

# Loss of Vision

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Slides adapted from Dr. Gail Feinberg's 2021 Lecture

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Oroville 70 mi north of Sacramento

Chair for 3 yrs

not very available for office hours

very image heavy - starring certain things for exam and for ophthalmology in general

# Objectives

- Discuss the differential for loss of vision.
  - Identify eye conditions appropriate for primary office management vs. those that require referral to Ophthalmology.
  - Recognize the key features and approach to management of these conditions.
- ID eye conditions appropriate for you to deal with and when you need to consult

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There are 18k ophthalmologists in the USA, approximately 3% of all physicians in America. There are often geographic and insurance limitations as well. You need to advocate for your patient!

# Loss of Vision - Key Questions

- Duration?
  - Greater or less than 24 hours
  - Gradual loss or Sudden
- Pain?
  - Yes or No
- Adult or Pediatric - (NOT a key question, but will discuss pediatric at the end of lecture...)

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Keep in mind that many patients will need clarifying questions to determine the extent of vision loss. Total vision loss is very different than “everything is blurry up close until I move things further away from me”

# Loss of Vision: Key Questions

- Duration
  - Transient
    - Optic Nerve swelling
    - Amaurosis fugax
    - VBA insufficiency
    - Migraine
    - Optic nerve drusen
    - Orbital lesion

color coded - way to study

# Loss of Vision: Key Questions

Duration > 24 hours

## Gradual onset

- Refractive error
- Corneal dystrophy
- Cataract
- Glaucoma
- Macular degeneration
- Diabetic retinopathy
- Optic neuropathy

## Sudden onset

- Painful
  - Acute closure glaucoma
  - Corneal ulcer
  - Uveitis
  - Optic neuritis
  - Post traumatic
  - Endophthalmitis
- Painless
  - Vitreous hemorrhage
  - Retinal detachment
  - Vascular occlusion
  - Ischemic optic neuropathy (GCA)
  - Stroke

# Duration: Transient

will not talk about last two as much in this lecture

Papilledema/Nerve swelling  
Optic Nerve Drusen  
Amaurosis Fugax  
Migraine  
VBA insufficiency  
Orbital lesion

VBA causes vision loss due to poor blood flow to the occipital lobe, will be better covered in stroke lecture. Orbital lesions can cause visual loss from compression of optic nerve or vascular supply.

[intermittent compression](#)

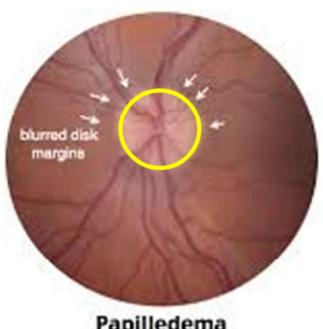
# Papilledema

- Swelling of the optic nerve from orbital congestion or transmission of elevated intracranial pressure
- Typically bilateral but may be asymmetric
- Loss of vision lasting seconds
- Pulsatile tinnitus
- May have afferent pupillary defect (APD; especially if asymmetrical swelling is present), loss of color vision, enlarged blind spot, visual disturbances (spots in vision), tunneling of vision

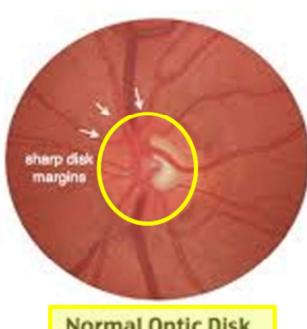
may hear heartbeat in ears

many reasons for optic nerve that looks swollen if not sure just say swollen not papil

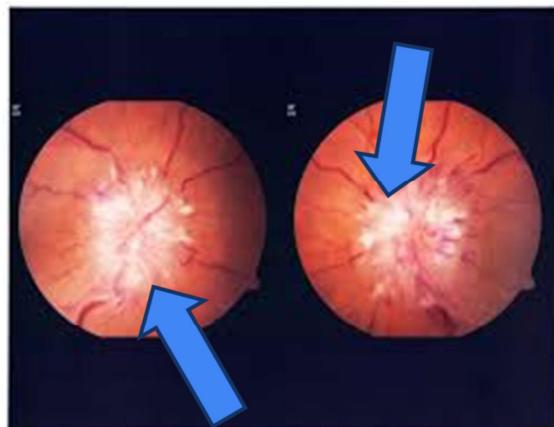
# Swollen Optic Nerve



fuzzy edges



Normal Optic Disk



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Average optic disc is 1.99mm vertical and 1.77mm horizontal. Average retinal artery at the disc margin is 80 microns (0.08mm) average retinal vein is 100um (0.1mm) at disc edge.

Note the title of this slide is “swollen optic nerve”. That is because not every swollen nerve is due to elevated intracranial pressure! If you use the term “papilledema” then the swelling is by definition due to increased intracranial pressure

when can't see the blood vessels at all the axons are so swollen they have lost their translucency

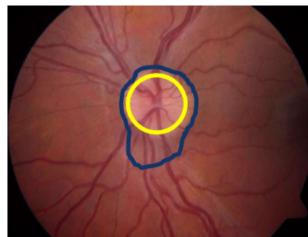
# Swollen Optic Nerve

- Check blood pressure in clinic [hypertensive emergency]
- Check vision need in order to refer to an ophthalmologist
- Refer to Ophthalmology to confirm swelling
- Brain MRI to rule out bleeding/intracranial mass, possible MRV if suspect venous sinus thrombosis
- If unilateral, detailed MRI orbits to assess for orbital mass
- If all negative, consider idiopathic intracranial hypertension - diagnosis of exclusion

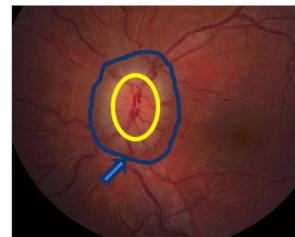
vessel is  
not  
straight  
and is  
bumped  
over  
swelling



mild



moderate



severe

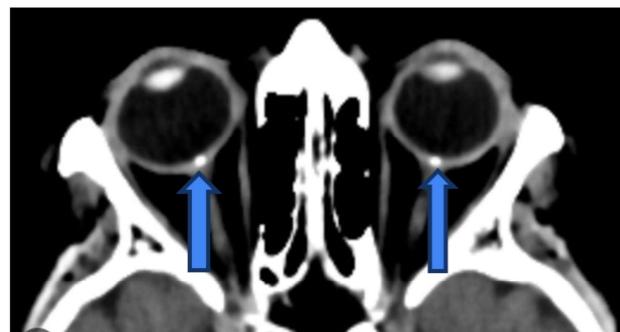
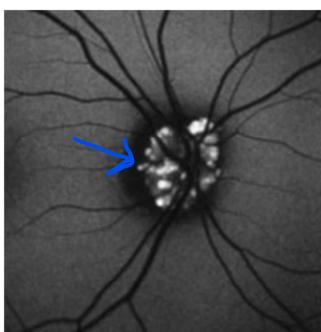
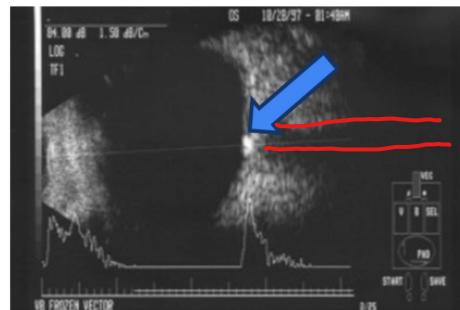
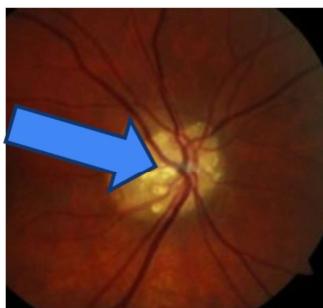
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young obese female - don't necessarily need to do LP each time but it's to exclude head bleed, mass, etc.

Yellow circles show the edge of the optic nerve rim. Note that in the mild swelling photo the cup is still visible but there is no discernable cup in the moderate or severe cases. The blue circles show areas of peripapillary swelling

Differential for swollen optic nerve includes infection (cat scratch, lyme, syphilis, viral meningitis) via direct infiltration or inflammation or intracranial pressure increase; inflammation (neurosarcoidosis, giant cell arteritis, non arteritic anterior ischemic optic neuropathy); toxic (many drugs including chemotherapeutic agents, antimicrobials, immunomodulators); autoimmune (multiple sclerosis and variants); compression (tumors with or without intracranial pressure increase)

# Optic Disc Drusen (pseudopapilledema)



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looks swollen but can see the vessels (not going up and over swelling) so axons not swollen

Clockwise from the top left: color photo, ocular ultrasound, CT scan showing calcifications at the optic disc, red free fundus photo demonstrating optic disc drusen autofluorescence. Optic nerve drusen (pseudopapilledema) are hereditary hyaline bodies in the optic disc head, possibly axonal byproducts. They are usually calcified and increase in size with age, the visual effects may be due to compression of vascular supply at the optic disc.

