lens **(posterior synechiae)** caused by trauma or chronic inflammation. While adhesions between the iris and the cornea are termed (anterior synechiae). 
Tonic pupil (Adie's pupil)	**Horner's syndrome**
– Benign condition no need for further investigations. – Young female | Unilateral (80%). – Sluggish, segmental pupillary responses to light. – Better response to near (it will be intact) followed by slow redilation. Called **"light near dissociation"** – **Diagnosis:** instillation of weak cholinergic agents (0. 1% pilocarpine) will cause constriction (briskly) of the tonic pupil and will give you "reverse anisocoria" it will be even smaller than the one without any medication because of **(denervation hypersensitivity).** Normally there will be no changes. + slow, sustained miosis on accommodation. – **Holmes-Adie syndrome: (1)** Tonic pupil **(2)** Diminished deep tendon reflexes **(3)** Orthostatic hypotension. 	– **Includes** a triad of: **(1)** Small pupil (miosis) **(2)** Ptosis mild **(3)** Anhydrosis. + enophthalmos, heterochromia, loss of ciliospinal reflex. – **Causes:** lesion anywhere along the **sympathetic pathway** (central, para (peripheral), preganglionic, postganglionic). Carotid dissection, carotid aneurysm & tumor can be associated with Horner.  **Scenario:** 18-year-old female presented to the clinic saying "I noticed my right eye got smaller after neck surgery". Anisocoria wasn't clear in the light so we examined her in the dark. The difference was around 2 mm. She also had mild ptosis. There is also lower retraction. **What is the diagnosis? Horner's syndrome**

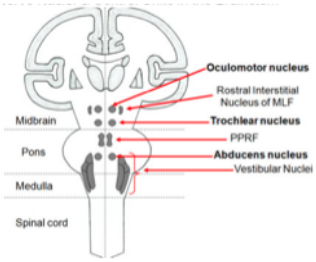
Most important life threatening cause that you can't miss, a young patient comes with history of trauma with **PAINFUL HORNER**, you have to rule out internal carotid artery dissection, the internal carotid is within the adventitia thus horner's may be the first sign. if you miss the diagnosis, patient might go into stroke shortly after. You need MRI/MRA.

Neuromotility Disorders

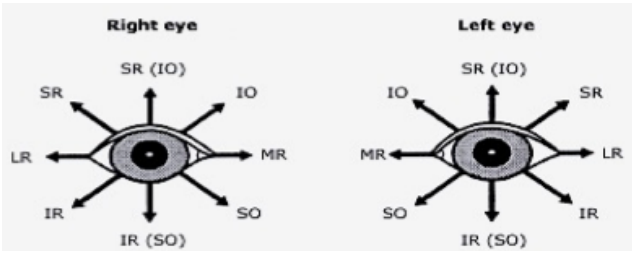
- **Anatomy & physiology** main nerve supplying extraocular muscles is oculomotor (third nerve)

EOM	Primary action	Innervation	Nucleus
Superior rectus	Elevation (maximal on lateral gaze)	Third cranial nerve, oculomotor	Midbrain
Inferior rectus	Depression (maximal on lateral gaze)	Third cranial nerve, oculomotor	
Medial rectus	Adduction	Third cranial nerve, oculomotor	
Inferior oblique	Excyclotorsion	Third cranial nerve, oculomotor	
Superior oblique	Incyclotorsion	Fourth cranial nerve, trochlear	
Lateral rectus	Abduction	Sixth cranial nerve, Abducens	Pons





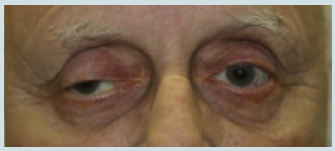
This picture is very important!



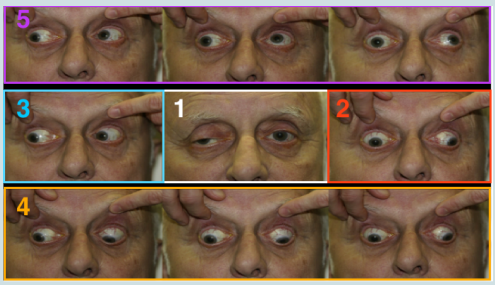
Keep this image in your head when examining **EOM movement**
SO: Down & In | IO: Up & Un | IR: Down | SR: Up | MR: In | LR: Out

Third nerve palsy

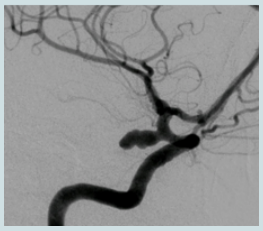
Case



65 years old presented to ER complaining of double vision.
Typical presentation of CN III palsy always keep it in your head!
Ptosis and Eyes are (down & out) pupillary dilatation & no accommodation.
The eye rests in a position of abduction, slight depression, and intorsion.



Next step: examination of extraocular movement:
1. Primary position: looking straight ahead.
2. Looking to his left: abnormal. | **3. Looking to his right:** normal.
4. Mild **infraduction** limitation. | **5.** Mild **spraduction** limitation.
Next step: check for pupillary involvement:
To differentiate between **surgical** (urgent, compression, pupil involved, needs neuroimaging) and **medical** (pupil sparing) third nerve palsy.Absence of pupillary involvement suggests a benign process that can be observed over a couple of weeks. A fixed, dilated pupil requires extensive neurologic evaluation



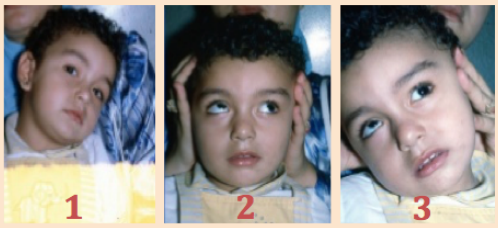
Diagnosis: posterior communicating artery aneurysm (life threatening)
(right internal carotid artery injection reveals a right posterior communicating artery aneurysm with a tubular configuration. A very small anterior communicating artery aneurysm is also identified) Magnetic resonance angiography (MRA) is the best investigation for PCA aneurysm
You always have to rule out aneurysm, why? Usually parasympathetic fibers goes on the outside (superficial) with CN III pathway so any compression will lead to CN III palsy with pupil involvement.

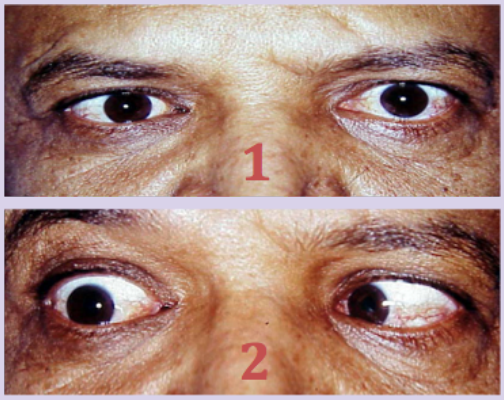
Anatomy & physiology

– Begins as a nucleus in the **midbrain** that consists of several subnuclei. Multiple nuclei
– Innervate:
- Most **extraocular muscles**: eye deviates **down & out**
- **Eyelids**: main lid retractor: Levator palpebrae superioris so paralysis will lead to severe ptosis
- **Pupils**: innervated by parasympathetic fibers which is not part of oculomotor nerve, they run together and have the same pathway. The parasympathetic pupil-constrictor fibers from Edinger-Westphal nucleus travel within CNIII, and their loss gives you a "blown pupil"

Etiology

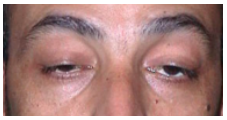
– Microvascular ischemia (DM, HTN, DLP). If risk factors are controlled, the palsy will resolve by itself.
– Intracranial aneurysm (posterior communicating artery) Not the most common but to ophthalmologists **YES**
– Trauma. Neuroimaging is indicated
– Brain tumor. Neuroimaging is indicated

Fourth nerve palsy	
	**Clinical presentation** – **Vertical diplopia.** Characteristic unlike CN III (can have different types.) They mostly complain about actions that require downgaze vision, like: going down the stairs, eating, reading and writing. – **Head tilt:** to the opposite shoulder. **(1)** You might think the kid is shy and he doesn't want to interact, but actually he is just trying to avoid his double vision . **(2)** If you try to correct his head you'll notice some **hypertropia**. **(3)** if you move his head to the same side of the affected nerve it will be worse.
Etiology	Trauma even minor ones not only severe, Idiopathic, Congenital (commonest)

Sixth nerve palsy	
	**Clinical presentation** – **Horizontal** diplopia: two images beside each other (worse at distance) "يقولك لما أمسك خط، لما أكون مسافر أو سايق السيارة تكون worse" ليه؟ Because with near vision you need convergence you don't need CN VI, but when looking far you need it for divergence (taking the eye out) – Esotropia.when you do **cover-uncover test** video (0.25 to 0.50), esotropia is more at distance than near. – Face turn in the direction of the paralyzed muscle. To avoid its action – Limited Abduction on the side of the lesion. **(1)** Primary position **(2)** Looking to his right, right lateral rectus is affected. **Diagnosis: right** sixth nerve palsy
Etiology	You have to rule out: Intracranial tumors, Trauma, Microvascular diseases (most commonly) and Increased intracranial pressure: we call it false localizing sign because you don't know exactly where is the lesion. The nerve passes through the Dorello canal at 90 degree angulation, this makes it susceptible to pressure due to any lesion in the brain. ex. a frontal tumor will cause pressure on this canal leading to 6th nerve palsy

Neuromuscular Disorders

• Ocular myasthenia gravis (OMG)



– Definition: chronic autoimmune disease affecting the neuromuscular junction.

– Clinical presentation:

- **Symptoms:** **PAINLESS** ptosis, diplopia (can present alone or together), **fatigability and variability** are characteristic (usually worse at the end of the day unlike other palsies which are constant. Ask the patient if symptoms are worse early in the morning or at the end of the day?). Ask about systemic weakness, difficulty in swallowing or breathing. majority present with ocular MG then eventually convert to general MG.
- **Examination:** **pupil is not affected**, Assess orbicularis strength (Ask patient to close eyes strongly & open them)

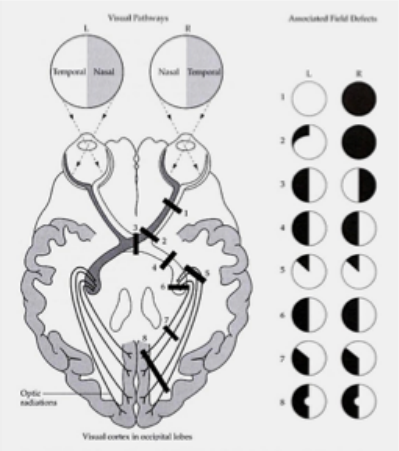
If: painful ptosis or there is pupil involvement don't say myasthenia gravis with your differential diagnosis

– Investigations:

- Blood test for acetylcholine receptor antibodies. 50% present in OMG
- **Tensilon test:** inhibits acetylcholinesterase and can transiently reverse signs of weakness due to OMG, such as ptosis and extraocular muscle paresis. Look for improvement in symptoms. Diagnostic but we don't have it here.

– Treatment: you don't need to know the details but we can give steroids or Acetylcholinesterase inhibitors.

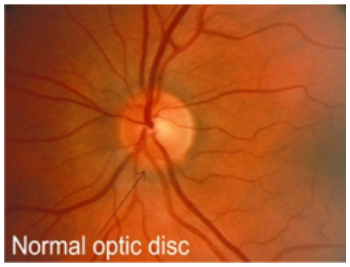
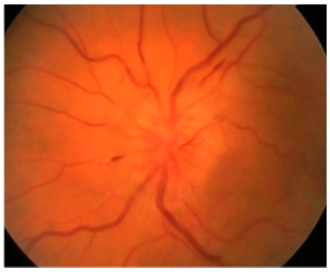
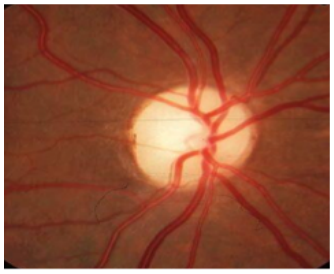
Visual Pathway Disorders



– Lesions anywhere in the visual pathway will produce visual field defect.
– **Important to know:** any lesion before or in front of the chiasm will give you one eye lesion, and if it is beyond the chiasm will give you bilateral VFD.
– Usually VFDs are opposite to the site of lesion.
– **Picture #3:** bitemporal hemianopia due to pituitary tumor compressing optic chiasm.
– **Picture #5:** pie in the sky indicating temporal lobe lesion (superior quadrantic hemianopia)
– **Picture #7:** pie on the floor indicating parietal lobe lesion (inferior quadrantic hemianopia)
– **Picture #8:** occipital lobe lesion because of macular sparing (dual blood supply, if one affected the other will supply the macula.

• Optic nerve disease

– Usually unilateral | Afferent pupillary defect | Central visual loss common, but can can present with any visual field defect. | Loss of color vision. use ishihara test or look for any red led of any medication & simply ask to compare. Patients with optic nerve disease will tell you the color isn't as bright (washed out) as the other eye " مطفي، متغير"
– How to assess the optic nerve in the clinic: visual acuity, visual field, color test and afferent pupillary defect.
– Can present with optic disc edema or atrophy depending on the pathology & the time of your examination.
– Learn how to do direct ophthalmoscope exam. whether you want to be ER physician, neurologist, internist..etc Link

		
Normal	**Optic disc edema**	**Optic atrophy**
Learn the normal to know the abnormal. There has to be sharp distinct margins, a pinkish rim and, usually, a central, pale, cup, cup-to-disc ratio and blood vessels are all intact, central retinal artery and vein enter the globe slightly nasally in the optic disc.	There is fluid all around the margins, blood vessels are especially small and not very clear.	Not to be confused with cupped disc (glaucoma), which comprises: enlarged cup, blood vessels nasally pushed and a healthy rim.

• Typical optic neuritis "disease of the young"

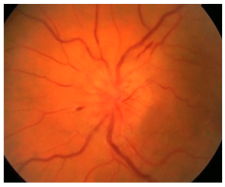
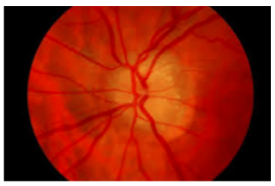
– Inflammatory demyelinating condition associated with multiple sclerosis. not always associated with MS!
– Most common type in young adults. most commonly females.
– **Clinical presentation of typical optic neuritis:** sudden visual loss, pain with eye movement (inflamed optic nerve which is in close proximity with medial rectus muscle. If they look horizontally they will feel minimal pain "discomfort", if pain is severe, this is **"atypical optic neuritis"**) and visual field loss (commonly central, but can present with any VFD).
– 20% of MS patients have optic neuritis as a first manifestation. Patient with optic neuritis needs an MRI of the brain and orbits to look for enhancing lesions for risk of MS. If MRI is normal, risk of MS within the next 10-15 years will drop to 25%. It won't eliminate it but at least if will make it less!
– IV steroids my speed up the recovery process but does not influence the final outcome. It is important to let the patients know that we give them steroids not to improve the visual outcome. Patients who take or do not take steroids will eventually improve if it is typical optic neuritis. Oral is contraindicated because of high recurrence rates [RCT: optic neuritis treatment trial]
– Good recovery.

• Ischemic optic neuropathy (ION) "disease of the old"

Non-arteritic ION	– Patients often have DM, HTN and other vascular risk factor. – Most common cause in older patients. above 40 years to 50. – Altitudinal visual field loss. Superior or inferior. – **Treatment:** no treatment, ask them to control the risk factors to protect the other eye.
Arteritic ION important	– (> 55) years old, associated with giant cell arteritis. Older than non-arteritic ION. – Present with severe irreversible visual loss (counting finger) more than non-arteritic (20/200) – Check: jaw claudication, proximal myalgia & arthralgia, scalp tenderness/pulseless, headache. – Elevated erythrocyte sedimentation rate (**ESR**) and C-reactive protein (**CRP**). – Temporal artery biopsy is the **gold standard for diagnosis.** – **Treatment:** systemic steroids, given immediately if suspected even before the biopsy, patient should not leave the ER without it to protect the other eye (within the first day they will go blind) – Binocular involvement occurs in third of cases, often within the first day.

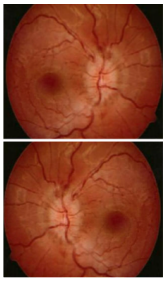
• Congenital disc elevation (< 1 %) *missing pictures from new slides*

– Optic disc margins blurred and the cup is absent but **NO** edema or hemorrhage can be observed.
– May be associated with hyperopia or drusen (high reflective calcium salts)
– You have to differentiate between congenital dic elevation & optic disc swelling (which requires neuroimaging, invasive investigations...etc looking for a reason)

Image		
Diagnosis	Optic disc swelling/edema (pathological)	Congenital disc elevation (pseudopapilledema)
Margins	Unclear. Hue around the disc (fluids)	Unclear without edema (fluids)
Blood vessels	Untraceable. Not clear because of fluids.	Very clear sharp vessels.
Drusen		Present
Abnormal branching		Present
Hemorrhage	Peripapillary hemorrhage	NO hemorrhage

• Papilledema very important, an emergency

– **Causes:** send all patients for neuroimaging: MRI + MRV (to rule out cavernous sinus thrombosis)
  - Intracranial mass you have to rule it out.
  - Severe systemic hypertension. check blood pressure for malignant HTN, controlling BP will make it subside.
  - If MRI is normal (no lesion) → Idiopathic intracranial hypertension (previously known as pseudotumor cerebri). **Common in:** young, slightly obese, female, OCPs, tetracyclines | **Presentation:** headache, tinnitus and visual obscuration she says "لما أسجد الدنيا تسود ثواني" | **Treatment:** Diamox (Acetazolamide: carbonic anhydrase inhibitor) it works by suppressing the production of the CSF **NOT** by its diuretic effect.

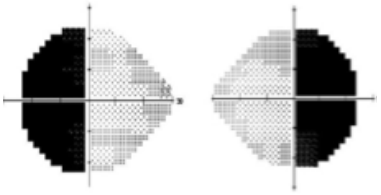
– **Bilateral** swelling of the optic discs secondary to increased intracranial pressure. – Hyperemia of the disc. | Tortuosity of veins & capillaries. | Blurring & elevation of disc margins. – **Peripapillary** flame shaped **haemorrhages**. Sign for acute papilledema. – **Examination:** take your time to look for **spontaneous venous pulsations.** It will be seen with the majority of normal patients. If visualized, the CSF pressure is typically less than 200 mm H_2O. (good & reassuring sign) [**ICP cut point in adult:** 250 mm H_2O or 25 cm H_2O] If you don't see it that doesn't mean it is papilledema, 20% of the normal population don't have it. If you're following-up with the patient and it was present at first then it disappeared → increased ICP	

• Other causes of optic neuropathy

– Infection: viruses, **TB**, cryptococcus and **syphilis**.
– Systemic connective tissue disease: **SLE.**
– Genetics : **Leber's optic neuropathy** (through a **mitochondrial** DNA mutation) If a male patient with Leber's optic neuropathy asked you about the risk of having a child with the same disease since it is heredity, what will be your answer? **0%** because of **maternal** inheritance, it is passed to both males and females the same. However, it shows on males more than females.
– Toxic and nutritional deficiencies. especially with young patients.
– Trauma.

MCQ

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



a) Optic neuritis
b) Tilted discs
c) Pituitary tumor
d) 6th nerve palsy

Answer: C

Extra from 435 team

– **Neuro-ophthalmology** also deals with ocular problems caused cranial Nerves and pupil pathway.
– **The key to diagnosis of pupillary disorders is to:** determine which pupil is abnormal & search for associated signs.
– **Disorders of the pupil may result from:** Ocular disease, Disorders of neural pathway, Pharmacological action.
– **Several diseases of the eye can cause pupil irregularity and alter pupil reactions:** Anterior uveitis, Intraocular surgery - Direct muscle injury, Blunt trauma. Rupture the sphincter muscle, causing irregularity or fixed dilation (Traumatic mydriasis), Acute or chronic High IOP - Acute glaucoma.
– **Tonic pupil:**
- Is due to ciliary ganglionitis which denervates the parasympathetic supply to the iris and ciliary body. On recovery from the ganglionitis, reinnervation is incomplete and the partially denervated receptors of the iris and ciliary body become super sensitive to muscarinic stimulation.
- Sluggish, segmental pupillary responses to light. Poorly reactive to light – because few of the innervating fibres were originally destined for the sphincter. Also, because of the irregular fibre distribution, pupil movement in response to light consists of a slow, worm-like (vermiform) contraction,on biomicroscopy.

– Parasympathetic pathway is much shorter than the convoluted sympathetic pathway, so potential causes for damage are more benign. Parasympathetic plexus sits behind the eye and can be damaged after a benign viral infection.
– **Holmes-Adie syndrome:** is a diagnosis of exclusion. Patient most of the time will be asymptomatic. However they might complain of photophobia because of dilated pupil. Sometimes they will have abnormality of accommodation in near vision. We can provide them pilocarpine will release the photophobia and help them with the accommodation It takes few months and the pupil will go back and constrict.
– **Horner's syndrome**
- May be congenital, in which case the iris colour may be altered when compared to the fellow eye (heterochromia).
- **Anhidrosis:** when the sympathetic pathway is affected proximal to the base of the skull. This catches fibers travelling with the branches of the external carotid, which innervate the skin of the face.
- **Enophthalmos** (posterior displacement of the eyeball): due to paralysis of levator palpebrae muscle.
- **Ptosis:** due to paralysis of muller's muscle.
- **The sympathetic pathway may be affected by multitude of pathologies like:**
  - Syringomyelia, expanding cavity within the spinal cord.
  - Small cell carcinoma at the lung apex. Involvement of the brachial plexus cause shoulder and arm pain and to T1 wasting of the small muscles of the hand (Pancoast's syndrome).
  - Neck injury, disease or surgery.
  - Cavernous sinus disease - catching the sympathetic carotid plexus in the sinus.

- **Do we need to image the patient urgently or give him the next available appointment?**
  - Acute or chronic: Acute within 2 weeks: immediate neuroimaging. | Chronic within several months or he has a surgery: follow up.
  - Painful or painless: Painful: immediate neuroimaging.

Table 13.1 Drugs having a pharmacological effect on the pupil.

Agent	Action	Mechanism
Topical agents		
Dilates	Muscarinic blockade	Cyclopentolate
		Tropicamide
		Atropine (long-acting)
	Alpha-adrenergic agonist	Phenylephrine
		Adrenaline
Constricts	Muscarinic agonist	Pilocarpine
Systemic agents		
Dilates	Muscarinic blockade	Atropine
	Alpha-adrenergic agonist	Adrenaline
Constricts	Local action and action on central nervous system	Morphine

Table 15.1 The causes of isolated nerve palsies.

Orbital disease	e.g. neoplasia
Vascular disease	Diabetes (a 'pupil sparing' third nerve palsy, i.e. there is ptosis and extraocular muscle palsy but no mydriasis)
	Hypertension
	Aneurysm (most commonly a painful third nerve palsy from an aneurysm of the posterior communicating artery. Mydriasis is usually present)
	Caroticocavernous sinus fistula (also causes myogenic palsy)
	Cavernous sinus thrombosis
Trauma	Most common cause of fourth and sixth nerve palsy
Neoplasia	Meningioma
	Acoustic neuroma
	Glioma
Raised intracranial pressure	May cause a third or sixth nerve palsy (a false localizing sign)
Inflammation	Sarcoidosis
	Vasculitic (i.e. giant cell arteritis)
	Infection (particularly herpes zoster)
	Guillain–Barré syndrome

– Medial and lateral rectus muscles have only horizontal actions.
– Superior and inferior rectus muscles are the primary vertical movers of the eye.
– 2 Oblique muscles: Superior and Inferior oblique muscles.
- This vertical action is greatest with the eye in the abducted position.
- The secondary action of vertical rectus muscles is torsion. The superior rectus is an incyclotorter (inwards rotator), and the inferior rectus is an excyclotorter (outwards rotator). The tertiary action of both muscles is adduction.

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

– Quick introduction to Isolated nerve palsy: "u can skip it if u want"

- **Pathogenesis:**
  - Disease of the third, fourth and sixth nerves and their central connections gives rise to a paralytic strabismus.
  - Each nerve may be affected at any point along its course from brainstem nucleus to orbit.
- **History and examination:**
  - The patient complains of diplopia. There may be an abnormal head posture to compensate for the inability of the eye to move in a particular direction.
  - A sixth nerve palsy results in failure of abduction of the eye.
  - A fourth nerve palsy results in:
    - Defective depression of the eye when attempted in adduction.
    - It produces the least noticeable eye - movement abnormality.
    - Patients may notice vertical double vision with some torsion of the image, particularly when going downstairs or reading.
  - A third nerve palsy results in:
    - Failure of adduction, elevation and depression of the eye.
    - Ptosis.
    - In some cases, a dilated pupil due to involvement of the autonomic fibres.

– Fourth nerve palsy:

- **If you want to rule out 4th cranial nerve palsy along 3rd nerve palsy what will you do?** Ask the patient to look down, if the eye intorted the 4th cranial nerve is intact.
- The fourth cranial nerve is the skinniest nerve and runs the longest distance inside the cranial vault. This long passage makes it more susceptible to injury and neoplasm. The fourth nerve is also susceptible to being pulled from the root where it exits from the back of the brainstem. Trochlear paralysis is the hardest cranial nerve palsy to diagnose.

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

– Ocular myasthenia gravis (OMG), other tests:

- **Ice test:** ask the patient to put an ice pad over the ptosis for two minutes and then check for any improvement (measure the degree of ptosis after) may be helpful in establishing that ptosis is due to ocular myasthenia gravis, since cold improves neuromuscular transmission.
- **Sleep test:** measure the degree of ptosis then ask the patient to sleep and re-measure after the patient awakes (improvement = positive test)

– Visual pathway disorders:

- Visual field defects: if unilateral then think about optic nerve pathology, if bilateral then the pathology is at the optic chiasm or beyond.
- Chiasmal defects are always bitemporal.
- Homonymous visual defects could be due to stroke or tumors.
- Left eye blindness due to Left optic nerve damage
- Binasal hemianopia due to bilateral carotid artery aneurysm compressed optic chiasm
- Bitemporal hemianopia due to pituitary tumor compressing optic chiasm
- Right Homonymous hemianopia due to Left optic tract damage
- Right superior quadrantic hemianopia due to Left optic radiation at temporal lobe lesion (pie in the sky)
- Right inferior quadrantic hemianopia due to Left optic radiation at parietal lesion (pie in the floor)

– Optic neuritis:

- Is termed **papillitis** if the optic nerve head is affected and **retrobulbar neuritis** if the optic nerve is affected more posteriorly with no disc swelling.
- Patient will come with sudden visual loss/ visual field loss/ color vision loss which may progress over a few days and then slowly improve.
- Signs: positive afferent pupillary defect. Optic disc edema (**Normal disc in retrobulbar neuritis; a swollen disc in papillitis.)** Ocular pain while moving the eye. **Why ocular pain happened?** Because **optic nerve sheath is attached to medial rectus muscle sheath.** In retrobulbar neuritis because rectus muscle contraction pulls on the optic nerve sheath.
- Vision slowly recovers over several weeks, although often it is not quite as good as before the attack. Repeated episodes may lead to optic atrophy, a decline in vision and a persistent scotoma.

– Relative Afferent Pupillary Defect (RAPD, Marcus Gunn Pupil):

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

– Ischemic optic neuropathy:

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

– Arteritic optic neuropathy:

- Signs: Reduction in visual acuity, Field defect, Swollen and hemorrhagic disc with normal retina and retinal vessels – in AIOP the disc is swollen and very pale. (unlike NAIOP where the disk is swollen but it is not pale), (Remember the blood supply to the anterior optic nerve and retina are different), Tender temporal artery, suggestive of giant cell arteritis, Binocular involvement occurs in third of cases, often within the first 2 days, mall normal fellow disc with a small cup in non arteritic disease.
- Autoimmune vasculitis: It affects arteries with an internal elastic lamina, which therefore includes the ophthalmic artery, but NOT the retinal artery.
- Scalp tenderness “e.g. on combing” , pain on chewing “jaw claudication”..
- GCA can also present as a central retinal artery occlusion when the vessel is affected secondarily to arteritis of the ophthalmic artery.
- **Investigations:** Platelets may also be raised. If NAIOP; do CBC to exclude anemia, check BP and blood sugar. “Both hypertension and diabetes may be associated with the condition”.

- Temporal artery biopsy is the gold standard for diagnosis. But again mat not lead to a diagnosis. (it should be long enough because it has skipping lesion criteria (2.5 length)

– Drusens: "yellow deposits under the retina made up of fatty proteins". B-scan ultrasound can discover drusen (lipid collections)

– Papilledema:

- **Symptoms:** Headache, worse on awakening and made worse by coughing, Nausea and vomiting if the raise in ICP is severe, may be followed by loss of consciousness, pupillary dilatation and death, Pulsatile tinnitus, visual symptoms often are absent [In the short term there is no visual loss. However, in some patients with advanced papilloedema, a fleeting visual loss may occur, lasting seconds, when posture is altered from lying to standing (*obscurations of vision*)] , Diplopia (Double vision), due to 6th nerve palsy.
- **Signs:** There is no spontaneous venous pulsation of the central retinal vein: This has a physiological basis. The central retinal vein is exposed to CSF in the subarachnoid space of the optic nerve, as it leaves to join the veins of the orbit. Normally, venous pressure in the retinal veins at the nerve head is just above ocular pressure. Venous pulsation occurs because the vein collapses briefly with each rise in ocular pressure caused by arterial inflow during systole. When the CSF pressure is higher than the ocular pressure, as in papilloedema, the pressure in the veins at the disc rises above the ocular pressure and spontaneous venous pulsation is lost.
- **DDx:** Adult optic neuritis, Hypertension, Idiopathic intracranial hypertension, Pseudopapilledema (Some normal optic nerve heads appear to be swollen, due a crowding of nerve fibres entering the disc. This is termed pseudopapilledema and occurs particularly in small, hypermetropic eyes where the nerve entry site is reduced in size)
- **Investigations:** CT or MRI followed by lumbar puncture (to measure the ICP and rule out meningitis, Papilledema is a diagnosis of exclusion should be confirmed by lumbar puncture) ,B-scan ultrasonography to rule out buried disc drusen. Fluorescein angiography.
- **Treatment:**
  - Medical: Diamox, diuretics
  - Surgical:
    - Optic nerve fenestration: slit cut of optic nerve sheath > fluid will come out and release the compression
    - Shunt: for patient who has severe headache and blurred vision.

– Papillitis: edematous or inflamed optic dist.

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

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

Lids, Lacrimal & Orbit Disorder

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

Objectives:

- **Orbit:**
  - Anatomy and evaluation techniques.
  - Orbital trauma.
  - Proptosis.
- **Lids:**
  - Anatomy and evaluation techniques.
  - Trauma.
  - Lesions.
  - Malpositions.

Done by: Reem AlAgeel, Deema Alfaris, Majed Aasbali.
Revised by: Rawan Aldhuwayhi, Munerah AlOmari.
Resources: Slides + Notes + 433 / 435 Team.

F1 Doctor repeated the same notes in BOTH groups. Nothing new

Anatomy of The Orbit

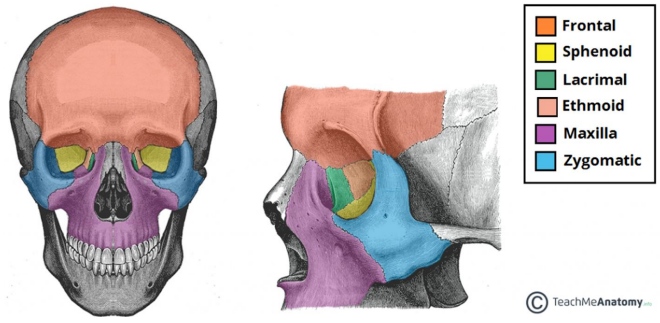
Orbital walls

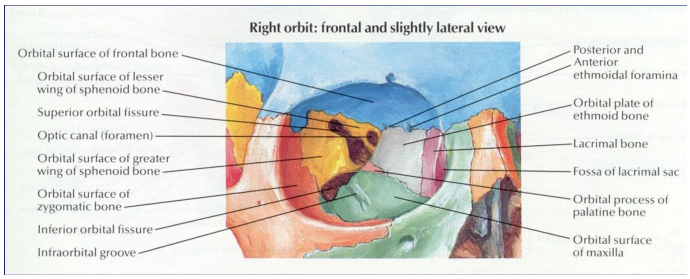
- **Roof**: **frontal** bone, and **lesser wing** of sphenoid bone

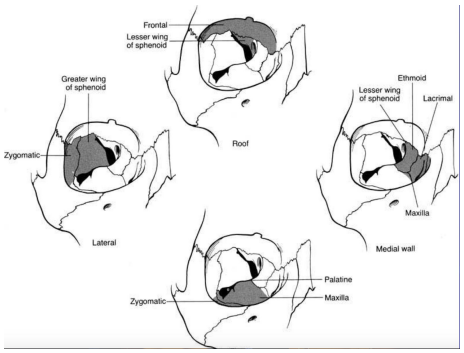
- **Medial wall:** **maxillary** bone, **lacrimal** bone, **ethmoid** bone, and **lesser wing** of sphenoid bone.

- **Lateral wall:** **zygomatic** bone (anterior), and **greater wing** of sphenoid bone (posterior).

- **Floor**: **maxillary** bone, and **zygomatic** bone, and the **far** back is made from the palatine bone.







EXAM

Q1: Which one of these walls is the thickest/strongest bone ?

The lateral wall; because the eyes are in most danger from the lateral side.

Q2: Which wall is the thinnest?

The medial wall.

Q3: Which bone is the thinnest ?

Ethmoid bone (0.3 mm) that is why it is easy to get fractures in facial trauma, and it is also easy for infections in the sinus to go to the orbit.

Q4: What other name is there for the ethmoid bone?

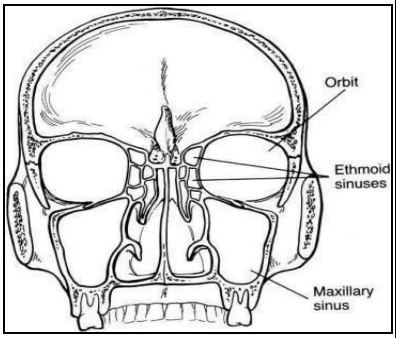
Lamina papyracea (paperlike), because it is the weakest/thinnest bone.

- The orbit is surrounded by 4 sinuses:

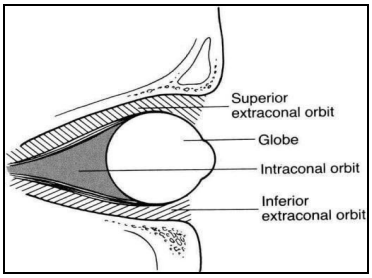
- **Maxillary sinus:** formed at birth.
- **Ethmoidal sinus:** formed at birth.
- **Frontal sinus:** formed at the age of 5 and above.
- **Sphenoid sinus:** formed at the age of 1-2.

* In trauma children are more prone to orbital roof fractures, because they don't have frontal sinus, theoretically due to that frontal sinuses are assumed to have cushioning effect on the orbital roof in trauma.

Sinuses

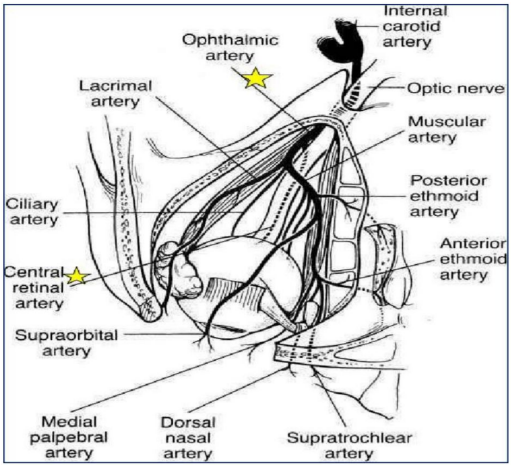


Orbital components



The bony orbit is cone shaped, inlet (anterior) is bigger than the outlet (apex) (posterior).

Blood supply



- The main blood supply is ophthalmic artery, first branch of the internal carotid, supplies the **orbit and the eyeball**, it gets inside the orbit through the optic canal.
- The ophthalmic artery gives so many branches inside the orbit, the most important branch which is the **central retinal artery**, which pass through the optic nerve.
- **Why is the central retinal artery the most important branch?**
  - Because it supplies the retina and if it gets cut, it will lead to total blindness, since it has no collaterals.

Annulus of Zinn:

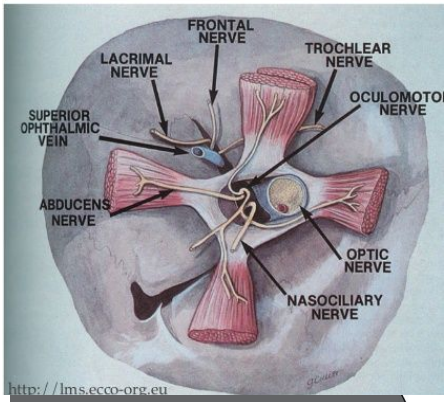
What is it?Annulus of Zinn is a form of condense fibrous tissue, it gives the origin of all the recti muscles (Superior rectus, inferior rectus, medial rectus, lateral rectus).

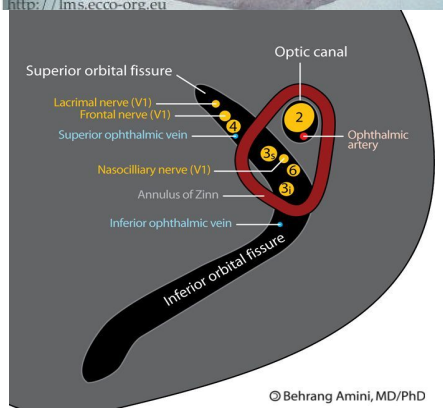
What are the nerves passe <u>within</u> the annulus of Zinn?

- there is the **optic canal** which has the optic nerve, and the ophthalmic artery.
- <u>some</u> of the nerves that pass through **superior orbital fissure are <u>inside</u> the annulus of Zinn:** superior division of oculomotor, nasociliary, and abducens nerves.
- **To understand why they are passing through the annulus of Zinn?**
  - Because all recti muscles get the nerve supply from their inner surface, and in order for the nerves to do that they need to get inside the annulus of Zinn.

What are the nerves that transmit through the superior orbital fissure BUT passe <u>outside</u> the annulus of Zinn?Lacrimal nerve (first division of ophthalmic) , frontal nerve, and trochlear nerve (to remember "LFT").

Figure 2. Right superior orbital fissure viewed anteriorly

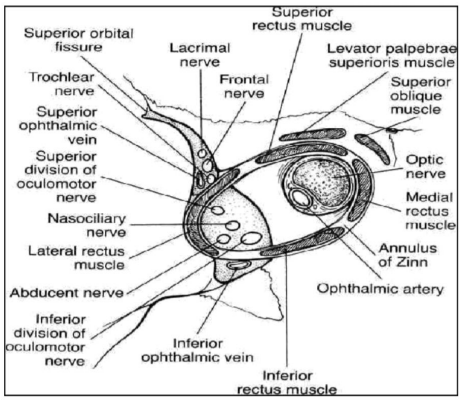




Extra Pic from the web just for u to imagine

- All the extraocular muscles origin from the orbital Apex except the superior oblique muscle which originates behind the inferior orbital rim, near the nasolacrimal duct.

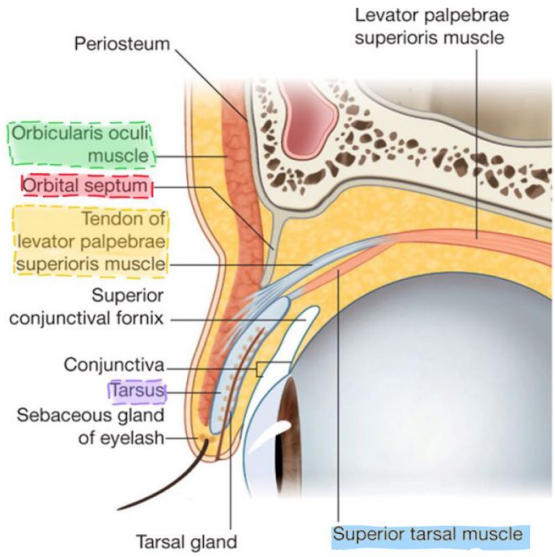
Pic from the slide i don't like it but i have to put it :o)



- **Nerves function:** not in the slides but the doctor mentioned them
  - **Lacrimal Nerve**: Going to the lacrimal gland.
  - **Abducent**: Supplies the lateral rectus (**LR6**).
  - **Trochlear**: Supplies the superior oblique muscles (**SO4**). it is outside the annulus of Zinn just like the superior oblique muscles.
  - **Nasociliary**: Supplies the tip of the nose, ciliary muscles, and the cornea (by the long branch) (hutchinson's Sign: when the tip of the nose has vesicles and involved in herpetic infection, you need to check also for the ciliary muscles, and cornea).
  - **Frontal**: It is a sensory branch form the Trigeminal. its name will change to supraorbital nerve that supplies the entire skull to the back. so a patient with supraorbital nerve injury (or frontal) will complain of numbness in this area.
  - **Oculomotor**: Supply all recti muscles **except** 2: (SO4, LR6).
    - **Superior divisions: supplies the superior rectus and levator palpebrae.**
    - Inferior division: supplies the medial and inferior rectus.