# Amaurosis Fugax

- TRANSIENT ischemic attack (TIA) of retina resulting in transient total monocular blindness, lasting minutes
- Most often from distant carotid or cardiac embolus that subsequently breaks up and vision returns quickly
- Treat as TIA equivalent with appropriate workup and referral

[stroke work up](#)

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Amaurosis is Greek meaning “darkening” or “obscurcation” and fugax is latin for “ fleeting” Once stroke is ruled out, still need ophthalmology referral because causes of total monocular vision loss (both < and >24 hours) are vascular (thromboembolic from carotid or cardiac source, GCA, vasospasm), neurologic (retinal migraine), or ophthalmalic (impending retinal vein occlusion, optic disc drusen, optic nerve swelling, orbital tumors )

# Ocular Migraine

- NOT true visual loss; rather, “scintillating scotoma” lasting minutes, sometimes hours,

doesn't black out completely  
aura before migraine often have 20 min before headache  
doesn't work that way for ocular migraine bc visual changes are ~20 min  
low dose ca channel blocker, propranolol if having multiple times a day



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typically no headache with “ocular migraine” but could be a visual aura preceding migraine headache. Etiology of migraines not fully understood, but thought to be due to vascular spasm, thus some treatments if refractory or disabling due to frequency are vascular stabilizers such as beta blockers or calcium channel blockers in lower doses than used for blood pressure control.

**Duration: > 24  
hours - gradual**

Refractive error  
Corneal dystrophy  
Cataract  
Glaucoma  
Macular degeneration  
Diabetic retinopathy  
Toxic optic neuropathy

# Refractive Error

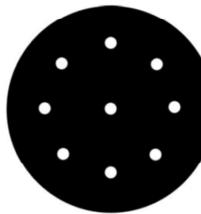
Leading cause of blindness worldwide

- Pinhole testing:
  - Vision is tested without correction
  - Vision is then tested with pinhole in place
  - If significant improvement, it is correctable with glasses or contacts

don't often need a full ophthalmologist, optometrist

- Presbyopia: inability of lens to focus for near vision
  - Starts at age 40-45, earlier for hyperopes
  - "my arms aren't long enough"
  - OTC reading glasses help

loss of flexibility in lens so cannot accommodate anymore



Pinhole aperture



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Ways to check in clinic: near card (Rosenbaum chart) and distance testing (Snellen chart, tumbling E.) Allen pictures are old and not terribly accurate. Pinhole helps by blocking out unfocused rays and eliminating divergent rays of light.

Per the WHO website, leading cause globally for vision loss is refractive error, then cataract, then diabetic retinopathy followed by glaucoma and AMD. Lancet has a study showing 35% vision loss cataract as much as 50% refractive error, AMD only <12%. Due to availability of cataract surgery in high income countries, Mac degeneration (19 million in USA, 1.5 million vision threatening) and glaucoma (3 million in USA) are more common cause of vision loss in >60 years but Diabetic retinopathy most common in 25-75 range. In low income countries refractive error and cataract most common. WHO report 2023.

# Granular Corneal Dystrophy

starts early on and increases



- Autosomal dominant, bilateral, noninflammatory condition
  - Results in deposition of irregularly shaped opacities in cornea by adulthood
  - Treated with either laser or corneal transplant
    - If erosions, treat to prevent worsening

most not visible without a slit lamp - this is the one kind easily visible without one

white, quiet eye (sclera and cornea) are clear in 40-0s so many deposits it starts breaking through epithelium tx with laser<sup>15</sup>

Corneal dystrophies can occur in the epithelial, stromal, or endothelial layers, many not visible without a slit lamp. This is one example that is visible without specialized equipment: in granular corneal dystrophy protein deposits (from keratocytes) appear as early as 1<sup>st</sup> decade, worsening with age but vision can remain good until 5<sup>th</sup> decade of life although may have glare and photophobia. Type 1 more common in Europe, Type 2 first described in Italian province of Avellino but more common in Korea, Japan. In US <1% of population. Can break through Bowman's membrane causing painful epithelial erosions. Recurrence after various superficial laser procedures around 3 years, with penetrating corneal transplant or lamellar transplant recurrence around 13 years.

# Cataract

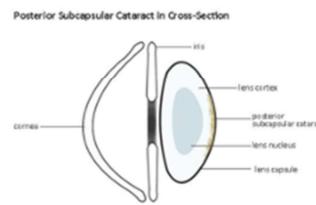
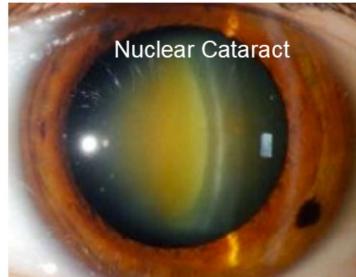
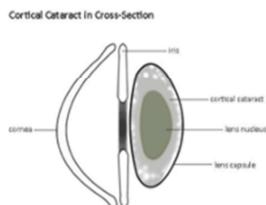
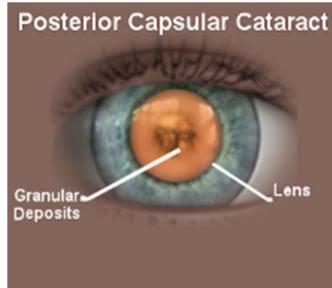
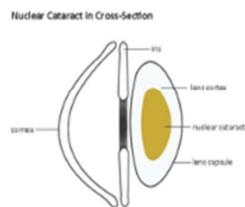
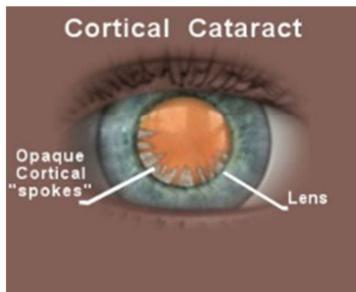
Second most common cause of blindness globally

- Opacified lens
- Slowly progressing visual loss or blurring over months to years
- Other s/sx include glare, decreased contrast sensitivity
- Risk factors for accelerated formation
  - Trauma
  - Diabetes
  - **Steroid use** lifelong asthma, autoimmune diseases, arthritis
- Only effective treatment: Surgical extraction with intraocular lens placement.
- “Secondary cataract” : Opacification of the capsule after cataract surgery; Treatment: Easily opened with laser.

patient won't complain about the same thing, seeing halo, losing contrast sensitivity - turning on all the lights, stop driving at night

Per the WHO website, leading cause globally for vision loss is refractive error, then cataract, then diabetic retinopathy followed by glaucoma and AMD. Lancet has a study showing 35% vision loss cataract as much as 50% refractive error, AMD only <12%. Due to availability of cataract surgery in high income countries, Mac degeneration (19 million in USA, 1.5 million vision threatening) and glaucoma (3 million in USA) are more common cause of vision loss in >60 years but Diabetic retinopathy most common in 25-75 range. In low income countries refractive error and cataract most common. WHO report 2023.

# Types of Cataract



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natural lens is completely translucent

**Cortical:** Starts at periphery and moves into center - slow vision loss until moves into center. Associated with glare and vision changes in different light settings. Common in diabetes (pathophysiology is swelling of lens fibers due to glucose or sorbitol, also deposits of protein causing opacity) [tends to start from edges and coming in](#)

**Posterior Subcapsular:** Can be due to age related changes - frequently seen with trauma, DM, steroid use, inflammation inside the eye. Begins centrally - very symptomatic. [may look granular](#)

**Nuclear:** Most common type in the U.S. Starts centrally and grows out to the periphery. As cataract grows/becomes more dense, lens becomes larger in size - changes prescription of glasses (if pt has glasses). In early stages, some patients may note improved ability to read without glasses (majority of cataracts cause myopia/nearsightedness). As condition progresses, lens loses the ability to focus light and vision becomes increasingly blurry. Proteins of lens (crystallins) undergo changes including coloration, oxidation, cross linking all of which affect translucency. Also lens grows from periphery, pushing older fibers to compact in the center (nuclear sclerosis)

[occurs over decades](#)

[if don't have dilating drops, turn lights down and don't shine like directly do from a few feet away to look for red reflex](#)

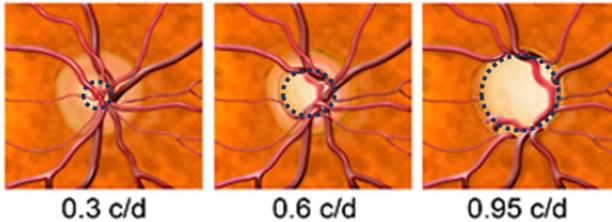
# Glaucoma

- Progressive optic nerve damage with a characteristic pattern of visual field loss. There are many risk factors.
  - **Intraocular pressure (IOP)** reduction and smoking cessation were the only known treatable risk factors, but recent meta data shows patients with obstructive sleep apnea may have a 40% increased chance of developing normal tension glaucoma especially unilateral glaucoma, mechanism unclear
  - Mechanism by which IOP damages the optic nerve is not completely understood.