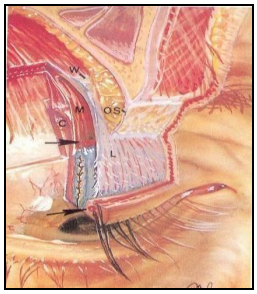
Anatomy of The Eyelids

- **The upper eyelid anatomy:**

From outside: skin → orbicularis muscles (supplied by facial nerve) → orbital septum (a dense fibrous tissue) → orbital fat (also called Preaponeurotic fat) → levator muscle which will be changed to tendon called levator aponeurosis, will attach to tarsals Muller muscle of conjunctiva.	 Extra Pic from the web just for u to imagine
Tarsal is a condense fibrous tissue that is forming the skeleton of the eyelid, within tarsals are meibomian glands: fat secreting (sebaceous) glands, opens on the lid margin. forming the fatty layer of the tear film around 30 in the upper lid and 20-25 in the lower lid.	

- The important thing in the orbital septum (anterior boundary of the orbit).
- Anything behind the orbital septum = orbit = intra orbital, anything anterior to the orbital septum = extra orbital = preseptal.
- **What is the difference between levator muscle and Muller muscle ?**
  - They both elevate the eyelid. however, they differ in the nerve supply & the type of muscles:
  - levator muscle is a skeletal muscle supplied by the oculomotor nerve, and **muller** (AKA superior tarsal muscle) muscle is a smooth muscle supplied by sympathetic nerves.

Pic from the slide i don't like it but i have to put it :o)



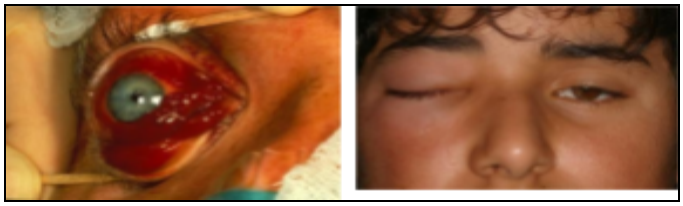
Evaluation

- **When we get a patient with orbital changes we ask about the 7 P's:**
  1. **Pain** (most of orbital pathologies are painless)
  2. **Progression**
  3. **Proptosis** (bulging of the eyes)
  4. **Palpation**
  5. **Pulsation**
  6. **Periorbital changes** (Exophthalmos - thyroid related)
  7. **Past medical history**

1. Pain

- Infection
- Inflammation (Orbital).
- Hemorrhage (Orbital).
- Malignant lacrimal gland Tumor.

Other than these is usually painLess

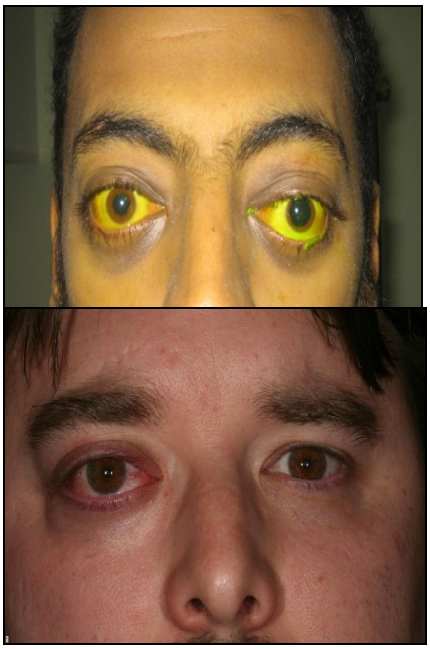


2. progression

If progression from <u>minutes to hours</u>:

Patient comes with proptosis (or any orbital pathology) for minutes or hours, what do we think of?

- Hemorrhage (due to trauma, spontaneous, post-op).
- Lymphangioma (abnormal lymphatic vessels tend to bleed).
- Varix (upon valsalva) varix is (malformed and abnormal enlargement of venous blood vessels that tends to bleed and thrombose).
- Orbital emphysema
- **What is orbital emphysema?**
  - Air around the eye (inside the orbit).
- **How do we get in the orbit?**
  - Sinuses fractures. (the air comes from the sinus)
- **Which sinuses that commonly get fractured?**
  - Ethmoidal, Maxillary bones/sinuses. medial wall is the commonest
- **Why do we worry about orbital emphysema?**
  - Because with orbital fractures, the air will move from the sinuses to the orbit, and the orbital pressure will go very high, and the **air will compress the central retinal artery**, which will lead to retinal ischemia and subsequent loss of vision.
  - So whenever you have a patient in the emergency with orbital fractures or sinus fracture ask him not to blow the nose to prevent the orbital emphysema (so that the pressure won't increase in the sinuses and the air build up around the eye or in the orbit - one way valve mechanism).



If progression from <u>days to weeks</u>: (These are just examples, you do NOT need to memorise them)

- **Children**: Capillary hemangioma, rhabdomyosarcoma, Retinoblastoma, Neuroblastoma,Leukemia. (malignant tumors in general).
- **Inflammatory disease**: Idiopathic orbital inflammatory disease, thrombophlebitis, thyroid orbitopathy, recurrent inflamed dermoid.
- **Infections**: orbital cellulitis, abscess, cavernous sinus thrombosis.
  - if you have a bilateral proptosis think of infection and inflammation.
- **Trauma, post surgical, hemorrhage**: Orbital hemorrhage, lymphangioma.
- **Malignancy**: Rhabdomyosarcoma, metastasis, granulocytic sarcomas, adenoid cystic carcinoma.
- **Carotid-cavernous (C-C) fistula**: Part of the carotid artery course is to pass through the cavernous sinus. In trauma, the sinus builds up a high blood pressure leading to eye congestion, because the ophthalmic vein drains in the cavernous sinus. Then any blood coming from the eye will be congested.

If progression is from months to years (think of benign masses)

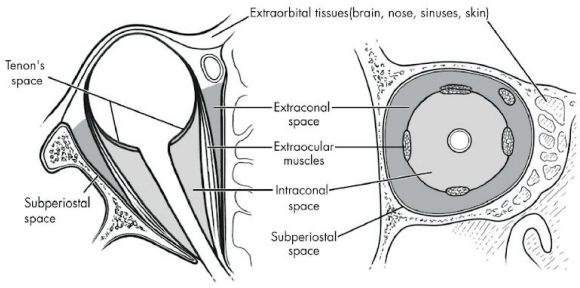
Dermoid Cysts	Fibrous histiocytoma
Benign mixed tumors	Osteoma
Neurogenic tumors	Lipoma
Cavernous hemangioma	Glioma
Lymphoma	Meningioma

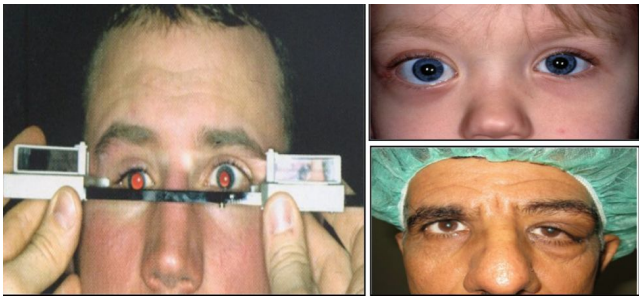
3. Proptosis	
Bilateral	**Unilateral**
• seen in inflammatory conditions (typical condition is thyroid eye disease in Grave's) • immune processes or systemic diseases	• Primary orbital neoplasms usually unilateral (mass occupying lesion)

Causes:

Inflammatory	Infection	Vascular	Neoplasm
• **Thyroid disease** (the most common)Note that thyroid disease can cause BOTH bilateral and unilateral ptosis • Orbital pseudotumor • Wegener granulomatosis	• Orbital abscess • Cellulitis	• Orbital hemorrhage • Lymphangioma (sudden) • C-C fistula • Orbital varices • proptosis with valsalva.	**Benign:** cavernous hemangioma, lymphangioma **Malignant:** adenoid cystic carcinoma, lymphoma, glioma **Contiguous:** sinus, intracranial nasopharynx, skin **Metastatic:**lymphoma, leukemia, neuroblastoma,Rhabdomyosarcoma

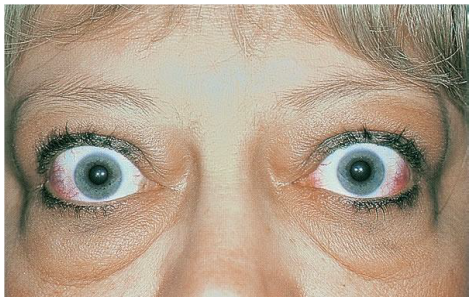
Proptosis can be either: axial , non axial, or pulsatile



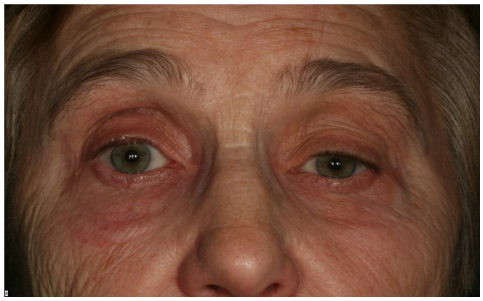


Pseudoproptosis

- **Mostly it is a lid retraction without proptosis**
- The opposite to proptosis is **enophthalmos**. تكون العين داخله على جوا, فيحسبون العين السليمه طالعه فسموها سودوبروبتوززز
  - Most common cause of enophthalmos (sunken of the eyes) is fractures of the medial wall and the floor.
  - The orbital content will herniate inside the sinuses and that will lead to enophthalmos.



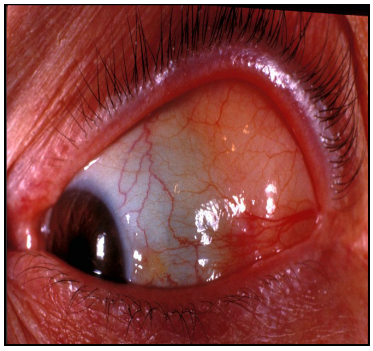
Lid retraction upper eyelid

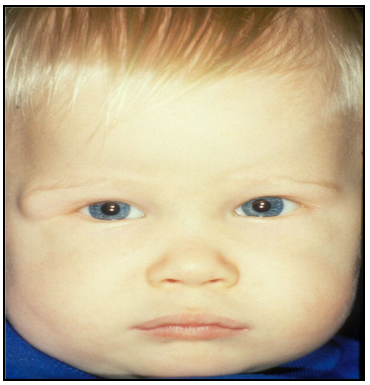


Endophthalmos left eye

4. Palpation

Dermoid cyst is very common and need to be palpated during Examination. It happens in the suture line during development, this baby was born with it.

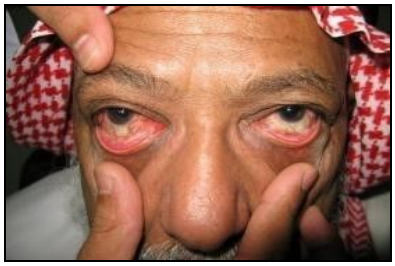


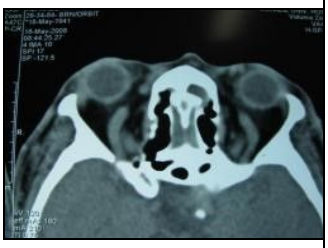


5. Pulsation (was skipped)

With bruits

- Cavernous carotid fistula
- Orbital arteriovenous fistula
- Dural arteriovenous (a-v) fistula



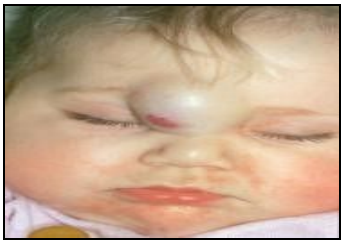


Without bruits

- Meningoencephalocele
- Neurofibromatosis
- Orbital roof defect (condition after surgical removal of orbital roof, sphenoid wing dysplasia)

6. Periorbital changes

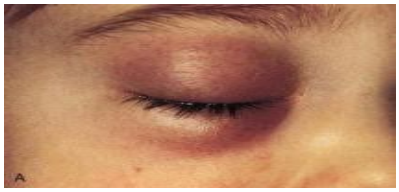
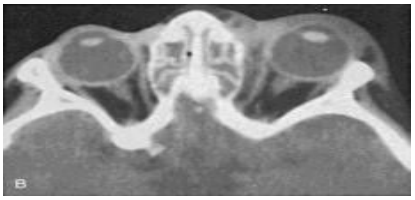
This patient had a skin tumor that was removed from his cheeks. But it recurred with orbital extension and the eye is pushed up.



Encephalocele

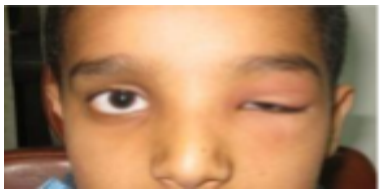
Infections

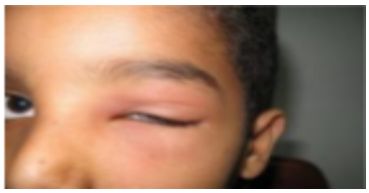
- Signs for infection or inflammation in the eye: a triad of slight redness, hotness, and tenderness
- There are two types of infections, **Preseptal** cellulitis and **Postseptal** cellulitis
- How to differentiate between them? Both will have their eyelid **swelling** and **redness,** however:

Preseptal Cellulitis (extraorbital)	Postseptal Cellulitis (orbital)
Clinical Picture:	**Clinical Picture:**
Vision, eye motility, pupils, VF, optic disc are **Within Normal Limit**. And the globe itself is not proptotic, only the eyelid is swollen and red. They are usually healthy, afebrile people with normal vital signs (patient present with only eyelid swelling, redness and pain, no fever) 	Decreased vision, eye motility problems, and pupils are **usually not normal**, and the globe is proptotic. If they have lid swelling and redness suspect orbital cellulitis. They look sick and may have a fever. Very serious 
Causes:	**Causes:**
• Insects bites • Sinuses or infection in sweat glands or meibomian glands • trauma/abrasions.	• **90% secondary to sinus disease (most of the time ethmoidal sinusitis), the patient can get it from septic emboli, or trauma or surgeries. (Imp!)**
	Complications: - It has a high risk of preventable morbidity and mortality and serious potential complications, including: -Brain and Orbital abscess, Cavernous Sinus Thrombosis.
Treatment	**Treatment**
Oral antibiotics. and send them home (**outpatient**). (except children under 1 year we admit them because they can't monitor them self properly)	**Admission** for close observation, give **systemic IV antibiotics** with referral to ENT, consult ID to help with antibiotics selection, and surgery if they have an abscess collection (drainage) or not responding to medical treatment.

Allergic Eyelid Swelling

Allergic swellings are **very common,** mainly due to insect bites.





How to differentiate between allergic swelling and inflammatory swelling (by history) and (by examination)?

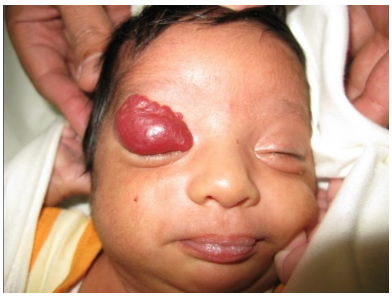
History:	Examination:
• Shorter duration with allergic: develops within hours, the pt usually wakes up with it, whereas preseptal cellulitis takes ~2 days to develop. • Presence of the trigger • Previous episodes (recurrence).	Triad of eye inflammation: • **Redness** • **Hotness** • **Tenderness** **The triad should be absent in allergic swelling**

Treatment: Antihistamine and cold compressors

Capillary Hemangioma

Types of Hemangioma:

Capillary Hemangioma



- usually in children.

***Senario:*A 4 months old baby, the family noticed something started on his eye at age of 2 months and decreasing? Dx Capillary hemangioma**

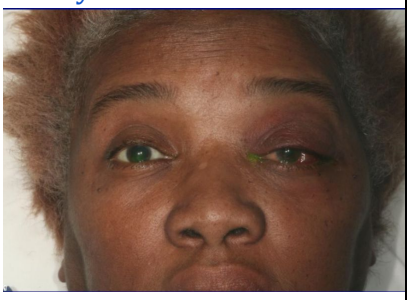
- Do we have to treat this pt? yes even tho it will resolve with time, but we have to treat early to preserve vision. Treat the child to prevent amblyopia (lazy eye) picture because his vision is not mature yet.
- (For vision maturation, the eye input should be intact).
- The younger the child the more critical the case.

Treatment:

1. **Beta blocker (FIRST LINE**, most of cases respond very well**)** typically propranolol (non-selective) if asthmatic patient, think of selective,
2. If no response: Steroids either Injected into the lesion or systemic.
3. surgery.

Cavernous Hemangioma

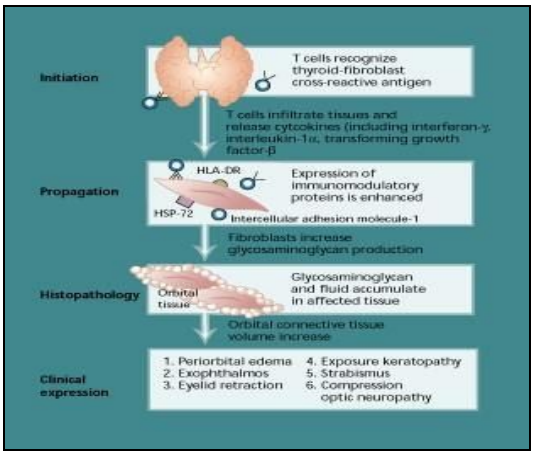
Usually in adults



Inflammation

1. Graves disease

– **Most common cause of unilateral or bilateral proptosis**
– Graves may occur with any thyroid status (euthyroid, hypothyroid, but commonly with hyperthyroid)
– The eye disease is not controlled by thyroid ablation. Why?
Because since it is an autoimmune condition, there are thyroid antigens that attract the antibodies. Also, there are similar/ simulating antigens around the eye. If we remove the thyroid gland, we are removing the antigens of the thyroid gland, but the antibodies are still circulating around the eye. Hence, they will still attack the eye.



What other parts of the body that may harbor simulating antigens?
Pretibial myxedema, Grave's disease is in 3 places: Eyes, thyroid and pretibial (pretibial myxoedema).

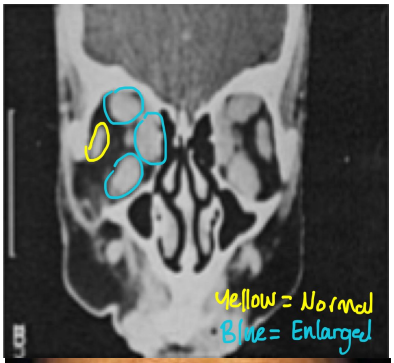
What are the signs of thyroid eye disease?
- Lid lag - Strabismus - Lid retraction - Decrease vision - Lid swelling - Conjunctival injection (chemosis) - Exophthalmos

- **What is the difference between exophthalmos and proptosis?** They are the same thing, but..

Exophthalmos: if the eye protrusion is caused by Grave's disease, so it is specific to Grave's.
Proptosis: more general term (e.g. tumor causing proptosis of the eye)

- **How we get visual loss in a patient with grave's disease?**

One cause is the enlargement of the extraocular muscles (which is suggestive of Grave's disease) which will lead to compression of the optic nerve, sometimes causing diplopia and strabismus
Other cause is like the patient in the picture, because he can not close his eyes the cornea will be dry so they will lose vision because the cornea isn't clear and sometimes they may get corneal infection (exposure keratopathy)



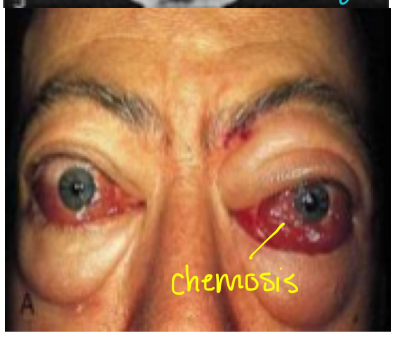
- **Why do the muscles enlarge in Grave's?**

Due to the deposition of glycosaminoglycans

- **Which extraocular muscles are typically affected by Grave's?**

Inferior and medial recti are most commonly involves

- **Why do I care about thyroid eye disease as an ophthalmologist? why do I need to treat it?**Because of these 4 complications:
  1. Exposure keratopathy (Dry cornea); because the eye is bulging and not closing well.
  2. Strabismus; because of enlarged extraocular muscle
  3. Compressive optic neuropathy. (because the optic nerve is compressed from the large extraocular muscle)
  4. Cosmetic



Treatment options(depends in the condition):
1. Steroids
2. Radiation if steroids aren't effective
3. Optic nerve decompression now the surgery can be urgent (done regardless of the stage of disease) or inurgent[1] where it's carried out during the inactive stage (main indications are strabismus and for cosmetic purposes)

[1] You're right, it a made up word.

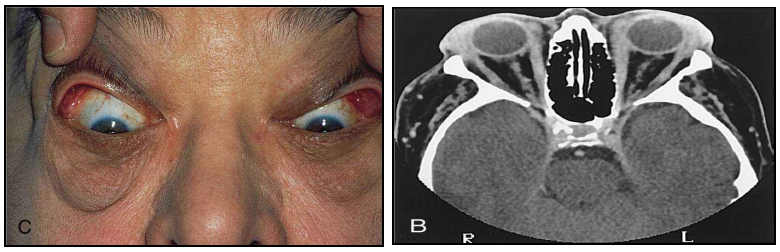
The doctor didn't focus in the following he just said any type of inflammation can cause orbital pathology.

2. Idiopathic Orbital Inflammation

- Orbital pseudotumor[2]
- Prompt response to steroids
- Myositis
- OU or systemic → think vasculitis (*except in kids)

3. Sarcoidosis

- Lacrimal gland (sarcoid infiltration) followed by extraocular muscle involvement.
- ~20% of those with ophthalmic sarcoid disease have ocular or lacrimal involvement , presenting as ptosis, proptosis or ophthalmoplegia.



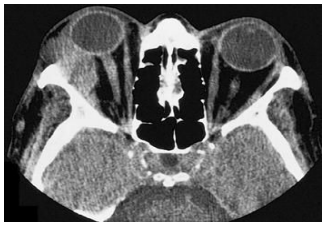
Bilateral enlargement of the lacrimal glands

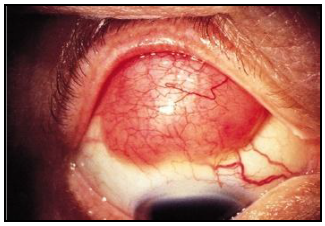
4. Vasculitis

-GCA (giant cell arteritis) - PAN(polyarteritis nodosa) - SLE - Wegener's granulomatosis

5.Lymphoproliferative disorders

1. **Lymphoid hyperplasia and lymphoma:**
   - –20% of all orbital mass lesions
   - – salmon patch appearance
   - –molds to orbital structures
   - – 17% bilateral
   - –50% arise in lacrimal fossa


infiltrative mass in the right lateral extraconal orbit



2. **Plasma cell tumors**

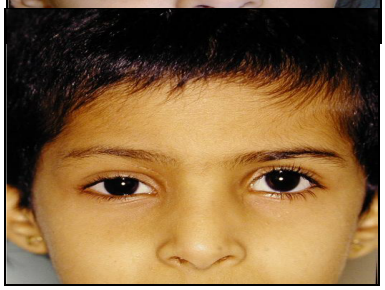
3. **Histiocytic disorders:** macrophage based d/o
is an excessive number of histiocytes (tissue macrophages),that can lead to organ damage and tumor formation

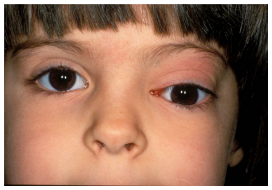
[2] AKA nonspecific orbital inflammation AKA idiopathic orbital inflammation, it is the most common cause of painful orbital mass in adults.

Rhabdomyosarcoma (***IMPORTANT***)

- **Very imp to keep in mind when making a diagnosis**

- **Most common primary orbital malignancy of childhood**
- Average age: 7-8 years, but can happen in any age.
- Sudden onset and **rapid** evolution of **unilateral proptosis** (within days!)
- 90% survival rate (IF DIAGNOSED EARLY)
- It's not very common, but it is life threatening.
- So, whenever you have a child with sudden onset of unilateral proptosis and progressing quickly take it **seriously**! There is high chance that it is Rhabdomyosarcoma until proven otherwise (or leukemia)
- **Tx:** it is a medical emergency, refer to get **chemotherapy** and **radiation** the response is very good.
- 2nd pic was taken 1 year after presentation (1st pic is at time of presentation).
- Note that it is a **PAINLESS** condition
- Very rapidly progressing





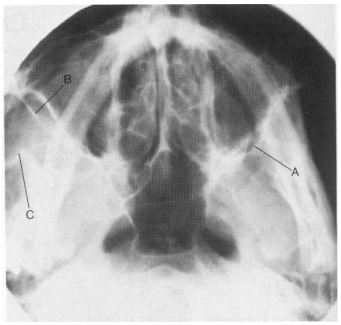
Imaging options:

- Plain films
- CT scan
- MRI
- Ultrasound

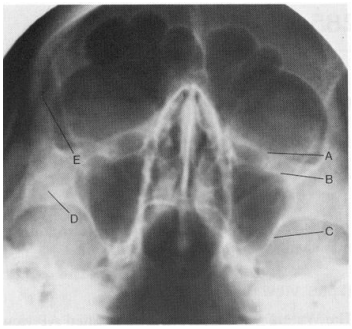
Plain film

Quick - Rule out foreign bodies - **Infrequently used**

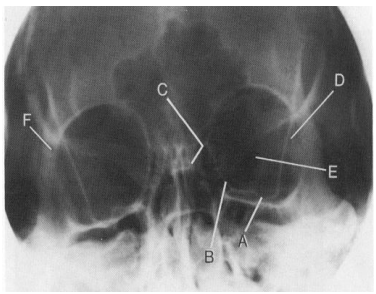
Regarding the x ray images below the doctor said: I don't know how to read them because we don't use x ray



Base view



Waters' view

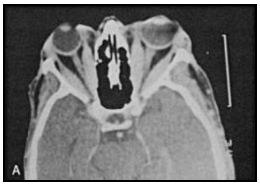


Caldwell's view

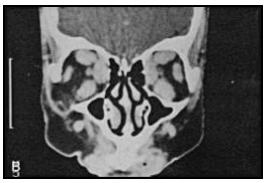
CT

Strengths:	Weakness:	Protocols:
• spatial resolution • bone: fracture, destruction, calcification • quick: emergencies trauma • cheaper	• radiation: 1-2 cGy. there is a question about radiation and risk of malignancy during childhood exposure so it's good to avoid CT in children • soft tissue definition • contrast iodinated: avoid it in case of allergy • may need MRI anyway(not cheaper)	• axial and coronal • +/-contrast

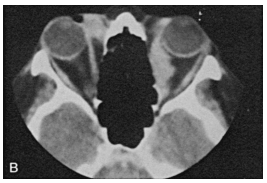
Examples



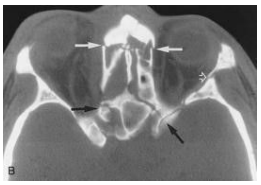
enlarged multiple recti muscles.
Grave's disease.



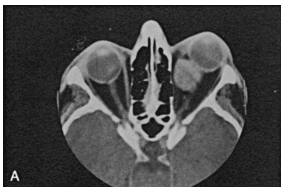
Enlarged recti muscles, suggestive of Grave's disease



Unilateral enlargement of rectus muscle. Could be Lymphoma (bc it's unilateral.. It's not grave's)



CT scan can show you the fractures also, as you can see here are multiple fractures. So, it a good tool to screen for fractures



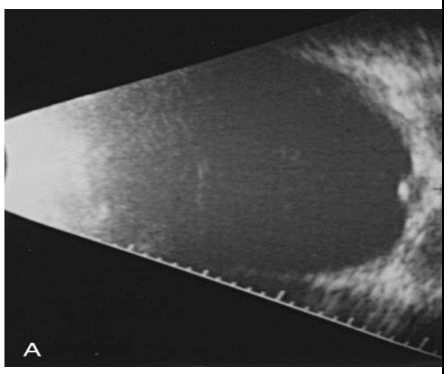
Axial cut.
There is an orbital mass behind the eyeball, this can be a differential but most likely it is cavernous hemangioma

Ultrasound (Orbital Echography)

It's **good for the eyeball** but behind the eyeball is not that accurate.The resultuation is not high as CT or MRI , i didn't rely in US b
We use it from time to time especially for anterior orbital masses
-It is not very good for deep orbital tissue, **but we use it for the eyeball**
-to measure the length of the eyeball prior cataract surgery to estimate the power of the artificial lens that is to be implanted into the eye

Features:

- Dynamic
- Less expensive
- +/-Availability variable



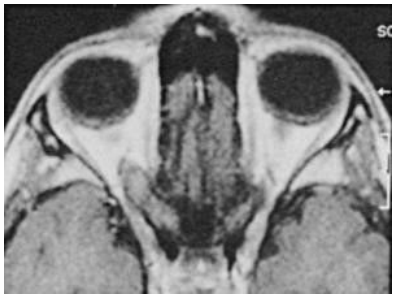
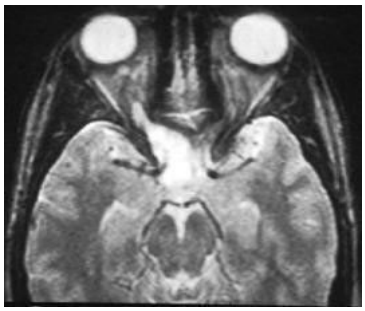
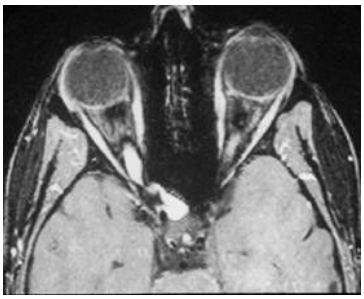
This is an ultrasound showing an orbital cyst behind the eyeball

MRI

- We use it when we are sure that we're dealing with soft tissue lesion(especially: orbital apex, optic nerve or cavernous sinus)

How to differentiate between T1 & T2 image?

- **Fluids appear dark in T1, and white in T2.** "I like to ask my students about this"
- The eye is filled with fluid like, so if the eyes are white -> T2 , and If the eyes are black -> T1
- T1 typically done with contrast(so whenever u see vessels with contrast in the image)

Strengths:	Weakness:	Protocols:
- Tissue - **T1: Anatomy** - **T2: Physiology** - No Radiation	- Magnetic pacemakers, surgical clips - Claustrophobia	● Axial/coronal/sagittal ● Gadolinium contrast ○ non-iodinated ○ allergies RARE ● Orbital lesions ● fat suppression We can do something called fat suppression which makes the fat black. bc the orbit is full of fat. This is helpful for finding pathologies.
Examples		
		
T1	T2	T1

Facial trauma and fractures

Facial Trauma:

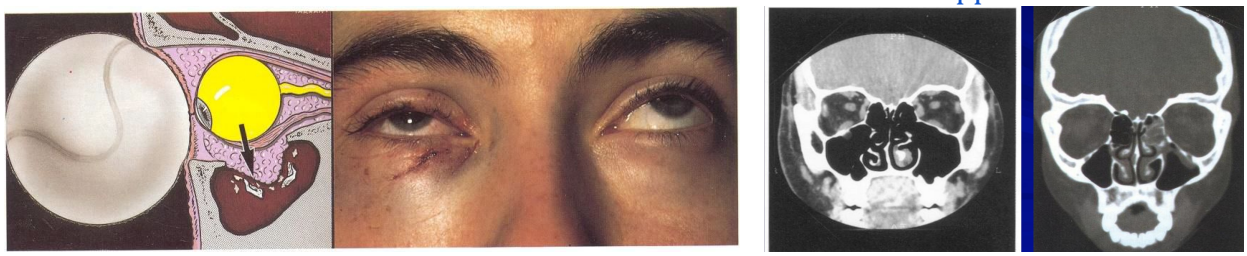
- **Midfacial fractures**
- **Zygomaticomaxillary Complex (ZMC)fracture**
- **Wall and floor fractures:**
  - medial wall: lamina papyracea
  - orbital floor: blow out vs rim involvement
  - lateral wall and orbital roof:less common
- **Optic canal fractures:** traumatic optic neuropathy

Orbital fracture is a big topic focus only on orbital floor fracture and entrapment

Orbital Floor Fracture with entrapment *IMPORTANT*

- Orbital floor fracture, also known as "blowout" fracture of the orbit.
- **Trapdoor Fracture=** fracture of the floor of the orbit + muscle entrapment
  - It's Very common with direct trauma to the eye, it's really common among children to have a minimum fracture with muscle entrapment without external signs, so it is really important if you got a child with Hx of trauma to check for eye motility (painful sometimes).
  - So whenever you have a patient with orbital trauma, you need to look at the eyes motility to rule out this condition.
  - **why is it common in children?** because their bones are softer so they'd open up and close like flap, creating a trapdoor and trapping the inferior rectus
  - The traction of extraocular muscles or compression of the nerves may lead to a **parasympathetic response** (oculocardiac response) manifesting as bradycardia, hypotension and possibly syncope.

Case: This is child had a fracture to the right eye,(you can see there is no much swelling and ecchymosis) and the patient is trying to look up in both eye, but he can't look up by the right eye, because when you look at CT there is a fracture in the orbital floor and the inferior rectus muscle is entrapped.



Rx: We need to operate him urgently, because if the muscle is kept entrapped for a long time, it will lead to ischemia and fibrosis, end up with permanent double vision

Lacrimal Disorders

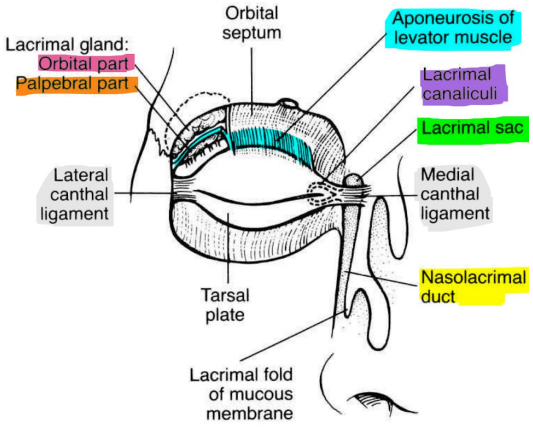
Structure and Function:

Anatomy:

The lacrimal gland is approximately 2cm long. It can be divided into two main parts:

Orbital: larger and sits on the lateral margin of levator palpabrae	**Palpebral:** smaller and located along the inner surface of the eyelid

The two lobes are separated by **levator aponeurosis**, which is the tendon for levator muscle

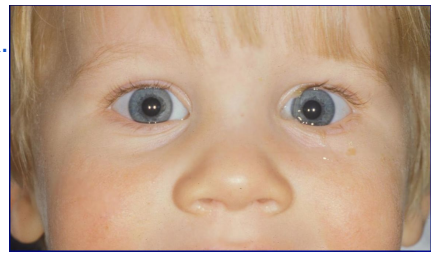


Physiology:

from the **palpebral lobe** there is small ductioles secreting the tears to lubricate the eye, these ductioles open into the superior fornix, the tears will lubricate the cornea, and then will be drained through the lacrimal drainage system, starting with the lower punctum which is a small round opening and upper punctum, and from there, there are small ducts called canaliculi (canaliculus), the upper and lower canaliculi will meet to form **common canaliculus**, then it will go inside the **lacrimal sac**, then from the lacrimal sac the **nasolacrimal duct** will take the tears to the inferior meatus.

You can see in the picture **medial canthal ligament (cover the lacrimal sac), and **lateral canthal ligament** 2 important structures to stabilize the eyelid to the medial & lateral wall.

Congenital Lacrimal Duct Obstruction(common problem):

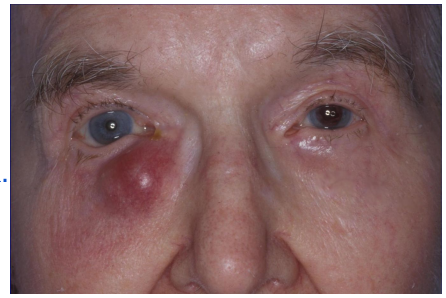


What is it?

- Normally the canalization of the nasolacrimal duct should be completed at birth. However, some children will have a delay of the canalization of the duct: Congenital nasolacrimal duct obstruction

Where does the obstruction occur?

- At the distal part of nasolacrimal duct:valve of Hasner.



What is the clinical presentation?

- Typical presentation is **tearing with discharge**.

Why do they have tearing?

- The tears cannot pass through the lacrimal drainage system because of the obstruction, there is a membrane obstructing the system not allowing it to drain.

Why do they have discharge?

1. The tears stagnate in the area of obstruction, which gives a good medium for infections.So they present with discharge and infection. (see 2nd pic)
2. The lacrimal sac is lined by mucus secreting cells, similar to the mucus secreting cells in the nasal mucosa. So in these children, it will drain back to the eye.

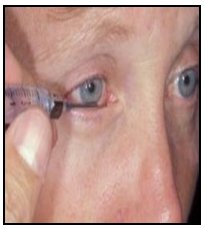
How to differentiate by clinical presentation?

- If a child comes with tearing and discharge, we think of **congenital nasolacrimal duct obstruction**.
- **IMPORTANT:**If a child comes only with tearing, we think of: congenital abscess – congenital glaucoma – eyelashes irritation, Foreign body (anything that irritates the eye)

What happens if we do not treat them?

- Acute infection (Acute dacryocystitis) → Abscess → orbital cellulitis
- Other thing is if you keep the eye watery in a child, the vision will not develop normally (possibility of Amblyopia)
- Patients who have nasolacrimal duct obstruction that is not treated may develop **dacryocystitis**. **Dacryocystitis** is an infection of the lacrimal sac, secondary to obstruction of the nasolacrimal duct at the junction of lacrimal sac. It causes pain, redness, and swelling over the inner aspect of the lower eyelid and epiphora(excessive watering of the eye).

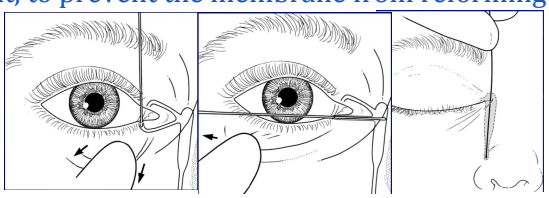
What are the findings on Examination?



- **In adults:** we pass a cannula all the way to the lacrimal sac, and then we inject the fluid, and to check for nasolacrimal duct patency.
- **In children:** We put a fluorescein dye (orange dye) then wait for 5 minutes. The dye should disappear from the eye (if the lacrimal system is intact). However, if there was an obstruction, the dye will stagnate inside the eye. (it won't help to differentiate between nasolacrimal duct obstruction or canalicular obstruction.)

What is the treatment?

1. Usually we ask the family to wait until the age of 1 year and to do **massage** for the lacrimal sac. Ask the mother to frequently (with every feeding for example) put her finger under the medial canthal ligament and push, this will compress the lacrimal sac because the lacrimal sac is behind the medial canthal sac,and when the pressure increases in the lacrimal sac, hopefully it will rupture the membrane.
2. If it didn't improve up to 1 year: we recommend **probing:** A small probe is introduced through the upper or lower punctum based on the place of obstruction and is advanced to the lacrimal drainage system, until it resolves the obstruction. Sometimes we put a stent, to prevent the membrane from reforming we keep it for 2 to 6 months, and remove it in the clinic.