most other risk factors  
can't do anything about -  
family history, diabetes,  
etc.

screen once in 20s,  
30s, by 50s at least  
1x/year

Optic nerve head cupping progression



normal c/d is 0.4 or less

slowly reducing field of vision,  
mostly from outside in but can  
get damage in the fixation point,  
by the time pts notice may have  
lost significant vision

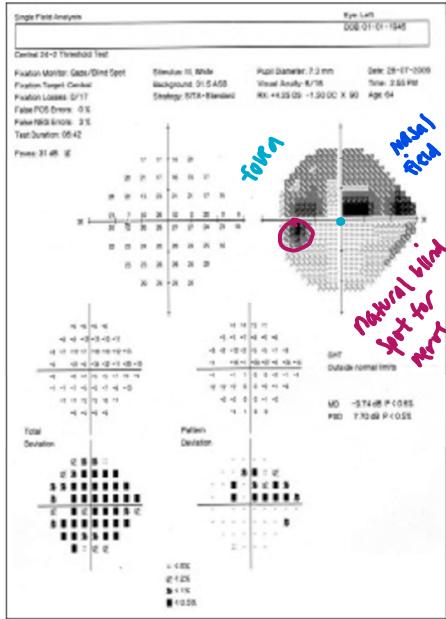
Mechanisms of changes to optic nerve being studied, likely disruption to axonal vascular supply, direct axonal damage, possibly non axonal damage such as to supporting cells (astrocytes). Ganglion cell death at the retinal level may also be caused by elevated IOP in susceptible individuals.

probably won't get tested on this unless go into neuro/ophtho

as if looking through the patient's eyes

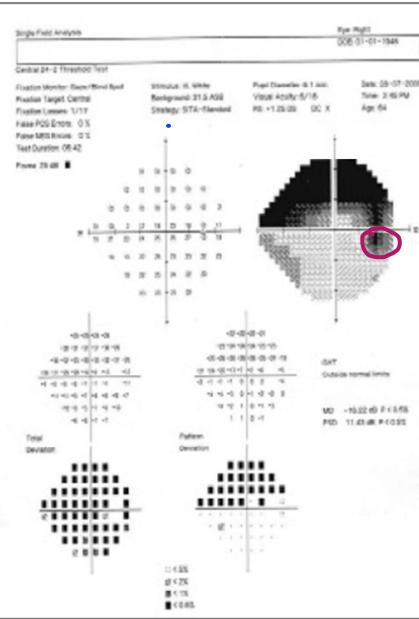
# Humphrey Visual Field

Left Eye from Patient's view



issues in upper fields

Right Eye from Patient's view



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split fixation, blind spot in center of fovea (central scotoma)

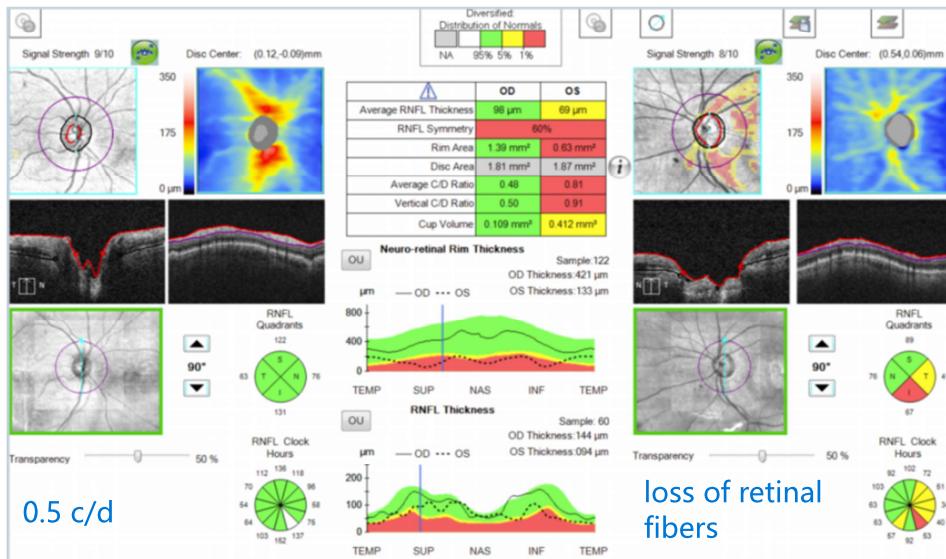
need to test only one eye bc other eye can fill in

Will describe during lecture how HVF is performed. Very subjective test, not objective.  
Now commonly combined with OCT (Ocular coherence tomography) which is more objective and can help track progressive loss of retinal nerve fibers

# Ocular Coherence Tomography

macula on this side and vascular arcades are curved toward

Right Eye as you are looking at the patient



0.9 c/d

Left Eye as you are looking at the patient

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Example of OCT. will not be tested on this but should be familiar with what it is used for. In the above example, the right nerve is essentially normal ; the left nerve demonstrates axon loss globally but especially in the superior-temporal and inferior temporal quadrants

objective test, given c/d ratio good to monitor patients with

# Major Types of Glaucoma

asian if not myopic - risk for low tension glaucoma

## ★ • Primary Open Angle:

- 90% of glaucoma in USA

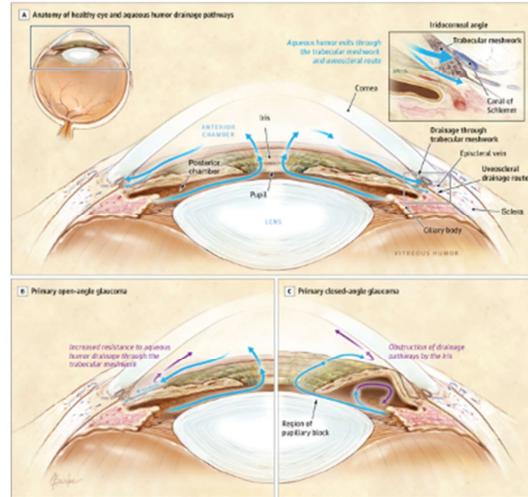
Risk factors almost any race except caucasian

- Race (AA, Hispanic, Asian if myopic)
- Family history
- Thin cornea
- Diabetes mellitus

## ★ • Acute (painful) and chronic (painless) angle closure

- Pigment dispersion
- Pseudoexfoliation
- Angle recession
- Neovascular

don't need to know as much about these



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Acute angle closure to be covered in more detail later in lecture. Pigment dispersion caused by zonules rubbing against posterior surface of iris causing liberation of pigment which then clogs trabecular meshwork. Pseudoexfoliation is thought to be a defect in elastin metabolism resulting in deposit of fluffy proteins seen on anterior lens capsule in dilated eye exam and will clog trabecular meshwork. 50% of people with PXE eventually develop glaucoma. Angle recession primarily found in trauma particularly if associated with hyphema. Thought to be due to long term scarring and fibrosis of trabecular meshwork/schlemm's canal. Neovascularization will "zip up" trabecular meshwork.

ciliary body creates the aqueous and drains through pupil and moves forward open angle everything is open no pupil block so fluid is not getting trapped behind pushing iris forward and mechanical blockage of trabecular meshwork like a clogged sink either remove the block and open the drain or turn off the faucet a lot of drugs do

# Glaucoma: Treatment

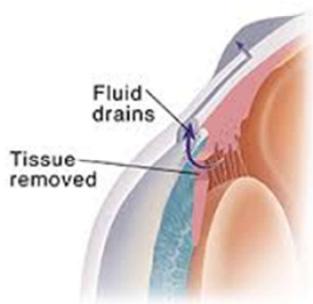
- Pharmacotherapy
  - Decrease aqueous production: topical beta-blockers, oral and topical carbonic anhydrase inhibitor, topical alpha-2 agonists
  - Increase aqueous outflow: miotic agents, topical prostaglandin analogs, rho kinase inhibitors
- Laser
  - Depending on type, can decrease production or increase outflow
- Surgery to create alternative drainage pathways

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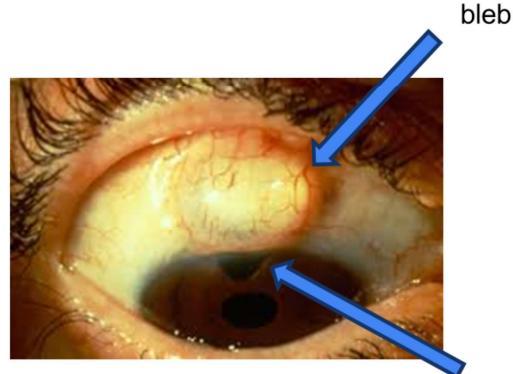
Medications of glaucoma will be covered in a pharmacology lecture. Trabecular laser to increase outflow is thought to cause scarring/shrinkage of the trabecular meshwork at the site of the laser burn thus pulling adjacent trabecular meshwork more open.