We don't do this procedure to adult it's useless b/c the obstruction in adult is due to adhesion and fibrosis unlike children where the obstruction due to imperforation of membrane

Eyelid Disorders

Eyelid Trauma:

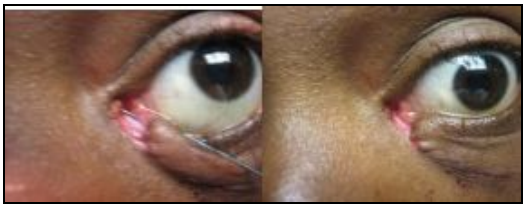
Types: Blunt, sharp/penetrating

Classification:

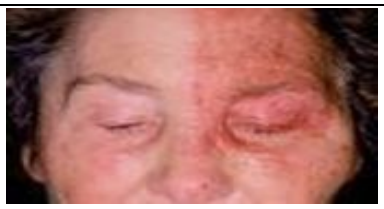
- if one or all of the following involved in an eyelids trauma call ophthalmology (lid margin, canthal, canaliculi)whenever you have eyelid trauma u need to look at 3 things((lid margin, canthal, canaliculi)

Treatment:

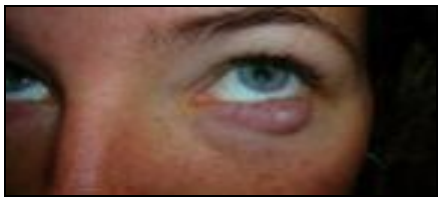
- **If the lid margin is spared (not involved):**
  - ➢ Skin and orbicularis only injured → skin sutures no need to suture the muscle just suture the skin.
  - ➢ FAT protrusion = septum violated, DO NOT suture the orbital septum. There is a very high chance to have open globe injury either corneal laceration or scleral laceration, so careful exam should be done to these pt.
- **If the Lid margin involves:**it will have an abnormal alignment, important to be repaired by an ophthalmologist
  what happens if the eyelids are not aligned together nicely?Every time the patient blink that will cause corneal irritation therefore will need suturing.
- **If the Canthals involve:** that means the eyelid is **unstable** so we "call ophthalmology":because the cantheals are important to stabilize the eyelid, they attach the eyelid to medial wall and lateral wall (repair the tendon)
- **If the Canalicula involves:** if it's involved we need to repair it because the patient will end up with tearing, "call ophthalmology"



Lid laceration with canalicular involvement

Blepharitis:	Herpes Zoster Ophthalmicus:
 **What is it**? Chronic inflammation around the roots of the eyelashes. **Clinical findings?** scales around the lashes, redness and irritation in the eye **Etiology?** Commonly caused by Staph, but can be caused by others like Streptococcus species. **Treatment?** topical antibiotics and eyelid hygiene. NOTE: The main problem is: very difficult to eradicate, chance of future recurrence, need to be treated again (you should tell that to the patient it's a chronic condition there's no magic treatment for it)	 **What is it**? AKA shingles, is a viral disease characterized by a painful skin rash in one or more dermatome distributions of the 5th cranial nerve, shared by the eye and orbit. NOTE: If the patient is elderly, it's okay because it happen in elderly. But if the patient is young, you have to investigate for immunodeficiency because Herpes Zoster Ophthalmicus uncommon in adult. **Treatment?** oral antiviral agents.

lids lesions:

Sty	Chalazion (very common)
What is it?Acute inflammation around the root of eyelashes, either from sweat glands or sebaceous glands. **Clinical findings?** Presents with abscess or pus collection **(pointing pus is a characteristic of sty)** Mainly anterior around the eyelashes **Treatment**: warm compressors with topical antibiotics, but it can improve by itself 	**What is it**?Granulomatous inflammatory lesion caused by obstruction of **meibomian glands**, which leading to accumulation of the sebaceous secretion from the meibomian glands. **Clinical findings?**It will begin with swelling and redness with time it will be like a small nodule Manly posterior **Treatment**: We give topical antibiotics + warm compresses it may resolve by itself. But if it does not improve after 1 month, we drain it (surgical removal) 

Xanthelasma

50% will have abnormal lipid profile, so you need to screen for hyperlipidemia

Treatment:
First we need to treat lipid abnormalities if there is any
If it is not improving we need to do surgery to excise it

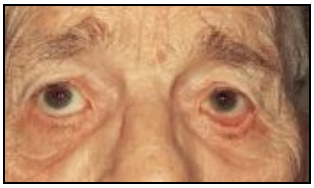
Lid malpositions:

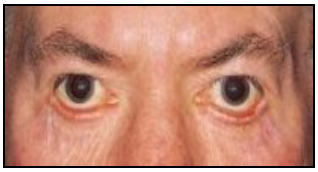
A. Ectropion	B. Intropian	C. Blepharoptosis	D. Retraction

A. Ectropion: outward turning of lid margin

Types:

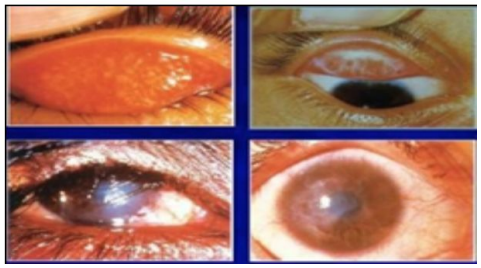
- Congenital
- Involutional:**Aging** is the most common cause of it The eyelid is sagging away from the eye, because of the laxity of the eyelid tendon
- Paralytic: in case of patients with facial palsy
- Cicatricial (scarring): like the second picture, the patient has scleroderma, this patient has a problem with his skin so we call it cicatricial ectropion because of scarring or contraction in the skin
- Mechanical





B. Entropion: Inversion of lid margin towards the eye

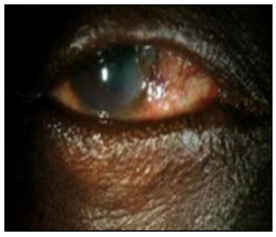
Types of Entropion:



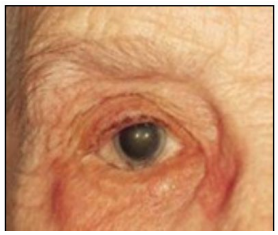
1- Cicaterical(scaring): (most common type in KSA, which is secondary to old trachoma, seen among elderly, we don't see active trachoma now)
2- Involutional: related to **aging** and eyelid laxity
3- Congenital
4- Acute septic

Cicaterical entropion and trachom:

- **Most common cause of Cicaterical entropion.**
- **What is the causative organism of Trachoma?** Chlamydia Trachomatis (bacteria), it has no cell wall, so it lives inside the cell like viruses.
- **What is the stain for Chlamydia Trachomatis?**Giemsa stain
- **How does trachoma cause entropion?**typically trachoma presents during early childhood with redness and discharge(pic1), if it is not treated it will lead to scarring of the conjunctiva and that will shorten the tarsus/tarsal plates(pic2), so the lid margin will be directed towards the eye, if not treated will lead to corneal opacity



1



2

Nowadays we don't see the 1st pic because trachoma has been eradicated from our country, we just see the sequelae of trachoma (pic2)

Treatment:

- If acute/active infection it is a bacteria that we treat it with tetracycline, azithromycin, clarithromycin, so it respond with C2 antibiotics
- Later stages: surgery, then if the corneal scar is too advanced we may do **keratoplasty**, or corneal transplant

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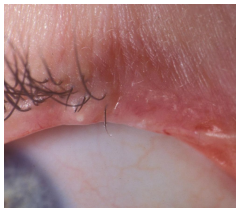
Trichiasis:

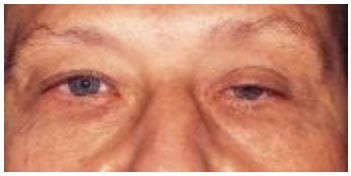
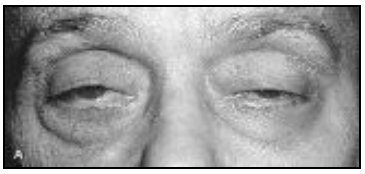
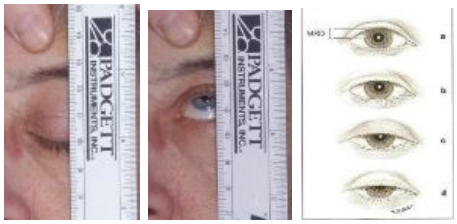
One single eyelash or two are misdirected toward the eye, the rest are ok.
Typically caused by trauma, but it can be caused by other causes like infections …etc.
If the whole lid margin is turning toward the eye, we call it: **entropion**



C. Blepharoptosis: is drooping or inferior displacement of the upper lid

Classification:
Congenital vs acquired

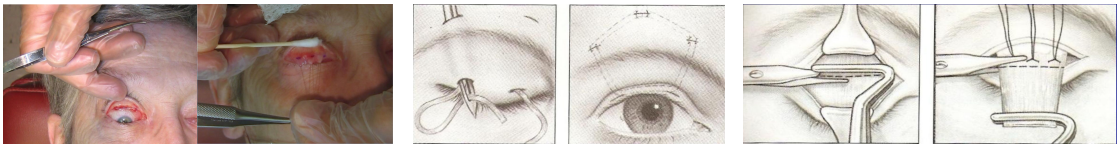
- Myogenic (like abnormal levator muscle)
- aponeurotic (the tendon is stretched repeatedly, can happen with patient using contact lens)
- neurogenic (from the 3rd nerve, horner syndrome)
- mechanical (a mass in the eyelid)
- traumatic (trauma to the muscle levator)

Myogenic	Aponeurotic	Neurogenic
Causes: **Congenital:**Dysgenesis of levator. **Acquired:** –Localized or diffuse disease –Muscular dystrophy –CPEO "Chronic progressive external ophthalmoplegia" –Myasthenia Gravis –Oculopharyngeal dystrophy Generally, in children, whenever the eyelid is blocking the eye, we need to do surgery to prevent amblyopia. In adults, we just fix it because patients want to see from both eyes, but it will not cause amblyopia  This child has right congenital ptosis. He is lifting his chin up so he can see from both eyes. We can't leave him like this; we need to do surgery because he may develop amblyopia and also neck problems.	-Most common form of ptosis (The muscle is normal but the tendon is stretched, usually secondary to aging + contact lens wear, because they stretch their lids, repeated stretching to the eyelid) -High lid crease with normal levator function  	**Acquired and congenital forms** ● **Acquired causes:** – **3rd nerve palsy **** – Horner syndrome – Myasthenia gravis  **Evaluation: Levator Function** **(**this is the way we evaluate the muscle) We ask the patient to look all the way down, and then we measure how much they go up. Normally it should be 15 cm and above, like in this picture. 

Treatment:

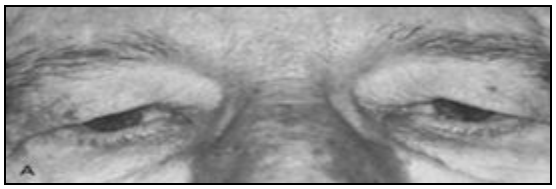
- Mild ptosis, good levator function: Mullerectomy
- Any ptosis, reasonable levator function: Levator resection
- Severe ptosis, poor levator function: Frontalis suspension

Mullerectomy:



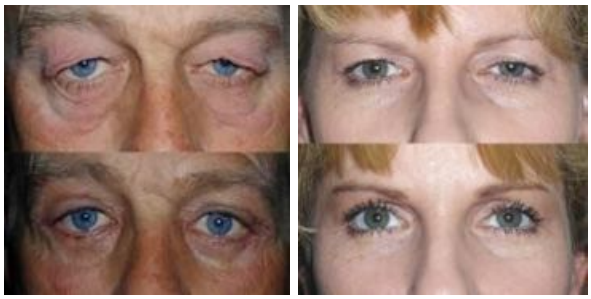
Dermatochalasis:

- Pseudoptosis: excessive skin in the eyelid. But the eyelid position is normal
- This is a very common condition; we usually do surgery for it.
- The procedure's name is **Blepharoplasty** and it is a very common cosmetic procedure.



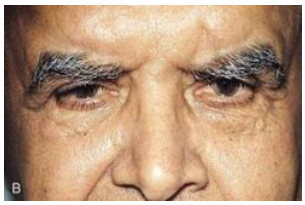
Before surgery →

After surgery →



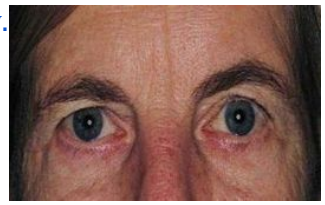
Brow ptosis

This patient has a normal eyelid position. But he has bilateral brow ptosis. Usually related to aging



Brow ptosis

Unilateral right brow ptosis, lid opening is ok, with normal lid margins.. It's usually a normal facial asymmetry, but another imp cause is facial nerve injury, bc it supplies the frontalis muscle that elevates the eyebrow.



Abnormal eyelid movements:

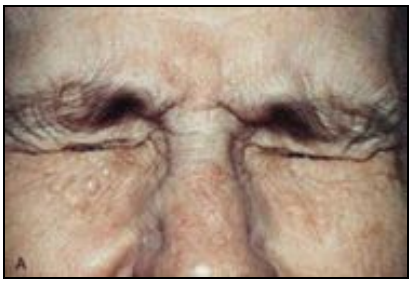
1) Blepharospasm	2) Hemifacial spasm	3) 7th nerve palsy

1) Blepharospasm:

What is it? Involuntary tonic, spasmodic contraction of orbicularis If it's both eyes we call it blepharospasm, if one eye it's hemifacial spasm

Cause:Most of the time we don't know the cause but you should First you rule out:

- Dermatochalasis-rubbing
- Brow ptosis-frontalis spasm
- Blepharoptosis-levator dehiscence
- Ectropion/entropion
- Dry eye
- Foreign body



Treatment:

- To relieve the spasm we inject **Botox** around the eye in most of the patients, and this will decrease the tone of the muscles, reinjection is required.Small minority of patients don't respond to Botox, surgery is required to excise part of the orbicularis muscle.

2) Hemifacial spasm:

- **What is it?** Intermittent contractions of the entire side of face
- Present during sleep
- Compression of 7th nerve at the level of the brain stem
- We need to order MRI in these patients to rule out 7th nerve compression

There is a common normal condition called myokymia: when only one eyelid is twitching.It is normal, and it does not indicate any pathological process.
If it involved both (Upper and lower eyelids), you need to think of hemifacial spasm.

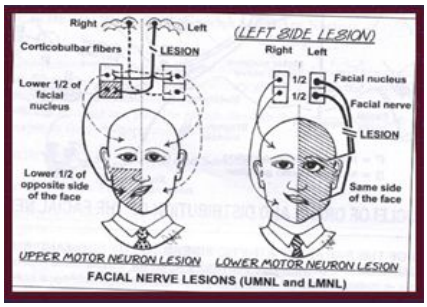
3) 7th Nerve palsy

Location of lesion:

- Supranuclear, brain stem, peripheral

Cause of paralysis:

- Bell's – Infection – Infarct – Demyelination – Neoplasm – Trauma – Miscellaneous.



In ophthalmology we see UMNL or LMNL patients?

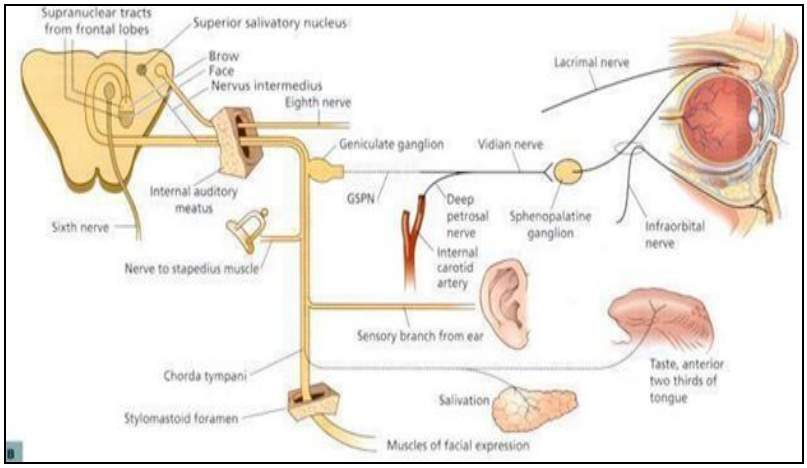
- UMNL: forehead is spared
- LMNL: all the side is affected
- ➔ So, we only see LMNL

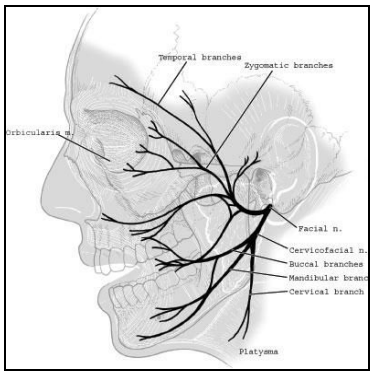
See the illustration to understand better (not in the lecture)

What are the ophthalmic manifestations of facial nerve palsy?

- Lagophthalmos (Inability to close the eye) (because orbicularis muscle gets paralyzed because its supplied by the facial nerve)
- Ectropion (dropping of the eyelids)
- Brow ptosis (because the facial nerve supplies the frontalis muscle)
- Tearing (when the eye cannot close well:there will be reflex tearing, and also when we cannot blink the tears won't pump from the eye to the nose)
- Exposure keratopathy(corneal damage b/c of dryness)(because the eye cannot close well) so we need to lubricate the eyes

The course of 7th cranial nerve





Treatment of 7th nerve palsy with ophthalmic manifestations?

Usually we lubricate the cornea to prevent infection, so conservative treatment with lubrication
If it persists up to months there is surgical options like: tightening of the lower eyelid, we may put a gold weight to make the eyelid to blink, we may do brow ptosis surgery…etc.
So different procedures depending on the patient's findings

Botox in ophthalmology

We use it to treat blepharospasm and hemifacial spasm. Also, we use it to treat strabismus.
When they treated the patients of blepharospasm with Botox they observed that the wrinkles in the glabella and in the frontal lines are gone, so from that came the cosmetic use of the Botox

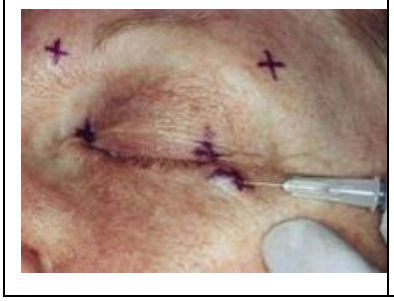
Botulinum toxin:

- Clostridium botulinum
- Neurotoxin types A,B,C1,D,E,F,G
- Botox = Botulinum Toxin A (**it's the most common type we use**)
- Blocks the cholinergic nerve terminals, thereby decreasing release of acetylcholine
- Onset 3 days
- Peak effect 1-2 weeks Duration 6-12 weeks

Uses

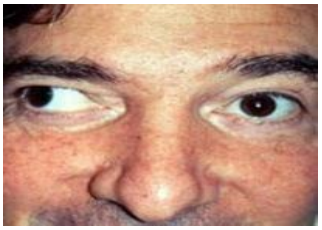
1) Blepharospasm

The treatment of option



2) Strabismus

We inject the lateral rectus muscle to be weak. So, the eye will be straight. Temporary



3) Glabella Botox

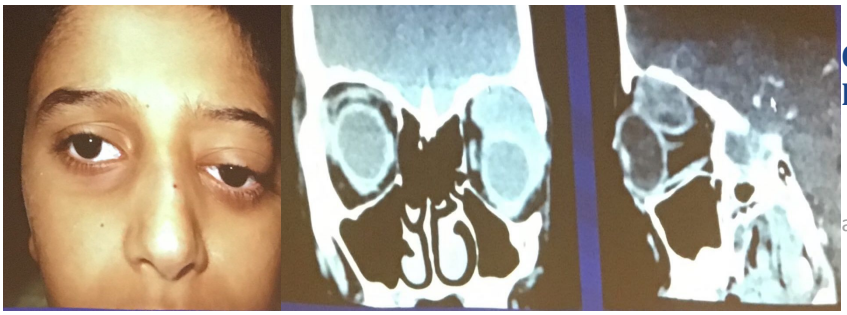
4) Botox for Crow's-Feet

Conclusion

- Knowing the anatomy helps to understand different pathological process, this is true for any medical speciality.
- Early diagnosis and proper management of **orbital cellulitis** save patients vision and lives.so early diagnosis of orbital cellulitis is really vital
- **aponeurotic ptosis** is the most common cause of ptosis
- **Thyroid eye disease** is an autoimmune diseases and it's consider the most common cause of proptosis either unilateral or bilateral
- **Unilateral recent proptosis** in a child should be taken seriously
- **Congenital nasolacrimal duct obstruction** commonly caused by membranous obstruction and typically present with tearing and discharge since birth
- **Chalizen** is a common condition results from blockage of melbonum gland and present as discrete swelling in the eyelid
- **Severe congenital ptosis** needs to be corrected as early as possible to prevent amblyopia.
- **ptosis in adult** need surgery when the ptosis blocks the pulpi if not it consider cosmetic and corrected by elective procedure any time
- Proper treatment of **exposure keratopathy** in pt with facial nerve palsy is critical to prevent corneal ulceration and scarring

Cases

Case 1: **an 9 y/o child brought to ER with history of recent unilateral proptosis, when the ER oncall asked the family about hx of trauma, they said we don't know we just noticed the eye bulging, however when the doctor asked the child he said yes i had trauma in my left eye.**



CT: shows mass in the left eye
Dx: rhabdomyosarcoma

التروما الي يقول عنها الولد ماسببت فراكتشر و لا لها دخل بالموضوع, فيا اما انه يكذب او انه الاصابه الي جته كانت خفيفه, المهم انك ما تعتمدين على كلام الطفل بلحاله فلازم ترجحين وتسوين احتياطاتك! your index of suspicion any recent unilateral proptosis in a child is tumor until proven otherwise

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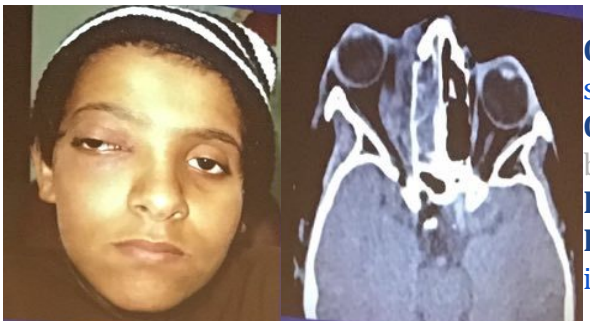
Case 2: **11 y/o boy presented with pain and redness in the right eye for 3 days duration.**



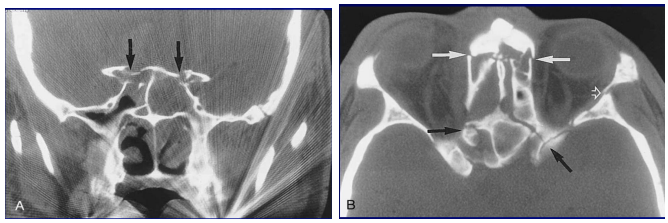
On examination: The right eye is deviated and pushed, some swelling
CT:subperiosteal abscess + ethmoidal sinusitis (normally the sinus is black but here the right one is opacified)
Dx: orbital cellulitis, it's not preseptal because there's ptosis
Rx:IV antibiotics admission, consult ENT and ID group.if no improvement with antibiotics→do surgical drainage

INFORMATION SKIPPED BY THE DOCTOR

Some information found in the old slides, the doctor didn't explain them, and he said focus only in what i explained.

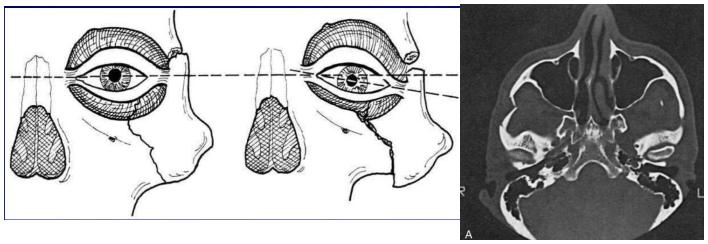
Other Facial Fractures

Optic Canal Fracture:

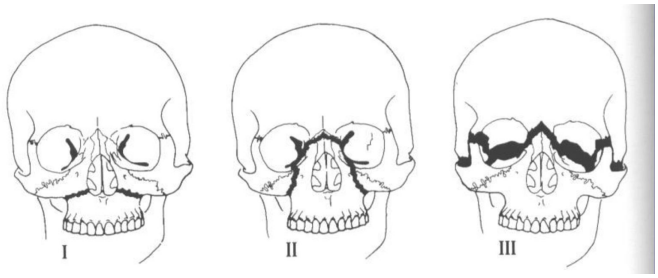


May be with or without displaced bony fragments

Zygomaticomaxillary Complex (ZMC) fracture

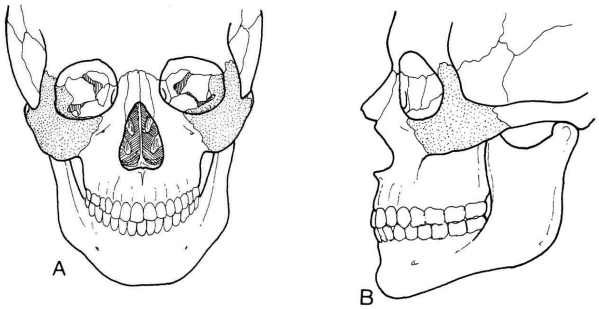


LeForte fracture



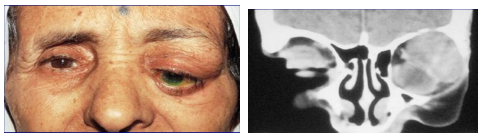
Class 1:transverse maxillary
Class 2:pyramid
Class 3:craniofacial disjunction

Zygoma



Lacrimal Gland Masses:

Inflammatory	Non-inflammatory
• Sarcoidosis • Orbital Pseudotumor • Vasculitis	• Lymphoproliferative • Epithelial neoplasms

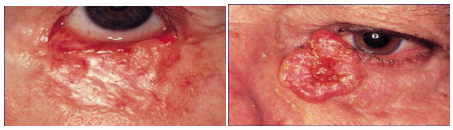
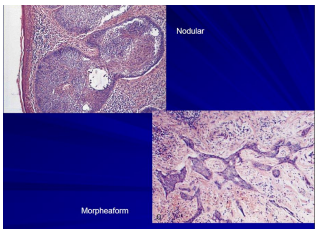
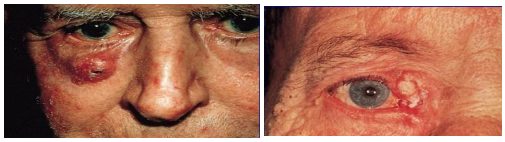
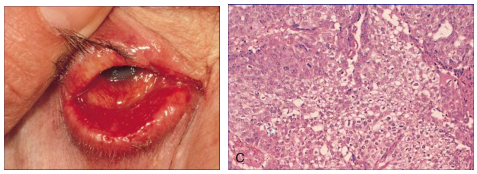


Pleomorphic adenoma

Lacrimal gland fossa lesions

Orbital pseudotumor	duration days to chronic	painful- yes	Ultrasound reflectivity: low	CT: localized or diffuse, molds to bone and globe	Management: systemic steroids, XRT
lymphoma	months	no	low	homogenous, oblong, molds to globe/bone	XRT, CTX (systemic disease)
pleomorphic adenoma (benign mixed tumor)	often > 1 year	no	medium to high, regular internal structure	well circumscribed, globular, possible bony expansion or excavartion	complete excision with capsule without biopsy
Adenoid cystic carcinoma, malignant epithelial tumors	< 1 year	yes (perineural invasion)	medium to high, irregular internal structure	round to oval mass with bony erosion	incisional biopsy, await permanent sections; exenteration

Eyelid Tumors:

Basal Cell Carcinoma	Squamous cell	Sebaceous Adenocarcinoma
• 90-95% of malignant eyelid tumors • Lower lid and medial canthal areas • Nodular and morpheaform types • Medial canthal lesions can be problematic • 3% mortality  	40 times less common than BCC More aggressive, associated with perineural invasion. Most arise from pre-existing lesions It has variable presentations 	• Highly malignant • 2x more common in the upper lid • Multicentric • Separate upper and lower lid lesions in 6-8% • Pagetoid spread 

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.

Done by: Shadn Alomran, Lina Ismael.
Edited by: Lamya Alsaghan.
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.**

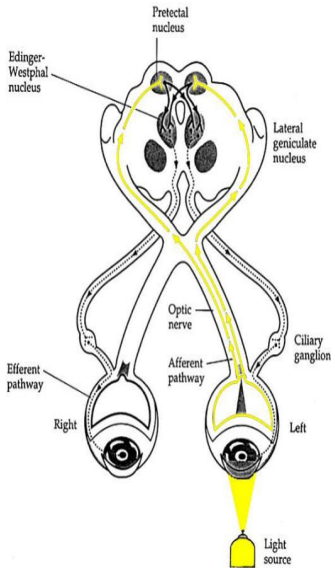
*** Special thanks to Shadn Alomran for her effort!**

Physiology

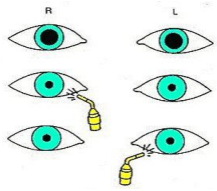
Extra from 435 team *the professor did not mention it but it is very helpful*

– Pupillary light reflex pathway

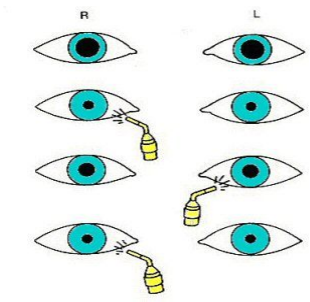
- It is very important to understand the pathway to locate the neurological pathology.
- Pupillary light reflex, which allows for the constriction of the pupil when exposed to bright light. This reflex serves to regulate the amount of light the retina receives under varying illuminations. The pupillary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

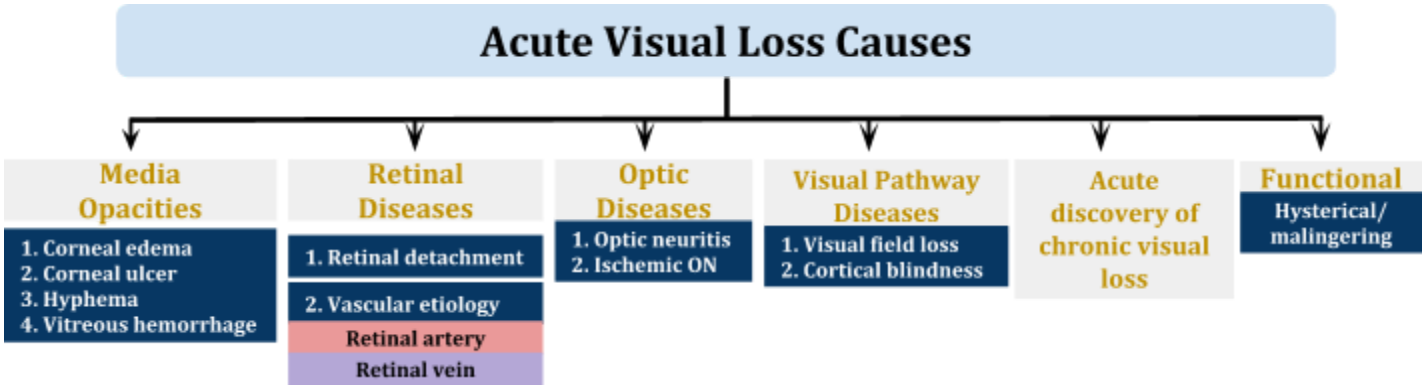
• What is acute visual loss (AVL)? (disastrous and requires urgent actions)

– 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.

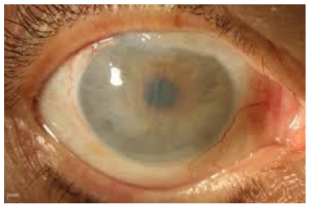
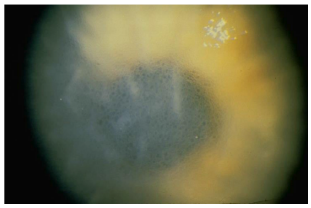


Media Opacities

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

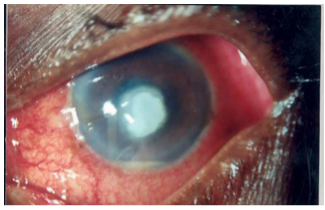
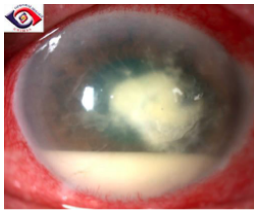
1. Corneal edema

– 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.

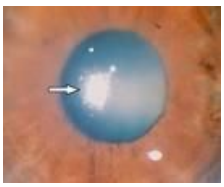
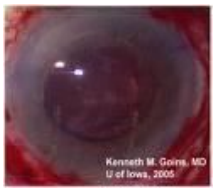
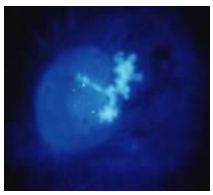
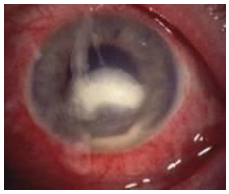
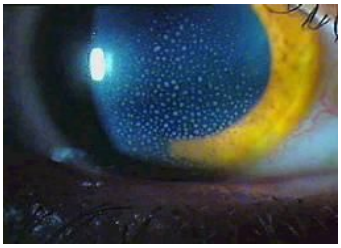
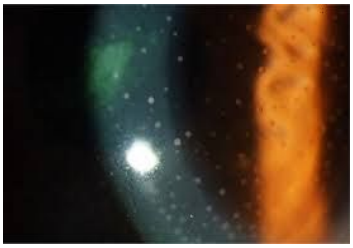
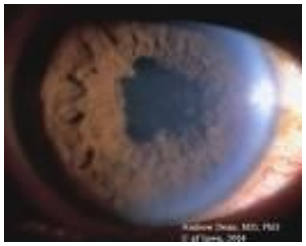
2. Corneal ulcer

– 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.

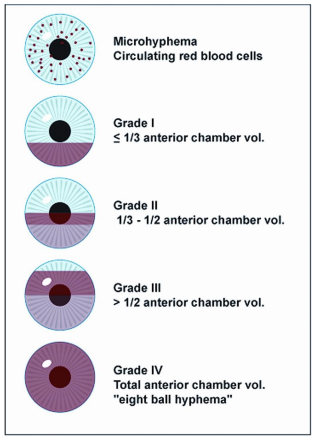
– Uveitis doesn't only cause visual impairment on the corneal side but also on the turbidity of the anterior chamber. In uveitis the inflammation leads to changes in aqueous humor contacts, usually there is a protein present in the anterior chamber and its concentration is 1% of that in the serum. In 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.

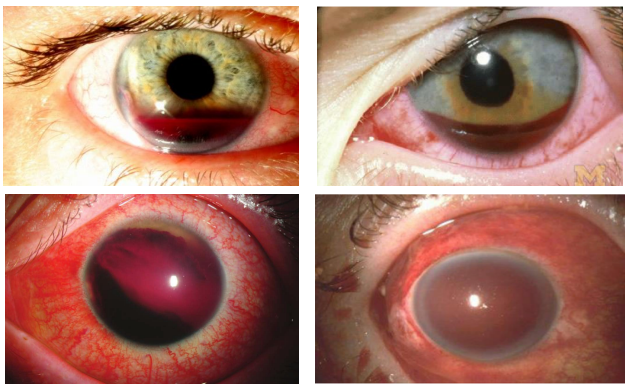
			
Abscess	**Edema**	**Viral herpetic herpes**	**1) Infection: abscess in the cornea with hypopyon[3].** 2) **Congested eye.**
			

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.

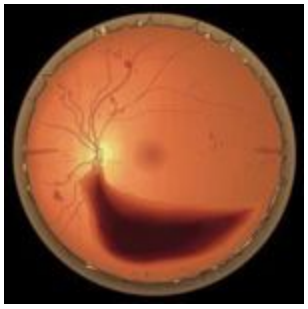
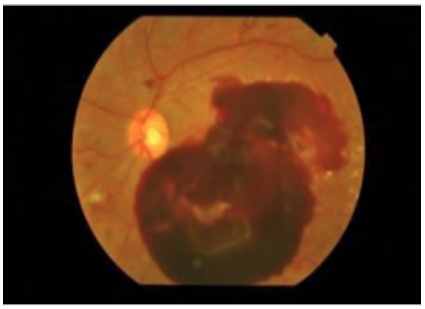
4. Vitreous hemorrhage (VH)

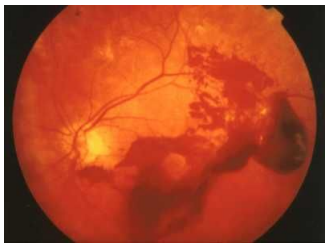
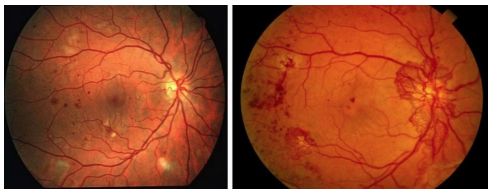
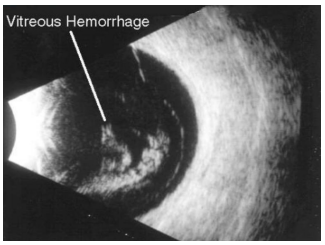
Mechanism

– 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)

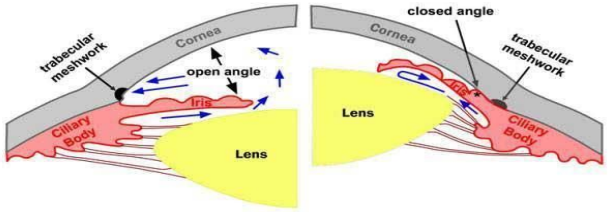
Features: patients who are **prone** to develop acute angle closure glaucoma have unique features:

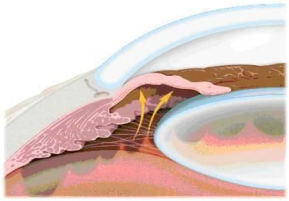
Shorter eyes "Axial length" (within the normal range). | Hyperopic vision. | Large lens.

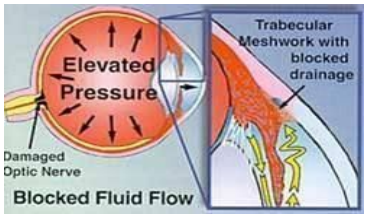
Mechanism

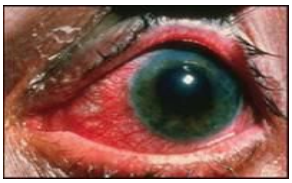
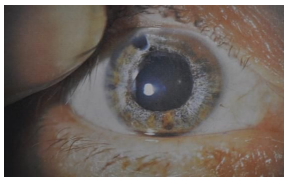
The most common mechanism is **pupillary block:** This occurs when the lens plasters up against the back of the iris, blocking aqueous flow through the pupil. **This resistance produces a pressure gradient IOP goes up** (this is the keyword) across the iris that forces the iris and lens to move anteriorly 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.

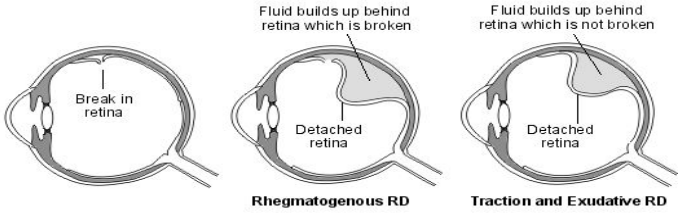
[5] Acetazolamide is administered intravenously and subsequently orally together with topical pilocarpine and beta-blockers. Pilocarpine constricts the pupil and draws the peripheral iris out of the angle; the acetazolamide and beta-blocker reduce aqueous secretion and the pressure across the iris. These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

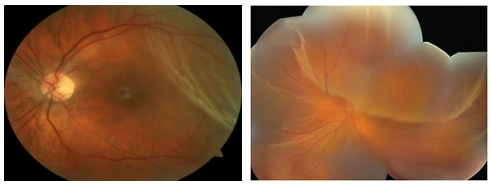
Signs & Symptoms

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.**

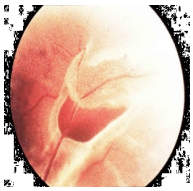
– The diagnosis is confirmed by ophthalmoscopy through a dilated pupil, and retina appears elevated with folds and the choroid background behind the retina is indistinct.

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)

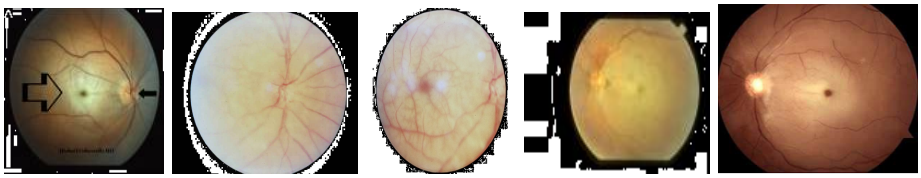


Horseshoe break:
f the break gets pulled more (by the vitreous) → can rupture a vessel → RD & hemorrhage

2. Retinal vascular occlusions

Artery

Central Retinal Artery occlusion

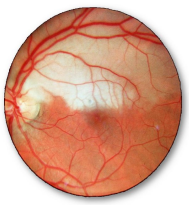


– A sudden, **painless** and often complete visual loss may indicate central retinal artery occlusion.
– Several hours after a central retinal artery occlusion, **the inner layer of the retina becomes opalescent (opaque, why? Bc of edema).**
– A cherry red spot is seen due to the pallor of the perifoveal 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

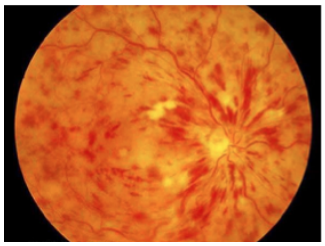


***Picture:** notice how white the affected retina (above) is compared to the normal retina. The ischemia stopped just short of the macula so this patient most likely didn't have his vision completely affected (but his lower visual field is gone)
Upper retina: lower. Lower retina: Upper Temporal: nasal.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

Vein

Cental retinal vein occlusion

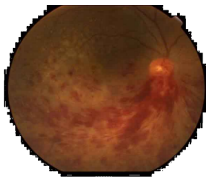
– Ophthalmoscopes picture of disc swelling, venous engorgement and tortuosity, **cotton wool spots (these are more white than exudates, and they are an infarctions of the nerve fiber layer) and diffuse retinal hemorrhages** like blood and thunder (flame shaped).
– Loss of vision may be severe. Bc it 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.



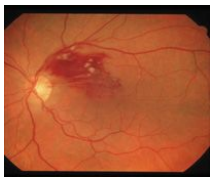
Types

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**

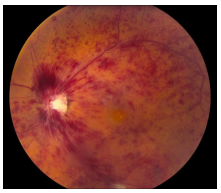
The way to differentiate between the two types is by visual acuity, and pupillary reflex, in ischemic type there must be an afferent defect.