# Glaucoma Surgery

Trabeculectomy - new channel is created so fluid can drain out to the subconjunctival space



...successful if see bleb

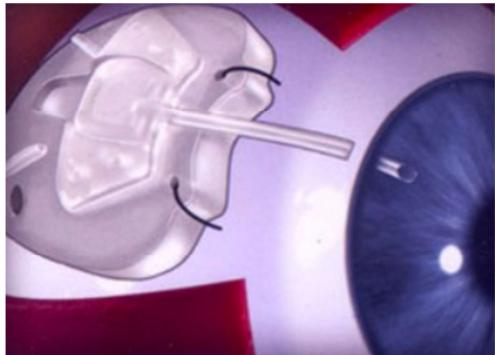


glaucoma surgery by someone trained 30 years ago bc is  
not the best practice now

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Trabeculectomy is rarely done anymore due to long term risks of endophthalmitis. But if you see a bleb and a surgical peripheral iridectomy (the triangular iris defect just inferior to the bleb shown in the photo), this patient has glaucoma!

# Glaucoma Drainage Implants

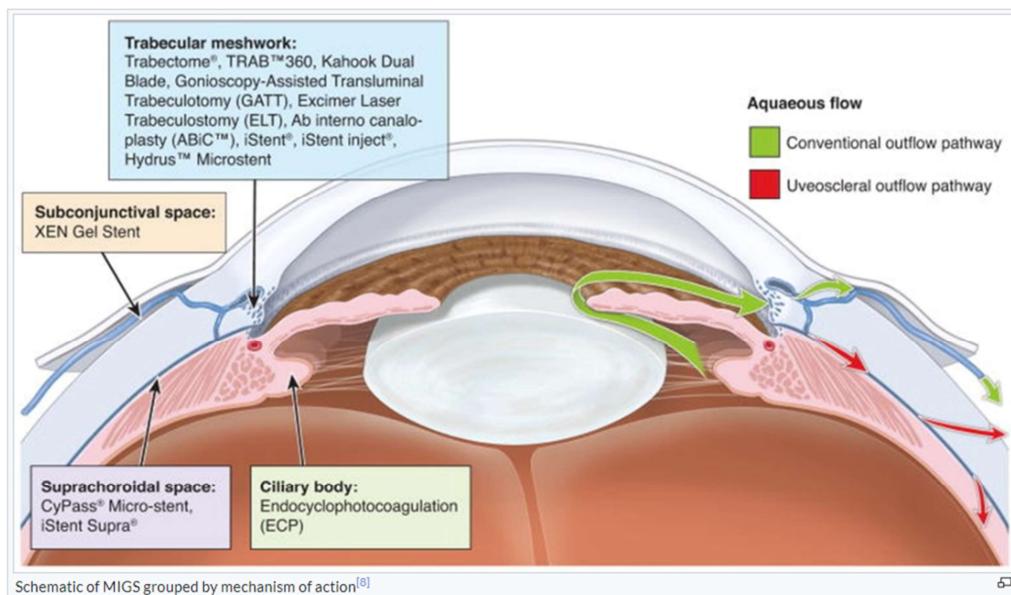


can see with naked eye, do not need slit lamp  
connected to a subconjunctival reservoir  
definitely have bad glaucoma bc is a major surgery

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Severe cases may get drainage implant, especially if other surgeries fail. If you see a tube in the anterior chamber, this patient has glaucoma!

## MIGS Approaches



sometimes getting this in addition to a cataract surgery

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MIGS = minimally invasive glaucoma surgery. More than you need to know, but be aware that many modern glaucoma surgeries are not visible to most observers. For instance, the iStent device is 380x80 microns in size

# Macular Degeneration

- Age related macular degeneration is the most common cause of legal blindness in the Western world
- Typically affects age > 55 years
- Risk factors: age, smoking, female sex, lighter pigmentation
- Exam findings: retinal drusen, retinal/photoreceptor atrophy, bleeding, retinal swelling/fluid
- 2 major types [age-related eye disease study 2 - high levels of antioxidant](#)
  - Nonexudative (dry): if severe, use AREDS2 vitamins to prevent progression [gradual loss](#)
  - Exudative (wet) due to choroidal neovascular membrane: treated with intraocular injections as well as AREDS2 vitamins  
[need to get to a retinal specialist immediately](#)

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Per the WHO website, leading cause globally for vision loss is refractive error, then cataract, then diabetic retinopathy followed by glaucoma and AMD. Lancet has a study showing 35% vision loss cataract as much as 50% refractive error, AMD only <12%. Due to availability of cataract surgery in high income countries, Mac degeneration (19 million in USA, 1.5 million vision threatening) and glaucoma (3 million in USA, about 120k blind from glaucoma) are more common cause of vision loss in >60 years but Diabetic retinopathy most common in 25-75 range. In low income countries refractive error and cataract most common. WHO report 2023.

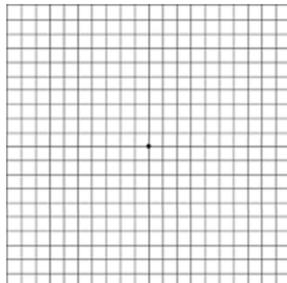
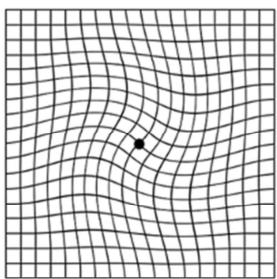
# Macular Degeneration



devastating bc can't drive, read, recognize faces

# Macular Degeneration - home testing

- Amsler grid
  - Tests central 10 degrees of vision
  - Patient instructed to note missing or distorted areas in the grid



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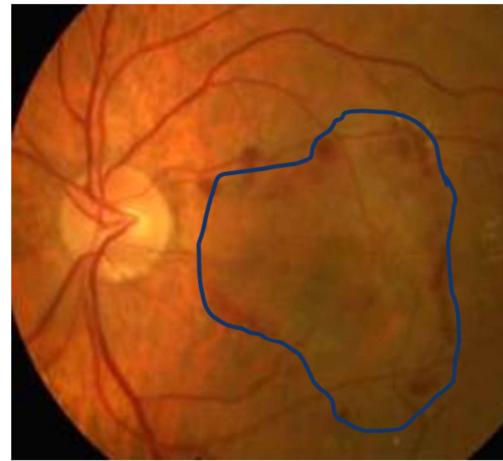
too subjective

Has fallen out of favor with ophthalmologists due to the grids being large. Patients may complain that there is distortion when reading or that lines look wavy on a page, usually not symmetrical so check each eye

screened more often the older pts get bc more likely to get things

# Macular Degeneration

Dry AMD 15% go on to  Wet/neovascular AMD



Wet AMD causes 90% of AMD advanced vision loss!

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Dry macular degeneration, also known as dry AMD, is the most common condition associated with age-related macular degeneration. Dry AMD is caused when yellow or white deposits called drusen accumulate under the retina.

When drusen begins to grow, or multiply, they cause a distortion in vision. Drusen may increase over time and cause damage, potentially destroying the cells in the macula. Patients with dry AMD may have blind spots in the center of their visual field and/or color perception loss as the central vision of the eye is reduced in advanced cases.

Wet (neovascular) macular degeneration is the most advanced form of AMD. It is accountable for roughly 90% of advanced vision loss associated with macular degeneration. Wet AMD occurs when the eye begins to grow new abnormal blood vessels from the choroid into the retina. This usually results in fluid and blood accumulating in the retina, affecting vision. Approximately 15% of patients with dry AMD go on to develop wet AMD.

# Macular Degeneration

if blind in one eye, start on AREDS2 vitamins

- Prevention

- Dark green leafy vegetables - [antioxidants](#)
- Do not smoke
- AREDS2 formulation of eye vitamin for the appropriate risk groups
  - Did not show benefit to people with very early AMD or no AMD
  - Difficult to get appropriate amounts from diet alone
  - Most people in AREDS2 trial also took a multivitamin
- Sunglasses - [reduce exposure to UV light](#)

- Treatment

- [Refer to Ophthalmology](#) for closer monitoring of vision and retinal structures (retinal OCT) and/or consideration of anti-VEGF injection

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AREDS2 is the name of the study that recommended the specific formula of vitamin (OTC), found to be helpful for those who have intermediate AMD but not early AMD or no AMD/only family history. High doses antioxidants (vit c 500 mg, vit E 400IU, zinc 80 mg, copper 2 mg (to prevent zinc related copper deficiency), lutein 10mg, zeaxanthin 2mg.) AREDS 1 formula had high dose of beta carotene but no longer used due to risk of increased lung cancer rate in smokers and former smokers.

Omega 3 fatty acids were not found to be of added benefit in the AREDS2 study but carotenoids lutein and zeaxanthin did incrementally slow progression and preserve vision over AREDS formula (minus beta carotene) alone

Anti-VEGF = family of medications that stop growth of new blood vessels.

Also brand new FDA approved injections to treat geographic atrophy (advanced dry AMD) but not widely used yet

# Diabetic Retinopathy

can go in and out depending on sugar control, if they've been treated

- Affects 40-45% of individuals with diabetes at some point in their life\*
- Due to microangiopathy, indicative of systemic vascular damage
- "Background"= nonproliferative: intraretinal hemorrhages, hard exudates, venous beading, microaneurysms will not see thing unless dilating but can get tested on so recognize in photos
- Proliferative: neovascularization leading to vitreous hemorrhage, worsening macular edema, tractional retinal detachment
- Macular edema: loss of vision from central retinal swelling
- Treatment
  - Control of diabetes, hypertension
  - Referral to Ophthalmology for regular exams and consideration of laser; anti-VEGF injection.

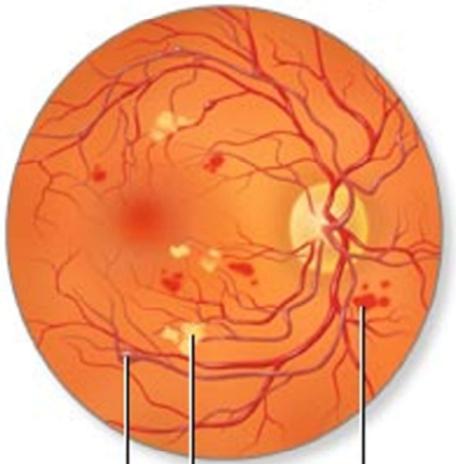
unless got to mod-severe non-proliferative do not need to tx from ophthalmology (need to tx macular edema usir intraocular injections) its more sugar control  
but at a certain point laser will improve vision regardless of sugar control

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\*In United States in 2021 CDC published : 9.6million with DR (26.43% of all diabetics, of these 1.84million (5% of diabetics) had visually threatening DR). Meta analyses done in 2023 estimated over 105million people worldwide DR, estimate 77% of type 1 in their lifetime and 25% of type 2.

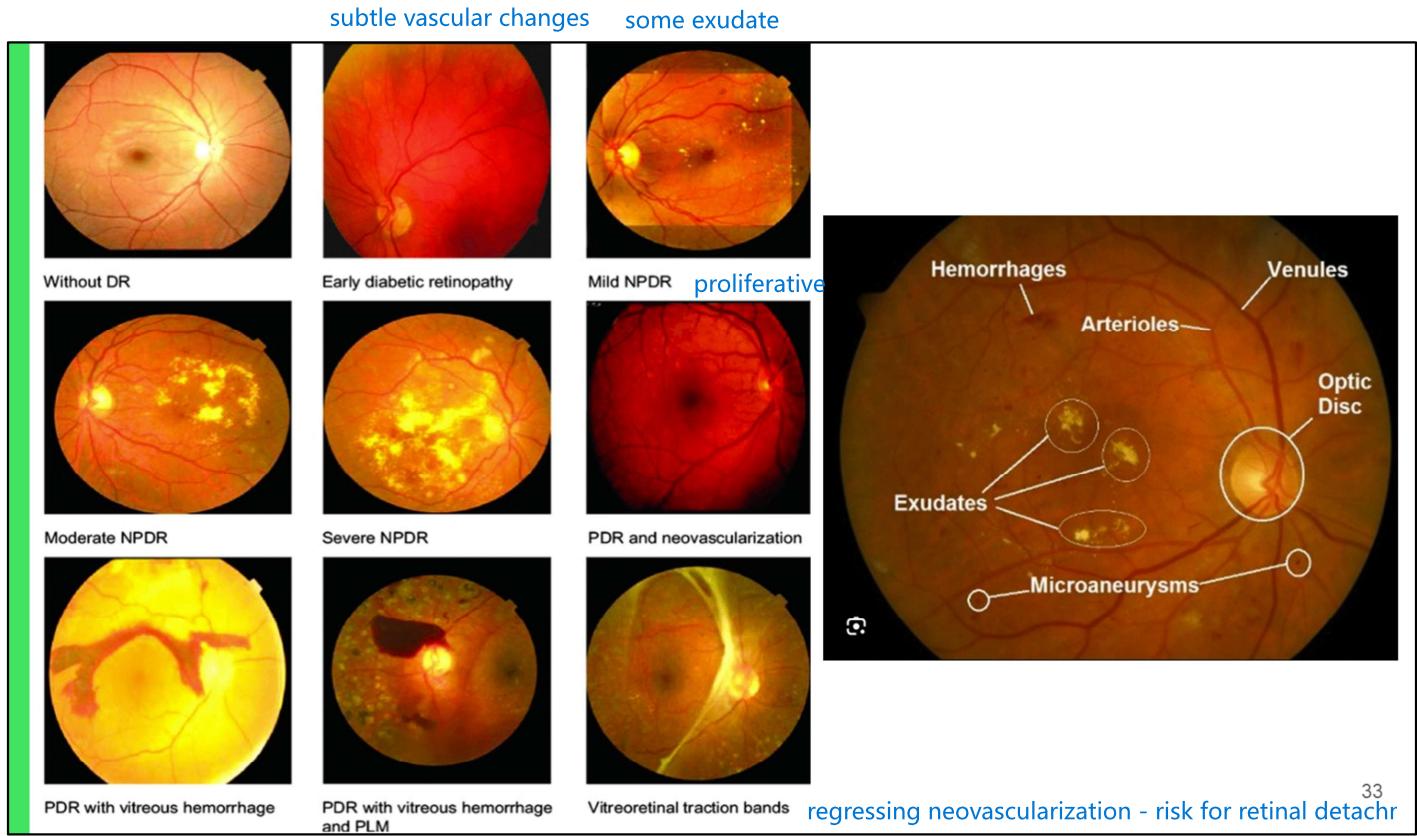
Per the WHO website, leading cause globally for vision loss is refractive error, then cataract, then diabetic retinopathy followed by glaucoma and AMD. Lancet has a study showing 35% vision loss cataract as much as 50% refractive error, AMD only <12%. Due to availability of cataract surgery in high income countries, Mac degeneration (19 million in USA, 1.5 million vision threatening) and glaucoma (3 million in USA) are more common cause of vision loss in >60 years but Diabetic retinopathy most common in 25-75 range. In low income countries refractive error and cataract most common. WHO report 2023.

Non-proliferative  
diabetic retinopathy



Proliferative  
diabetic retinopathy





old laser scars

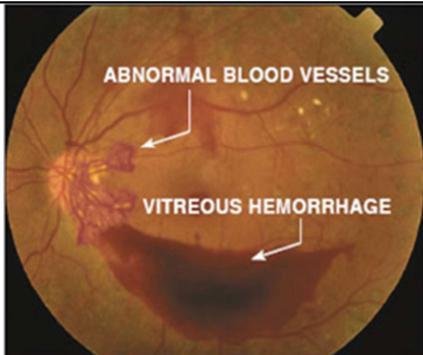
exudate - like bathtub ring

Images from the following sources:

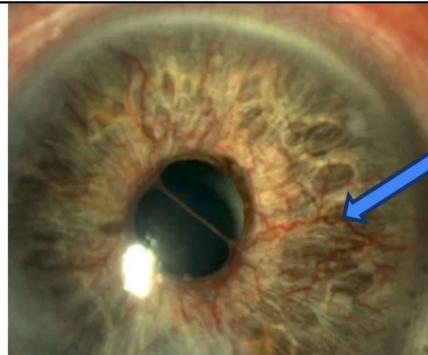
Kandhasamy, J pradeep & Balamurali, Saminathan & Kadry, Seifedine & Ramasamy, Lakshmana. (2020). Diagnosis of diabetic retinopathy using multi level set segmentation algorithm with feature extraction using SVM with selective features. *Multimedia Tools and Applications*. 79. 10.1007/s11042-019-7485-8. (9 images)

Murugan, R., Roy, P. MicroNet: microaneurysm detection in retinal fundus images using convolutional neural network. *Soft Comput* **26**, 1057–1066 (2022).  
<https://doi.org/10.1007/s00500-022-06752-2> (findings labeled within image)

proliferative vessels - tend to grow anteriorly into vitreous

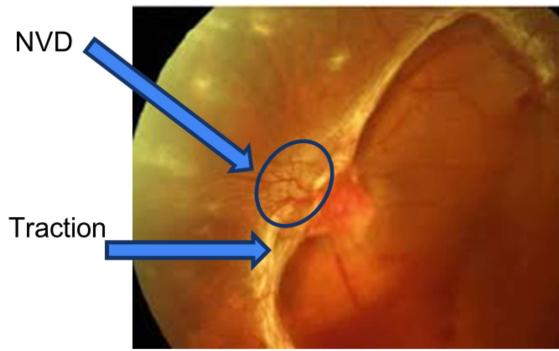


Vitreous hemorrhage

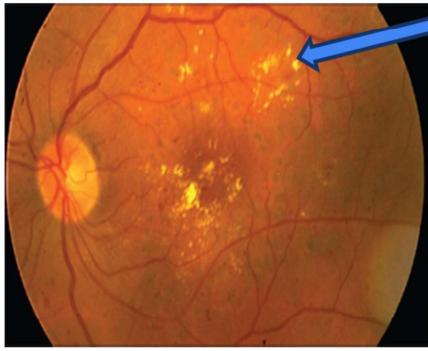


may be able to see in a slit lamp especially in light color eyes

Neovascularization of iris



Retinal detachment



Macular edema

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Note: Neovascularization of the angle leads to glaucoma.