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

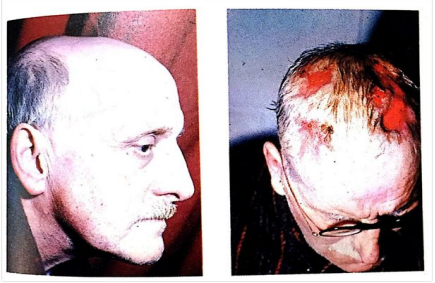
Optic Nerve Diseases

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)

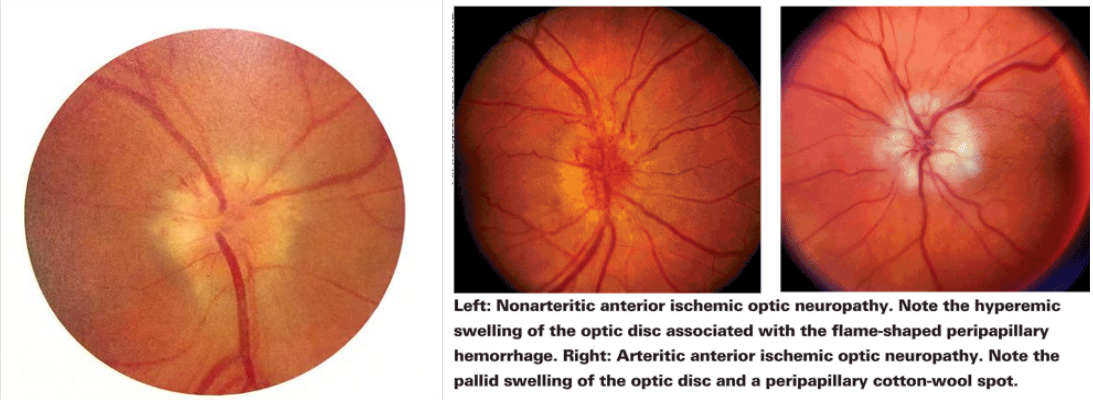
	Extra information: difference between		
	Papilledema	**Papillitis**	**Retrobulbar neuritis**
Definition	Swelling of optic nerve head due to increased ICP	Inflammation or infarction of optic nerve head	Inflammation of orbital portion of optic nerve
Uni/bilateral	Bilateral	Unilateral	Unilateral
Vision 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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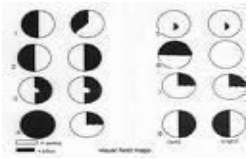
1. Homonymous hemianopia



– 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

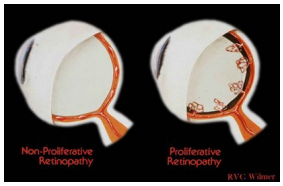
The doctor explained this briefly but said it's important, I tried to simplify it to the best of my abilities

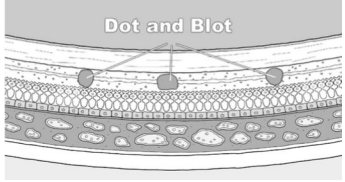
– Diabetic retinopathy is the term used to describe the retinal damage causing this visual loss. Diabetics have a high prevalence of retinopathy, and one out of every five patients with newly diagnosed diabetes will also show signs of retinopathy on exam.

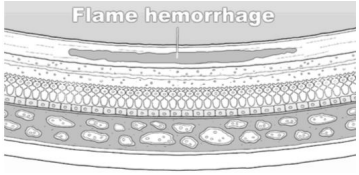
– **Mechanism of Vessel Breakdown**

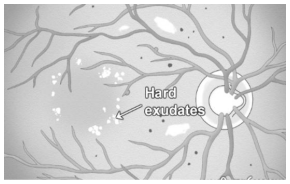
- Diabetes is a disease of blood vessels. With large amounts of glucose coursing through the circulatory system, a glycosylation reaction occurs between sugar and the proteins that make up blood vessel walls. Over time, this reaction promotes denatures the collagen protein within the walls, creating 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.







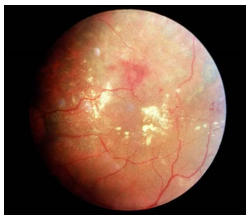


– **Retinal detachment in DM: i**n the areas where the neovascularization happens, fibroproliferation ensues and pulls on the retina.

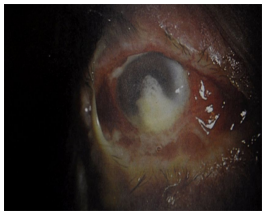
– **Treatment of DR (diabetic retinopathy)**

- 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.

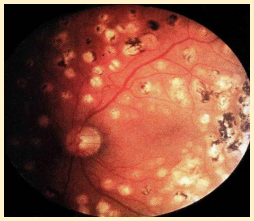
– **Complications:** visual field loss (tunnel vision)



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.

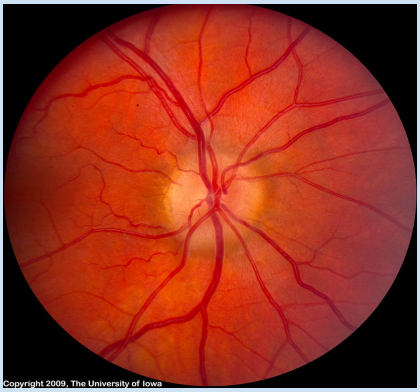
Answer: c

[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

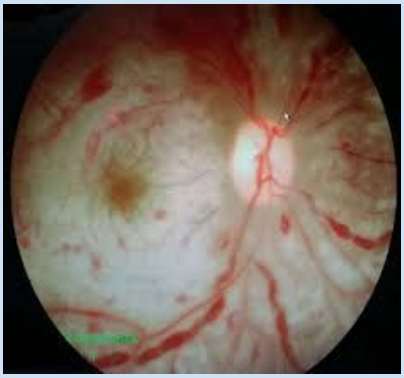
– **What is this?**
Leukocoria in a child

– **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

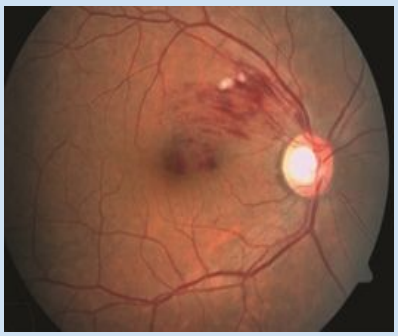


– **What is this?**
Disc edema

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

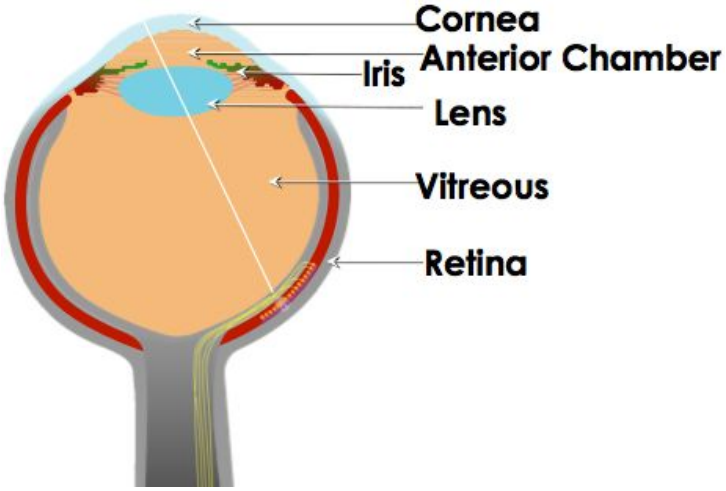


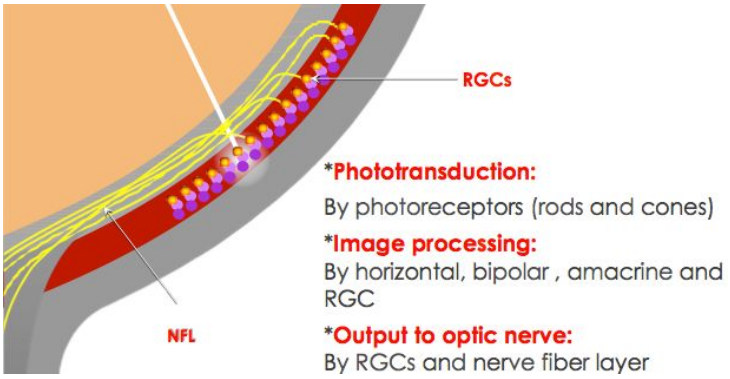
– Concurrent Central Retinal Artery and Vein Occlusion
– You can see a pale retina, a cherry red spot (artery)
– Cotton wool spots, flame shaped hemorrhage (vein) and if you follow the artery you can see points of occlusion

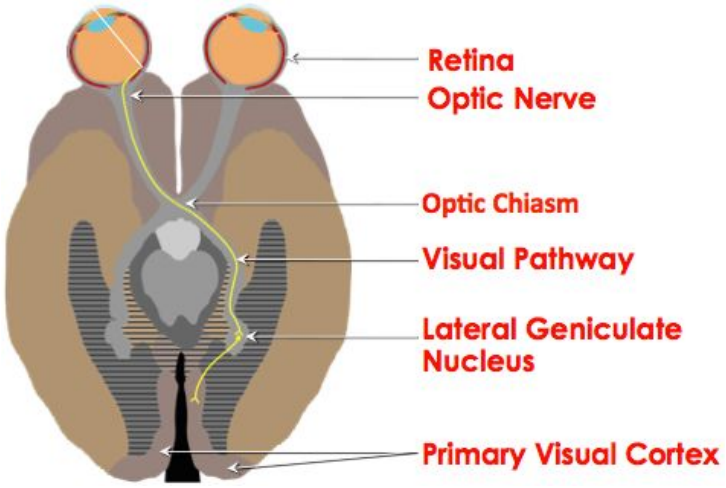


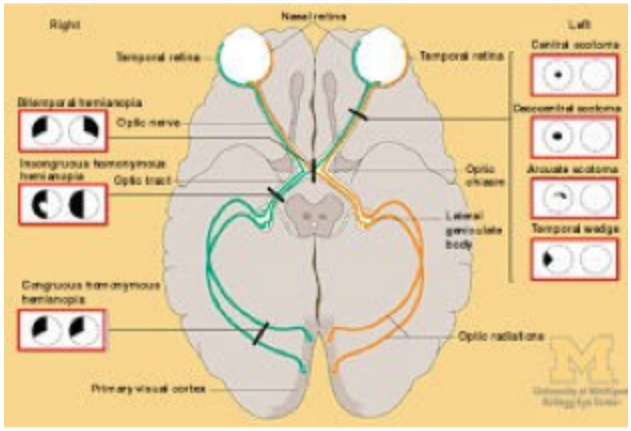
– Branch retinal vein occlusion (the whitish spots are cotton wool spots)

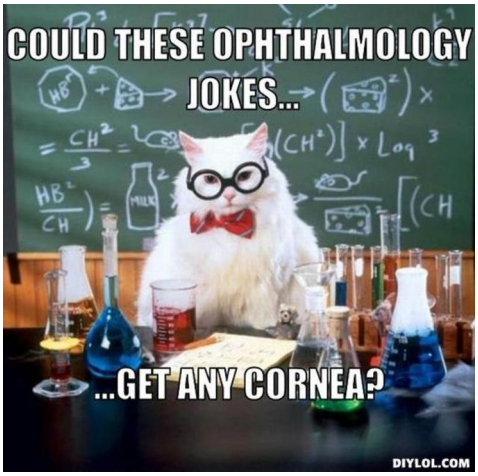
Extra from Dr. Almousa slides *the professor did not mention it but it is very helpful*











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

Strabismus, Amblyopia & Leukocoria

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

Objectives:

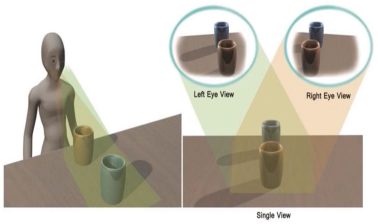
➢ Not given.

Done by: Jwaher Alharbi, Farrah Mendoza.
Revised by: Rawan Aldhuwayhi
Resources: Slides + Notes + 434 team.

NOTE: F1& F2 doctors are different, the doctor who gave F2 said she is in the exam committee so focus on her notes

Amblyopia

- **Definition**
  Decrease in visual acuity of one eye <u>without</u> the presence of an organic cause that explains that decrease in visual acuity. He never complaints of anything and his family never noticed any abnormalities
- **Incidence**
  The most common cause of visual loss under 20 years of life (2-4% of the general population)
- **How?**
  Cortical ignorance of one eye. This will end up having a lazy eye
- **binocular vision**
  It is achieved by the use of the two eyes together so that separate and slightly dissimilar images arising in each eye are appreciated as a single image by the process of fusion.
  It's importance 1. Stereopsis 2. Larger field
  If there is no coordination between the two eyes the person will have double vision and confusion so as a compensatory mechanism for double vision the brain will cause suppression.

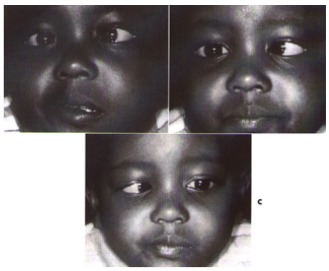


  The visual pathway is a plastic system that continues to develop during childhood until around 6-9 years of age. During this time, the wiring between the retina and visual cortex is still developing. Any visual problem during this critical period, such as a refractive error or strabismus can mess up this developmental wiring, resulting in permanent visual loss that can't be fixed by any corrective means when they are older

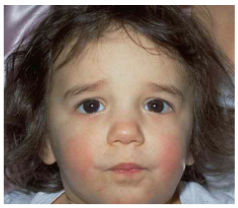
  Why fusion may fail ?
  1. significant anisometropia. Most common cause
  2. significant aniseikonia (difference in image size)
  3. Strabismus. 2nd common cause
  4. Higher cortical problems (head trauma, alcohol intake , drugs) rare in children
- **Diagnostic criteria**
  The only way to discover lazy eye is to take your child to an ophthalmologist
  - VA <20/40 OU (Latin term oculus uterque=both eyes) or in one eye.
  - Family history
  - History of visual deprivation during infancy
- **Classification**

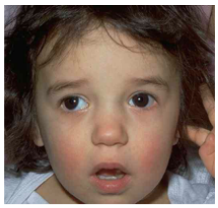
1- Strabismic

More details at the end of the lecture!

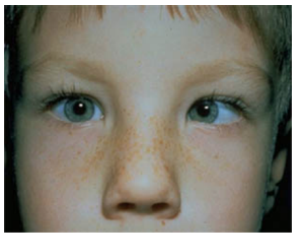


Alternating esotropia
this child is lucky he will not develop amblyopia
but we have to treat this child by surgery





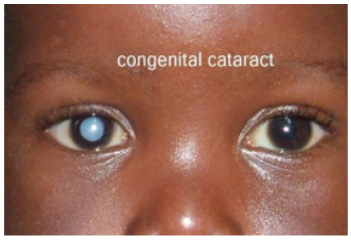
Intermittent Exotropia
in the first picture the child looks fine but in the second picture there is exotropia
this child will not have amblyopia
when it is fixed the child will develop amblyopia



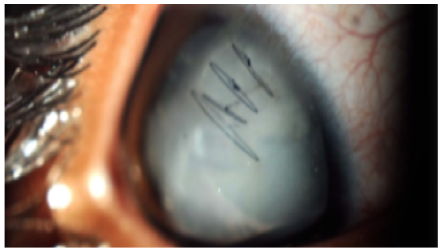
Esometropia (the most common cause of esometroipa is Hypermetropia)

2- Deprivation amblyopia

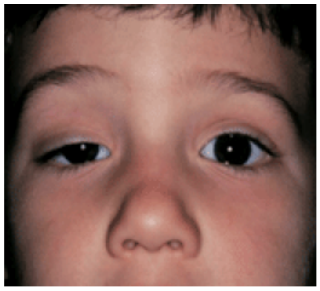
Results in more severe visual impairment than strabismic or refractive amblyopia



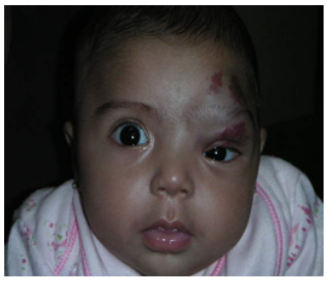
Congenital cataract
That's why we have to treat
it in the first 3 months of life



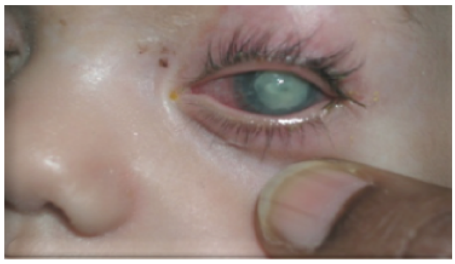
the cornea is sutured because of trauma
so this child developed cataract



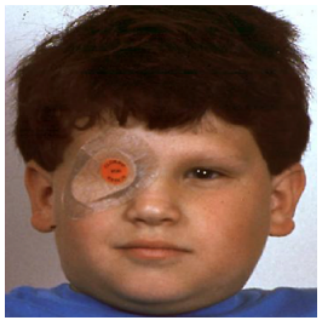
Congenital ptosis
we have to do lid surgery otherwise
he will develop amblyopia



Capillary hemangioma
it is treated with beta-blockers



Corneal infection (abscess in the cornea)
he took 2 months to respond to treatment
so during this 2 months he developed amblyopia



3- Anisometropic amblyopia

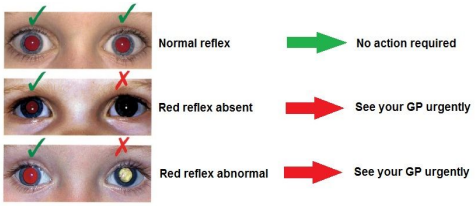
- Also called (Refractive amblyopia)
- The most common cause of amblyopia in children is difference in refraction more common with hypermetropic anisometropia
- For hypermetropia 1.5 diopter difference will cause amblyopia but for myopia 3 diopter difference will cause amblyopia

Treatment of amblyopia

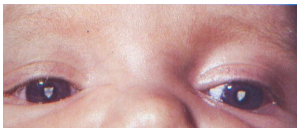
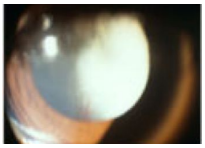
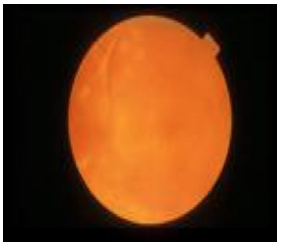
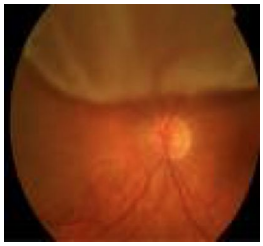
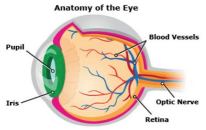
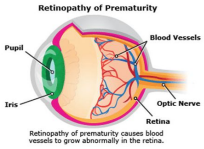
1. Treat the cause of amblyopia:
   correct the refractive error(optical correction) , remove the media opacity surgically
2. The younger the child , the better out come of amblyopia therapy
3. The first five years of child age is the sensitive period where amblyopia can be reversed
   after that it become more difficult
4. Occlusive therapy(PTO:part time occlusion) patch the good eye for 3 hours daily
5. Defocusing (penalization:optically by adding+3 to +5 lens on top of full cycloplegic refraction(atropine every 1-3 /7 in good eye)).انتبهوا مره سألناهم عنها وماعرفوا يجاوبون,نسويها اذا المريض رفض يحط باتش, ايش نسوي؟نحط قطرات سايكلوبليجك بالعين السليمه فتصير الرؤيه فيها مغبشه فكذا ينجبر انه يناظر بالعين الثانيه

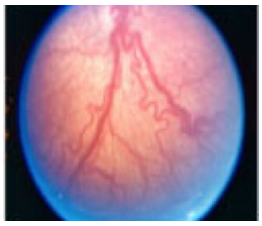
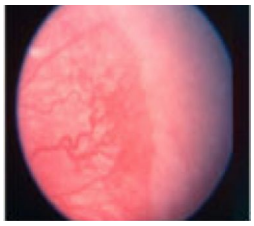
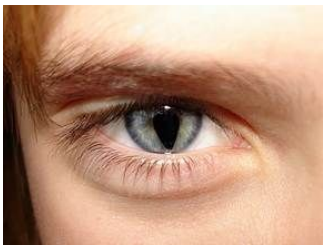
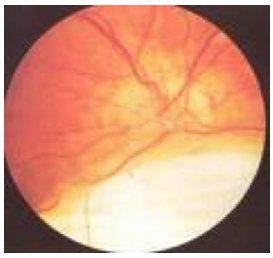
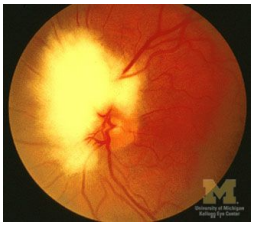
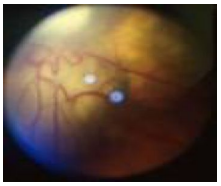
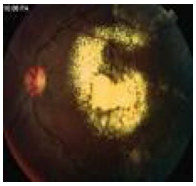
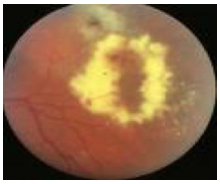
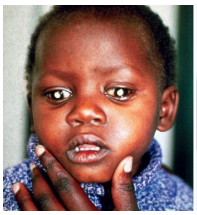
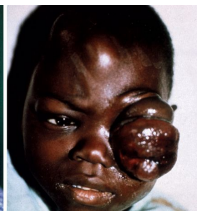
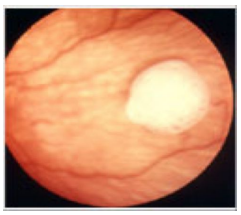
Leukocoria[EXAM]

- **Definition:** White opacity of the pupil
- If there is opacity and you can't examine the fundus you have to do ultrasound
- The red reflex comes from the retinal pigmented layer
- If you have any problem in the eye starting from cornea, anterior chamber, lense, vitreous and retina you will have Leukocoria



اقولكم ايها من الحين 100% لوكوكوريا بتجي بالإختبار بيطلب منك تعريفها و هاتي 3 اسباب لها *ساك و لله اعلم*

Cause	Description
Cataract can be congenital or acquired, usually causes blurred vision and glare Use the ophthalmoscope to see the red reflex	 
Persistent hyperplastic primary vitreous is a congenital condition caused by failure of the normal regression of the primary vitreous. It is usually associated with unilateral vision loss There is a connection between the optic disc and the lens during development and this connection used to supply blood vessels to the lens and it should go away this will cause: 1. Leukocoria 2. The globe will shrink (Small eye compared to the other)	  
Organized vitreous hemorrhage is usually secondary to a neovascular membrane or to a retinal tear. Patients may complain of a red haze, blurred vision, or floaters. As it starts to resolve, color changes to yellow then white and some fibrous sheets may persist. A B-scan (Brightness scan) is usually diagnostic and vitrectomy is usually required The most common cause of vitreous hemorrhage in babies is birth trauma	 
Retinal detachment risk factors include trauma and surgery, vitreous detachment, high myopia, retinal breaks or tears, retinal vascular disease, and history of detachment in the other eye. symptoms include flashes of light, floaters, curtain-like decrease in vision The retina is composed of many layers, the neuro-sensory retina will be separated from the retinal pigmented epithelium so you will not see the color of the retinal pigmented epithelium this will result in white opacity if it is big enough it will give you leukocoria	
Retinopathy of prematurity occurs in premature, low-birth-weight infants maintained on oxygen therapy. Signs include neovascularization, fibrous bands, retinal detachments and vitreous hemorrhage.	 

When advanced leukocoria can be present Prematurity in ophthalmology means that the child weight is 1.5 kg or less and the GA is 25 weeks or less The retina does not have enough time to have complete blood supply (the vessels will keep growing during pregnancy until 37 week) so ischemia will occur and and the body react to ischemia by formation of new blood vessels but these vessels are fragile so they will bleed and when they bleed they cause fibrosis and fibrosis will pull the retina and causes detachment The treatment is easy just go and kill this demand by burning the retina by laser to save the fovea and macula and definitely the visual field of this child will be affected	 
Coloboma congenital condition caused by incomplete closure of the the fetal fissure. Degree of visual loss to area affected (iris, retina, choroid, or optic nerve head The child is born without retina and retinal blood vessels are also absent just sclera Does coloboma causes problems? It depends if the macula and fovea are involved this will result in poor vision it it happens away from the macula and fovea the effect will be less Coloboma can affect the iris as well and will result in "keyhole" or “Cat eye” appearance In fundoscopy we see the sclera (white area)	 
Medullated nerve fibers congenital anomaly caused by myelination of the retinal nerve fibers and usually asymptomatic. When large areas are involved it can cause leukocoria In the reina the nerve fibers are not myelinated to allow the passage of light “Cat fur appearance”	
Coat’s disease typically a unilateral condition found in young boys. It is characterized by retinal telangiectasia and aneurysms that may cause exudative retinal detachment If we discover it early we can go and close the blood vessels by laser, the diagnosis is not by examining the retina only we inject the fluorescein stain	  
Retinoblastoma most common primary, malignant, intraocular tumor of childhood but still a rare tumor. Vast majority become apparent before age of 3yrs. It results from malignant transformation of primitive retinal cells before final differentiation. Presentation is most commonly (60%) with leukocoria and strabismus When you see Leukocoria always assume that this child is having retinoblastoma until proven otherwise (You should send the patient for B-scan ultrasound)	  

Strabismus

- **Ocular misalignment due to extraocular muscle imbalance**
- **Strabismus occurs in approximately 3% of children under 3 years of age**
- **Males and females equally affected**

  Strabismus is bad not only because of cosmesis it is related to amblyopia

- Tropia: الحول و اضح ينشاف misalignment that is always there, even when both eyes are open and attempting to work together. Large angle deviations are obvious. If small angle, you can detect it with the Cover-Uncover test.
- Phoria: الحول موب و اضح مايبين لك الا من الاقز امنيشن misalignment that only occurs some of the time, usually under conditions of stress, illness, fatigue or when binocular vision is interrupted i.e. when the two eyes are no longer looking at the same object such as when the synchronization between the eyes is broken by covering one eye. You can "break fusion" using the Cross-Cover test.

Causes:

- **Inherited** pattern (Most patients fall under this category, so it is important to ask about family history)
- **Idiopathic**
- **Neurological** conditions (cerebral palsy, hydrocephalus & brain tumors).
- **Down** syndrome
- A **congenital** cataract, eye tumor

Tests for deviation (how to test strabismus): F2 notes!

1- Hirschberg test (Corneal light reflex) please know how to do it very well because you will have it in the osce

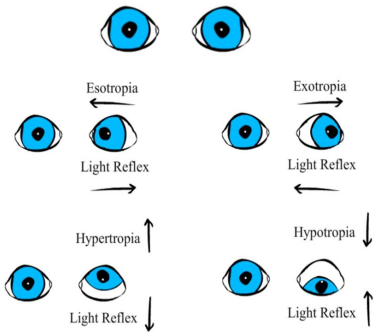
A test used to assess alignment of the eyes by shining a light in the person's eyes "1 meter away" and observing where the light reflects off the corneas. Every millimeter the corneal light reflex is off center, equals approximately 15 diopters of prism or 7 degrees احفظو ها هالنقطه مهمه.

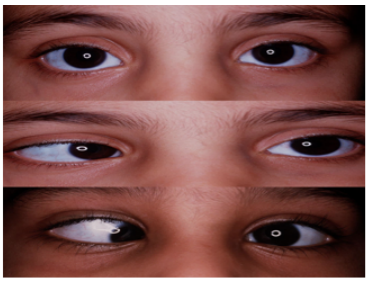
Note: roughly if corneal reflex is:

- At the pupil edge = 30 PD (15°)
- Midway between pupil and limbus = 60 PD (30°)
- At the limbus = 90 PD (45°)

When the corneal reflex is:

- in the center we call it orthoptic and that's the normal case.
- Light is shifted temporally or laterally → esotropia (inward deviation).
- Light shifted nasally → exotropia (outward deviation).



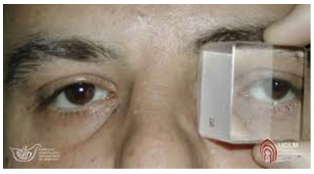


Top picture: Orthophoria (normal)
Middle picture: Esotropia ~30 PD (pupil edge)
Bottom picture: Esotropia ~90 PD (limbus)

Esotropia (inward deviation)light is shifted temporally

2- Krimsky test

The Krimsky test is essentially the same as Hirschberg test, except that we quantitate it better by using a prism. The prism is placed in front of the deviating eye and is used to move the light (corneal) reflex to the center of the pupil. The apex of the prism is directed towards the direction of deviation i.e. laterally if exotropic and medially if esotropic.

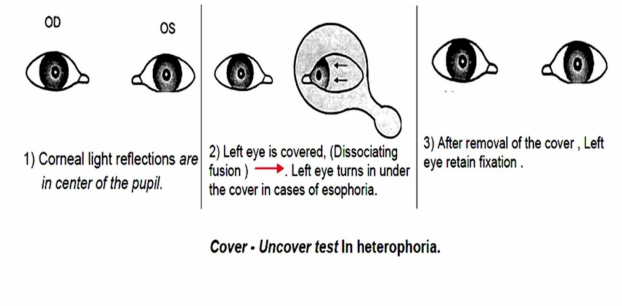


Krimsky test

3- Cover test(most important test) Watch this video and you'll understand everything!

There are 2 types of cover tests:

- **Unilateral** cover test (cover-uncover test): performed by having the patient focus on an object then covering the fixating eye and observing the movement of the other eye. If the eye was exotropic, covering the fixating eye will cause an inwards movement; and esotropic if covering the fixating eye will cause an outwards movement. It is used to detect tropias.
- **Alternating** cover test (cross-cover test): performed by moving the occluder from one eye to the other eye. Normally, the covered eye shouldn't move when the occluder is removed. It is the most accurate way to pick up subtle phorias and tropias since it breaks binocular vision.



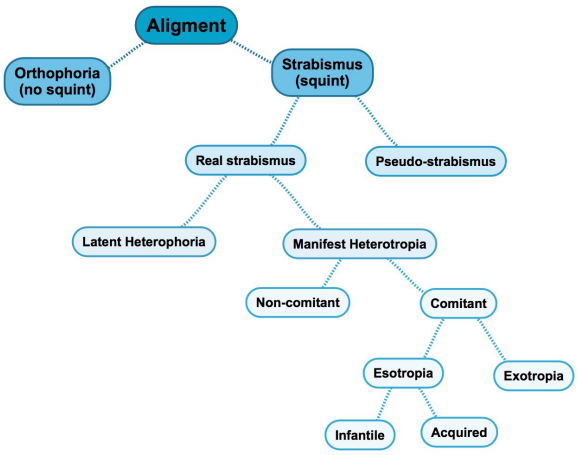
Cover - Uncover test In heterophoria.

EXAM دايما يسألون عنها بالاختبار

★ cover-uncover test → detect tropias

★ cover-cover test → detect phorias **and** tropias

• Classification:

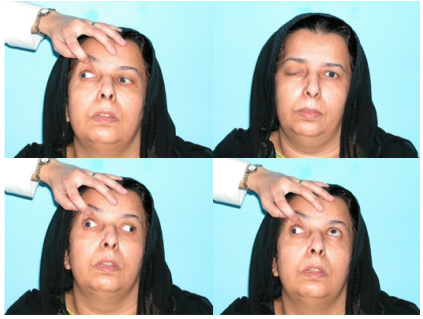


Comitant (XT or ET)	Non-comitant (XT or ET)
Angle: almost the same in any direction of gaze(within 10 PD range)	Angle: changes with direction of gaze (Paretic as in 6th and 3rd nerve palsy, restrictive as in thyroid disease)
Comitant strabismus ET20 ET 25 ET30 ET25 ET30	Non-comitant strabismus ET 20 ET 10 ET 12 ET40 ET18

Comitant: The angle of deviation has not changed whether the child looks up, down, left and right

Incomitant: Diabetes is usually associated with 6th nerve palsy, when the patient looks at you he looks normal you will notice that the patient has problem when he looks to the action of the paralyzed muscle (Occurs in certain gazes)[doctor said: forget about the incomitant type, it's not our topic today]

He is looking to the left
The right eye is working
But the lateral rectus is the left eye is not working
Probably he is having 6th nerve palsy
(non-comitant or paralytic strabismus)

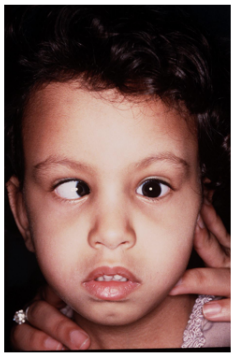


Classical picture of right third nerve palsy
1. ptosis
2. Large exotropia

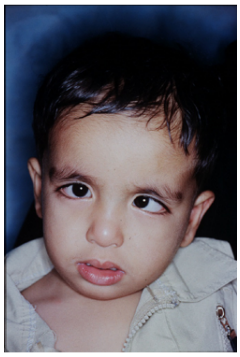
- **Infantile esotropia:**

- Is the inward deviation of the eyes noted before the patient reaches age 6 months (we don't say congenital because congenital means the child is born with this problem but here the child is born normally but in the first 6 months he developed esotropia)
- When the eyes are misaligned in childhood, binocular vision, or the ability of the brain to use the two eyes together, does not develop.
- Classic infantile esotropia is constant and involves a large angle of deviation exceeding 20 prism diopters (PD) on corneal light reflex measurement (we measure the deviation using the prism)
- **Infantile esotropia may be associated with a spectrum of clinical presentations, including:** amblyopia, impaired binocularity(impaired 3D vision "stereopsis"), central scotomas, Cross fixation (is the use of the right eye to view the left visual field and the use of the left eye to view the right VF), DVD(dissociated vertical deviation, in cover test: when u cover the eye then uncover it the eye will move up and down), latent nystagmus, and inferior oblique overaction(IOOA)(when looking up and in the eye will بترتفع مره لدرجة ان العدسه كلها تدخل لجوا)

Alternating esotropia
So this child will not have amblyopia



Right esotropia



Left esotropia

Management of infantile esotropia:

1. Treat the amblyopia by occluding the good eye.
2. ALWAYS Surgery for the extra-ocular muscles الحول هنا مستحيل يروح بدون جراحه (we go to the medial rectus and we detach it and then suture it backward by suturing the muscle backward we make it redundant according to severity of the strabismus the more severe the more we go backe and this procedure is called recession)surgery should be done at age of 10 to 11 months to achieve the binocular single vision
   the doctor said u don't have to know how it performed

Ophthobook

Treatment of strabismus Before taking anyone to surgery, correct all the non-surgical causes of strabismus: check for refractive error and treat any amblyopia. Many cases of strabismus will improve or resolve by just doing these things. Eye surgery consists of shortening or relaxing the extraocular muscles that attach to the globe to straighten the eye.
Strabismus surgery: To correct simple esotropias (cross-eyed) or exotropias (wall-eyed) we can weaken or strengthen the horizontal rectus muscles. A recession procedure involves disinserting the rectus muscle and reattaching the muscle to the globe in a more posterior position. This effectively weakens the action of this muscle and turns the eye accordingly.

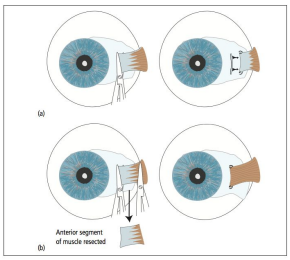
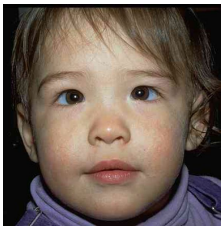


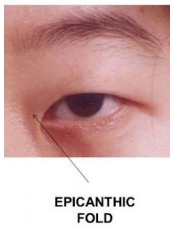
Figure 15.8 The principles of squint surgery. (a) Recession. The conjunctiva has been incised to expose the medial rectus muscle. The muscle is then disinserted and moved backwards on the globe. (b) Resection. Following exposure of the muscle the anterior tendon and muscle are resected, thus shortening them; the muscle is then reattached to its original position.

CASE: could come in the exam (MCQ)
A 4-month-old healthy child presents with a history of his eyes turning in most of the time, since about 8 weeks of age.How to approach this child:
1.history taking: family history, born at which week of gestation, mode of delivery
2.examination:

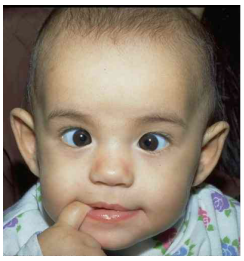
- Check the visual acuity, how to check the visual acuity in infant? fix and follow(following an object), CSM test "Central(no deviation) Steady (no nystagmus) Maintained(if u remove the cover can he maintained his eye in the center)".
- Check the extraocular movement:to rule out paralytic 6th nerve palsy, how to check?spinning ندور راسه اذا طبيعي عينه بتناظر على يرى باتجاه دوران الراس
- Check the amount of deviation: Hirschberg test, Krimsky test
- Check the refraction: cycloplegia is achieved by dilated drop called cyclopentolate(0.5% if child age less than 1, 1% if ≥1yo)
- Check for the associations that we just mentioned above:cross fixation, DVD, IOOA, and latent nystagmus
- Fundus exam:to check the optic nerve and retinal

- **Pseudostrabismus:**
  Is a condition in which alignment of the eyes is straight (also known as orthotropic) however, they appear to be crossed.
  This condition most commonly occurs in infants when a flat nasal bridge and prominent epicanthal folds tend to obscure the nasal portion of the sclera





- **Accommodative esotropia(very common):**
- Acquired after 6 months of age usually around 2 years
- Associated with **hyperopia** (Children with hyperopia will accommodate to see clearly and if he accommodate more he will converge more. In case of myopia it tends to produce exotropia due to lack of accommodation)
- Perform cycloplegic refraction on all children by using the retinoscope and loose lenses. Cycloplegia is achieved with Mydriacyl 1% and cyclogel 1%. (Cycloplegia will paralyze the accommodation and will give us the exact amount of refractive error)



- Refractive error usually from **+3 to +4** (not everyone with hyperopia will develop esotropia but they said that is more with clever children because they are interested to know the details of everything)
- May precipitated by acute illness or trauma
- Start intermittent and if not treated become constant
- Refractive error correction by **glasses** will treat the condition **FULLY** (<10PD residual near and distance)(If the farsighted glasses control the crossing of the eyes, eye muscle surgery is never recommended)

- **Partial Accommodative Esotropia:** F2 slides only!
- >10 PD residual for D+N(near and distance)with full hypermetropic correction.
- Treatment :Surgery for the **residual** deviation(do surgery for the part which not corrected by glasses فبعد الجراحه بيظل يلبس نظاره)

- **High AC/A ratio ET:** F2 slides only!
- **First what do we mean by AC/A ratio?** The accommodative convergence/accommodation (AC/A) ratio gives the relationship between the amount of convergence (in-turning of the eyes) that is generated by a given amount of accommodation (focusing effort).
- Esotropia with high AC/A ratio (also termed nonrefractive accommodative esotropia) ببساطه مشكلته انه ينحول اذا ناظر للقريب بس اذا ناظر للبعيد ماينحول
- **Treated by**: bifocal glasses should be bisecting the pupil

- **Divergence paralysis:** F2 slides only!
- ET at Distance > Near ينحول للبعيد بس اذا ناظر قريب ماينحول يكون مستقيم طبيعي
- Adult
- You should do urgent MRI to check for underlying cause could be: arnold chiari, pontine tumor
- First, treat the underlying cause then treat the esotropia with surgery

- **Sensory ET:** F2 slides only!
- ET due to unilateral blindness
- Treatment: Surgical usually for cosmetic purposes

- **Cyclic ET:** F2 slides only!
- Very rare
- Acquired (2-6yrs).
- Cycle between straight and ET (يجي ويروح)they are lucky because they won't develop amblyopia
- **Treatment:**
  - First if hypermetropia treat with glasses
  - if NOT hypermetropia treat by surgery

- **Exodeviation**
  The doctor said the only thing that you need to know about exotropia that it is not related to amblyopia (the other details from F2 notes)

a horizontal form of strabismus characterized by visual axes that form a divergent angle

Basic	Pseudo divergence excess	True divergence excess
• XT D=N • Tx: BLR weakening	• Xt D>N • Patch 30-60min • XT D=N • Tx: BLR weakening	• XT D>N • Patch 30-60min • XT D>N • Tx: R&R

Type	Definition
Basic	Distance and near measurements are equal.
Pseudodivergence Excess	Distance measurement initially exceeds near, but the near measurement approaches distance after 30–60 minutes of monocular occlusion.
True Divergence Excess	Distance measurement exceeds near measurement by ≥ 10 prism diopters even after monocular occlusion for 30–60 minutes.

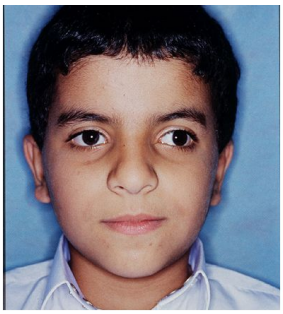
*XT: Exotropia *D:Distance *N:near *BLR:bilateral rectus *Tx:Treatment *R&R: Resection & Recession lateral rectus

- **Intermittent exotropia**

- Acquired.
- Early childhood.
- Intermittently controlled by fusional convergence.
- Close one eye in the bright light.b/c in bright light they can't fuse so to prevent diplopia they tend to cover one eye.

  Treatment:
  - First start with Non-surgical: alternate patching, over minus lenses(inducing accommodation to keep the eye in)
  - surgical involves lateral rectus muscle

  Surgery indications:
  - poor control.
  - The deviation occurs more than 50% of time.
  - Lost distance stereopsis.


This deviation may later progress to constant exotropia

- **Congenital exotropia:**

- Very rare.
- Constant large angle between the two eyes which is assessed by the prism
- Poor fusion prognosis
- High risk of amblyopia
- Associated with craniofacial abnormalities, albinism, Cerebral Palsy
- **Treatment:** Bilateral rectus muscle (BLR) Weakening.

- **Sensory exotropia:**

- Blind eye drifts outward
- **Treatment:** Surgery (cosmetic purpose)

- **Convergence insufficiency:**

- Inability to maintain the convergence on objects approaching from distance to near (moving pencil from distance to near your eyes will cross normally. In such patient their eyes will drift out)Exophoria X or exotropia XT at near N, Straight at distance D.
- Seen in elderly
- Symptoms: asthenopia (frontal headache), diplopia (when they look at near)
- Remote near point of convergence (normal 5-6 cm).
- **Treatment:**orthoptic exercise(pencil push up)