# Vision with Proliferative Diabetic Retinopathy or Diabetic Macular Edema



NORMAL VISION  
*Vision remains intact*



DIABETIC RETINOPATHY  
*Vision is obstructed by macular edema*

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like lava lamps with blobs everywhere unless really base vitreous hemorrhage - big cloud in front of eye

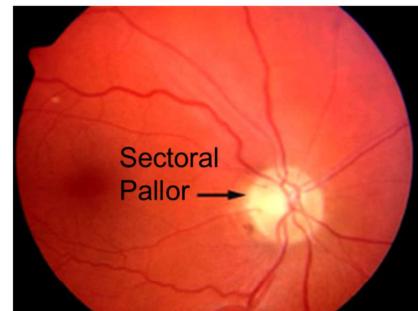
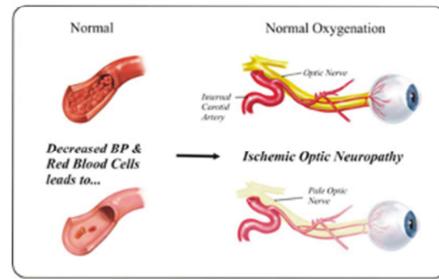
Not the best example of vision through diabetic retinopathy, but the take home concept here is that the vision loss from diabetic retinopathy can be “splotchy”, not just central as in macular degeneration or macular edema by itself without other retinopathy. A common complaint of patients with poorly controlled diabetes (with or without retinopathy) is that their vision seems to change day by day. This is usually due to daily changes in the cornea and lens due to sugar levels in the tissues and can resolve within a day. But diabetic retinopathy vision changes rarely wax and wane so rapidly.

# Optic Neuropathy

- Typically gradual loss of vision, loss of color vision, decreased visual field
- Optic nerve swollen or pale
- Causes
  - Inflammation such as sarcoid, multiple sclerosis
  - Infection such as syphilis, tuberculosis
  - Ischemic
  - Mass effect/compression
  - Toxic: smoking, alcohol, antifreeze, B12/folate deficiency, ethambutol
  - Infiltrative such as lymphoma, leukemia

like static on TV

## Ischemic Optic Neuropathy



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ethambutol causes optic neuropathy (TB treatment) - fav board qu

# Sudden Painful LOV

angle closure - pain comes from high pressure

need to  
know

- ★ Angle closure glaucoma
- Corneal ulcer
- Uveitis
- Optic neuritis
- Post traumatic
- Endophthalmitis

most have an inflammatory component

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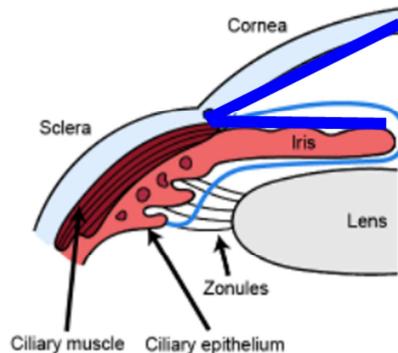
Endophthalmitis was discussed in the red eye lecture, typically there will be a large hypopyon and some form of entry of bacteria into the eye, e.g. a corneal ulcer, prior eye surgery, trauma, etc Rarely endophthalmitis can be endogenous.

Will not discuss trauma in this section, but any history of trauma associated with acute vision loss should have an urgent eye exam done by an ophthalmologist

# Angle Closure Glaucoma



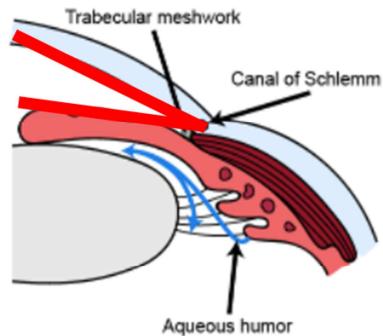
A) Normal



© Lineage

flat iris

B) Angle-closure glaucoma



Moises Dominguez

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lens stick to iris when get pupillary block - mid dilation  
dusk, stress, drugs with anticholinergic or  
sympathomimetic, benadryl, sudafed)

1. Lens “sticks” to the iris.
2. Pressure rises as flow through pupil is reduced or blocked.
3. Iris “bows” forward under the pressure behind it.
4. Angle is “closed” or narrowed.

# Acute Angle Closure Glaucoma

sometimes will do cataract surgery and replace lens with a thinner one

- Risk Factors
  - Age 55-70; female:male - 4:1; history of uveitis or other scarring conditions; bulky cataract formation **lens is getting thicker A-P so have less room between lens and iris**
- Acute closure precipitated by **pupillary dilation**
  - Stress, darkened room, drugs with anticholinergic or sympathomimetic action
- Symptoms
  - Monocular intense pain, loss of vision, photophobia, halos around lights, nausea and vomiting
- Signs
  - Conjunctival injection, lid edema, corneal edema with blurring of red reflex, mid-dilated pupil, IOP elevated

very rare to have bilateral acute angle closure glaucoma bc eyes are not usually the same



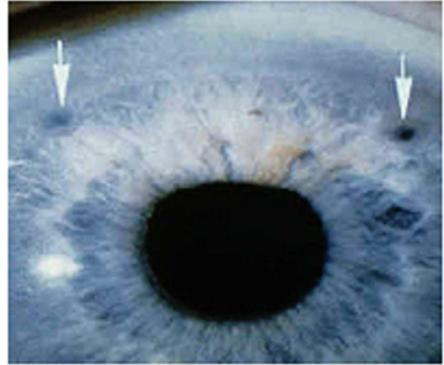
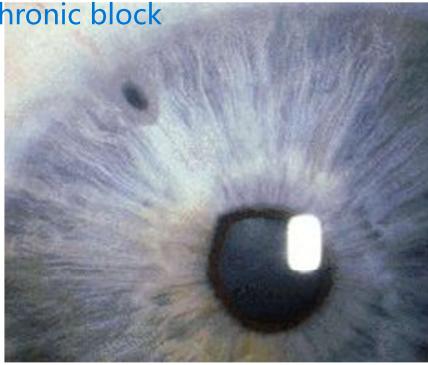
bc pupil block keeps it stuck

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.Angle closure can be caused by many things: meds (more than 60 have been identified), cataract growth (slow but causes pupillary block worsening over time) predisposition includes age, female, hyperopia (small, crowded eye) , family history.

# Treatment

- Emergent referral\*\* if not sulfa allergic
- All drops and oral acetazolamide (Diamox) on board
- Laser iridotomy to establish alternative flow if etiology is pupillary block  
way for fluid to get through without going through pupillary block but doesn't work as well with a chronic block



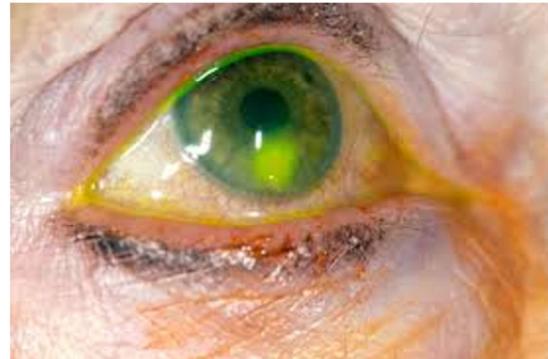
40

Diamox/acetazolamide is a carbonic anhydrase inhibitor. In an ER sometimes IV mannitol is used especially if the patient is allergic to sulfonimides (very low risk of cross allergy if sulfonimide allergy is IgE mediated)

# Corneal Ulcer

breached epithelium - moist environment

- Bacterial, fungal, viral, acanthamoeba
- Contact lens wear and trauma are major risk factors
- Same day Ophthalmology referral (urgent)\*\* for measurement and antibiotics, possible corneal scraping  
almost never culture bc is so small, but can scrape to debulk



after fluorescein - staining where epithelium is missing

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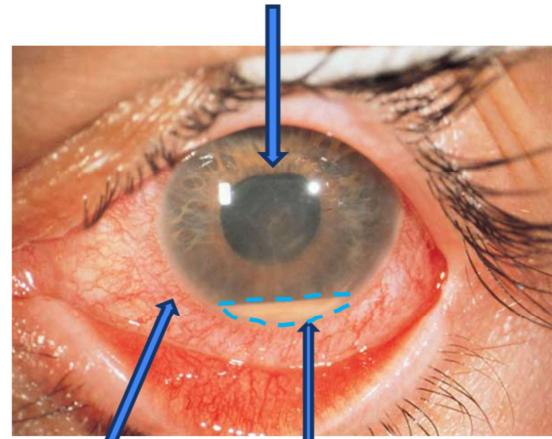
Realistically, very few community eye docs will do cultures, but precise measurements important for following progression and sometimes will do corneal scraping (to reduce infectious load)

# Uveitis

- Typically unilateral
- Blurry vision
- Photophobia
- Aching pain
- Injected “ciliary flush”
- May have pupillary abnormalities
- Can be traumatic

indicates stickiness - iris stick to lens behind it  
bc of inflammation

Pupil not round: superior edge flattened



Ciliary flush

Layered hypopyon

white cells in anterior chamber

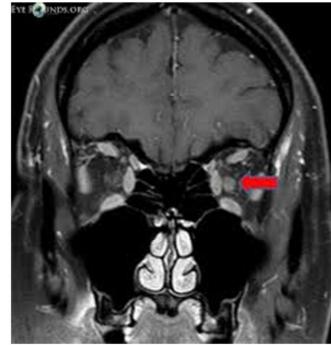
42

This photo shows a hypopyon (layered white cells in the anterior chamber) indicative of severe inflammation. Before layering out the white cells in the anterior chamber may not be visible without a slit lamp.

board question - optic neuritis and MS

# Optic Neuritis

- Typically **unilateral in MS** (classic)
- Blurry vision
- Pain with eye movement will turn whole body vs moving eyes
- Poor color vision
- Rule out infectious causes (syphilis, tuberculosis, Lyme disease)
- **Multiple sclerosis, NMOSD, MOG, CRION**
- If first episode: Ophthalmology evaluation, coordination with Neurology for IV steroid treatment.  
short term high dose steroids short term and better with IV than oral steroids



T1 coronal (top image) and axial (bottom image) post contrast left optic nerve enlarged and enhanced

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will recover whether you treat it or not if high risk for infections don't want them to be on steroids no diff long term for high dose steroid vs nothing will likely still recover vision

Neuromyelitis Optica Spectrum disease, (aquaporin 4 antibodies), Myelin Oligodendrocyte Glycoprotein antibody disease. Both originally thought to be forms of multiple sclerosis, but the discovery of specific antibodies and differences in natural course and treatments for the three diseases helped to define these as their own entities. Important because once you treat with high dose steroid then the AQPR4 and MOG antibodies can't be found for up to several months due to the immunosuppression. Chronic Relapsing Inflammatory Optic Neuritis almost always unilateral and without systemic findings, treatment may be IGG, immunosuppressives, and/or plasmapheresis. The Optic Neuritis Treatment trial showed that high dose steroid sped up visual recovery but did not change final vision compared to no treatment. Oral steroid at lower dose was actually WORSE than no treatment with regards to recurrence and progression of demyelinating plaques on serial head imaging. The top coronal image shows a T1 post contrast inflamed nerve that is enlarged and enhanced (red arrow). The bottom image shows T1 axial view of same nerve.

NAION is in the differential

## Differences between MS and NMOSD eye presentation

- MS typically presents with attack in one eye at a time
- Attacks are less severe and may have complete or near-complete recovery
- Demyelinating lesions most often found in the brain, the optic nerve, and sometimes in the spinal cord
- May have cognitive effects over time such as memory issues
- Negative for AQP4
- Long term treatment is usually disease modification
- NMOSD can attack both eyes at once
- Attacks are severe and a single attack can result in significant visual disability
- Demyelinating lesions most often found at optic nerve and spinal cord, sometimes in the brain
- Occurs in older population than MS
- May present with hiccups, nausea, vomiting
- Positive for AQP4 antibody
- Long term treatment is immunosuppression

\*Another demyelinating disease is MOGAD, which presents as diplopia/confusion/lack of coordination, N/V in children and presents with optic neuritis and transverse myelitis in adults. Less severe than NMOSD and long term requires immunosuppression.

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can't find antibodies if suppressed them all

- Neuromyelitis optica spectrum disease. Aquaporin4 antibody. Myelin oligodendrocyte glycoprotein antibody disease. Acute treatments for all of these can be IV steroid, PLEX, but long term medications for MS are immune modifiers (some oral, some IV) and for MOGAD/NMOSD different meds for suppression including IVIg. Much more detail than you will be tested on, just highlighting how the diseases can present differently.

# Sudden Painless Vision Loss

Vitreous hemorrhage

Retinal detachment

Vascular occlusion

★ Ischemic optic neuropathy  
(giant cell arteritis, GCA)

Stroke

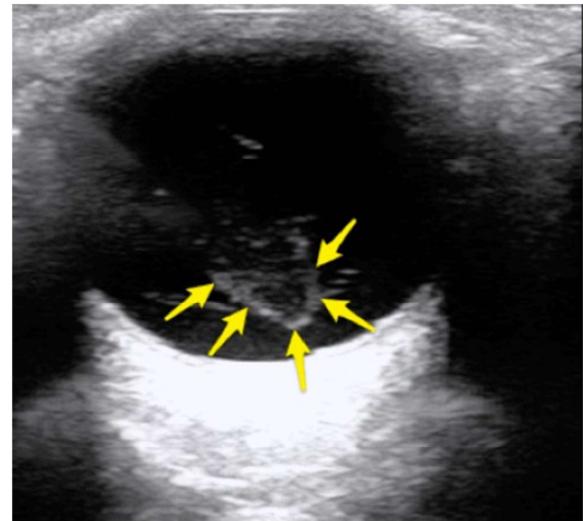
# Vitreous Hemorrhage

- Sudden monocular loss of vision with black spots, cobwebs, haze
- Etiologies **flashing lights and floaters**
  - Hemorrhagic posterior vitreous detachment (watch out for retinal tear!)
  - Proliferative diabetic retinopathy
  - Exudative AMD
  - Retinal vein occlusion
  - Sickle cell anemia
  - Trauma
  - Hypertensive emergency
  - Intraocular tumor

most need an eye exam, remember to check BP

# Vitreous Hemorrhage

orbital ultrasound - washing machine sign bc not attached to anything



blood is blocking light from getting to retina

# Retinal Detachment

- 3 main types
  - Rhegmatogenous
    - Starts with peripheral tear, liquid vitreous then enters subretinal space and dissects it away  
*curtain coming across eye and stays, if goes away = amaurosis f.*
  - Non-rhegmatogenous/serous
    - No tear, Starling forces lead to exudate accumulation in subretinal space  
*floaters, VF loss, etc.*
  - Tractional
    - Retina is pulled off by contracting scar tissue or regressed neovascularization (for example, in diabetic retinopathy)

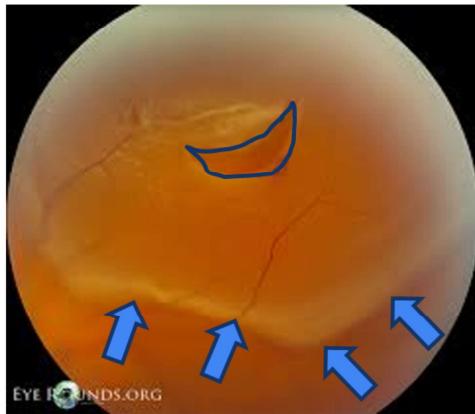
*once macula comes off ability to get baseline vision is harder if can catch before then recovery is much*

# Retinal Detachment

- Symptoms
  - Sudden numerous floaters
  - Flashes of light
  - Metamorphopsia (distorted vision)
  - Visual field loss
- Management
  - IMMEDIATE or URGENT referral for consideration of laser or surgery\*\*
    - Immediate if macular sparing
    - Urgent if macula is involved

# Retinal Detachment

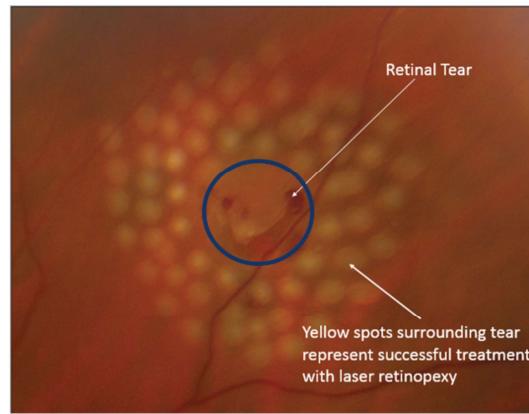
Example of detachment  
(rhegmatogenous)



Horseshoe retinal tear with edge of subretinal fluid indicated by arrows

not a lot of fluid can do laser where fluid is laser won't hold retina

Example of laser to prevent progression

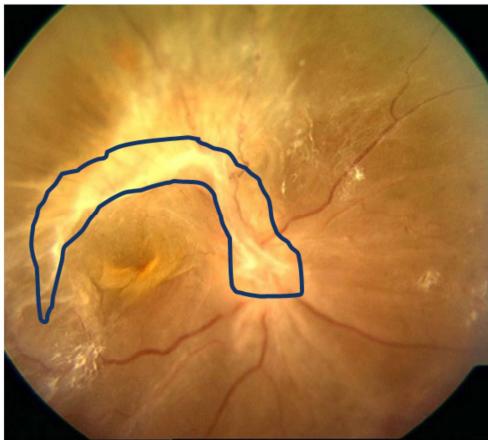


50

superior tear - curtain coming from below  
can improve through the day bc gravity helps the fluid stay down

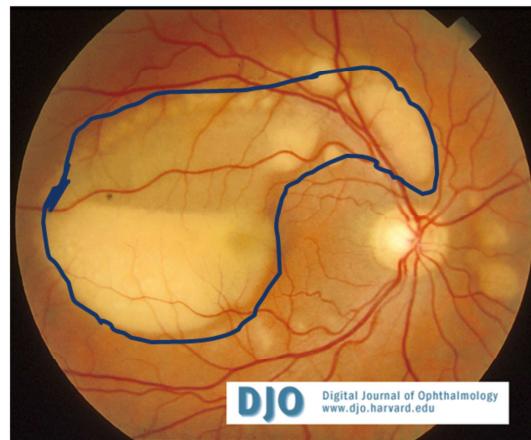
# Retinal Detachment

Tractional



Tractional band pulling retina anteriorly  
**fluid protein level**

Serous/exudative



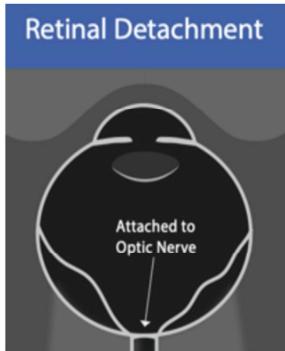
Collection of subretinal fluid lifting retina from behind

DJO Digital Journal of Ophthalmology  
www.djo.harvard.edu

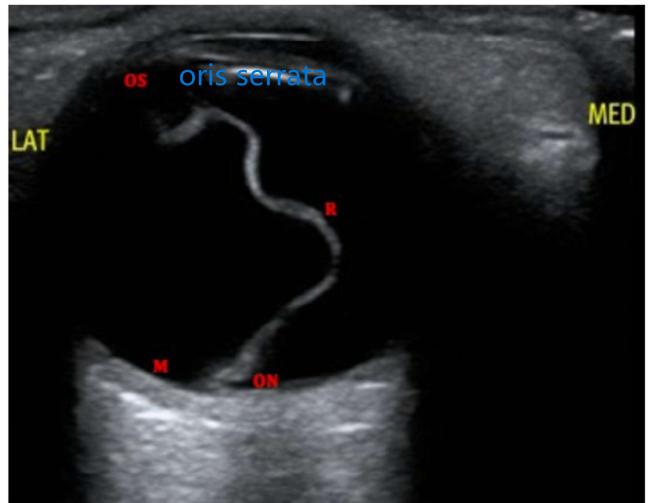
51

Serous detachments caused by inflammatory (ex: posterior choroiditis, posterior uveitis, sympathetic ophthalmia, relapsing polychondritis), infectious (TB, toxoplasmosis, syphilis, fungal infection, toxocariasis), or cancer (various choroidal or retinal malignancies, vitreoretinal lymphoma, hematologic malignancies.)

# Ocular Ultrasound



macular off retinal detachment



will see characteristic V for funnel detachment  
if white line floating above it is vitreous detachment

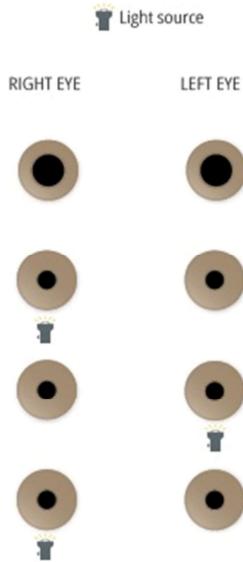
# Retinal Artery Occlusion

- Most often from distant carotid or cardiac embolus
- **Central retinal artery occlusion (CRAO)**
  - Sudden, painless, unilateral, near complete loss of vision maybe light perception
- **Branch retinal artery occlusion (BRAO)**
  - Sudden, painless, hemifield loss
    - e.g., a superior BRAO causes an inferior visual field defect
- **Signs**
  - Relative afferent pupillary defect (APD) (abnormal swinging flashlight test)
  - Might see embolus or **cherry red spot** if doing dilated exam

tend to show  
optic nerve  
problems

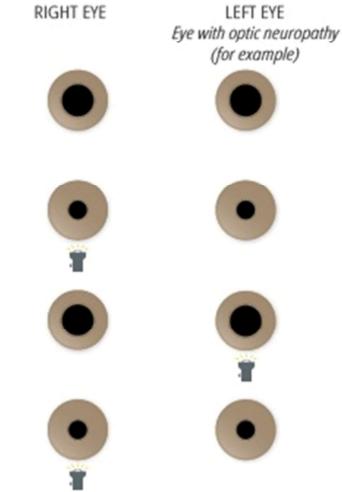
**Figure 2. Swinging-light test – normal (no RAPD)**

Illumination of either eye induces normal and equal pupil responses in both eyes (consensual responses).



**Figure 3. Swinging-light test – left RAPD**

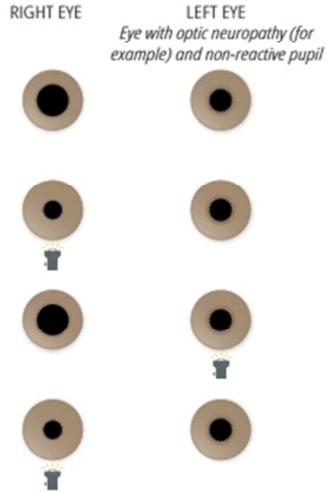
Illumination of the (more) normal right eye causes both pupils to constrict. When the light is moved to the (more) abnormal left eye (e.g. with optic neuropathy), both pupils dilate (constrict less), the left pupil dilating despite the light being shone directly at it. Returning the light to the (relatively) normal right eye results in constriction of both pupils again.



direct response weaker than consensual response

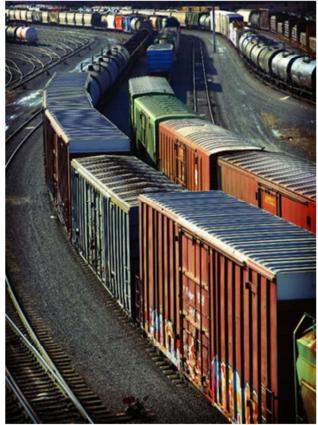
**Figure 4. Swinging-light test: left RAPD + non-reactive left pupil**

Illumination of the relatively normal right eye causes only right pupil constriction. When the light is moved to the abnormal left eye (e.g. fixed pupil and optic neuropathy), the right pupil dilates (constricts less). Returning the light to the right eye results in constriction of the right pupil again. In this situation it is only necessary to observe the eye with the reactive pupil in order to identify an RAPD.



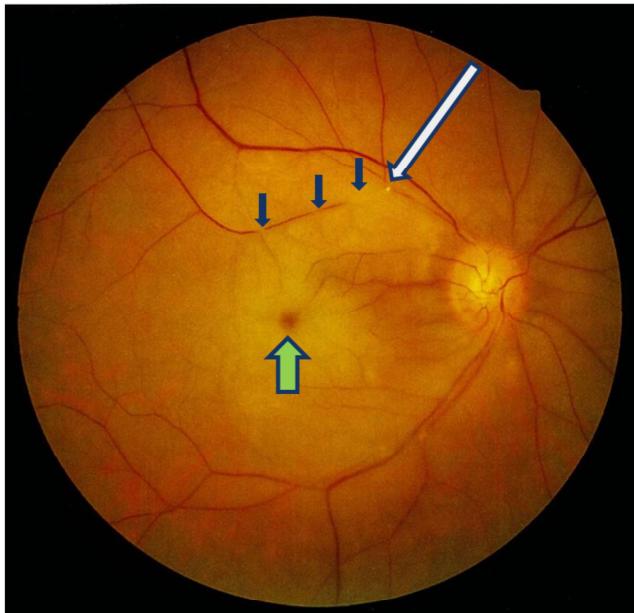
**Relative afferent pupillary defect:** The presence of a unilateral afferent pupillary defect. If a flashlight is swung back and forth between the two eyes (swinging flashlight test), the eye with the afferent pupillary defect appears to dilate in response to illumination. This happens because the pupil constricts in response to illumination of the contralateral eye (an intact indirect pupillary reflex) but fails to constrict with illumination of the affected eye (impaired direct pupillary reflex). This phenomenon does not occur with bilateral afferent pupillary defects.

# CRAO



box-carring

Boxcarring (blue arrows), Hollenhorst plaque (white arrow)



is a branch and not complete CRAO but good pic of cherry red spot

Cherry Red Spot (green arrow)

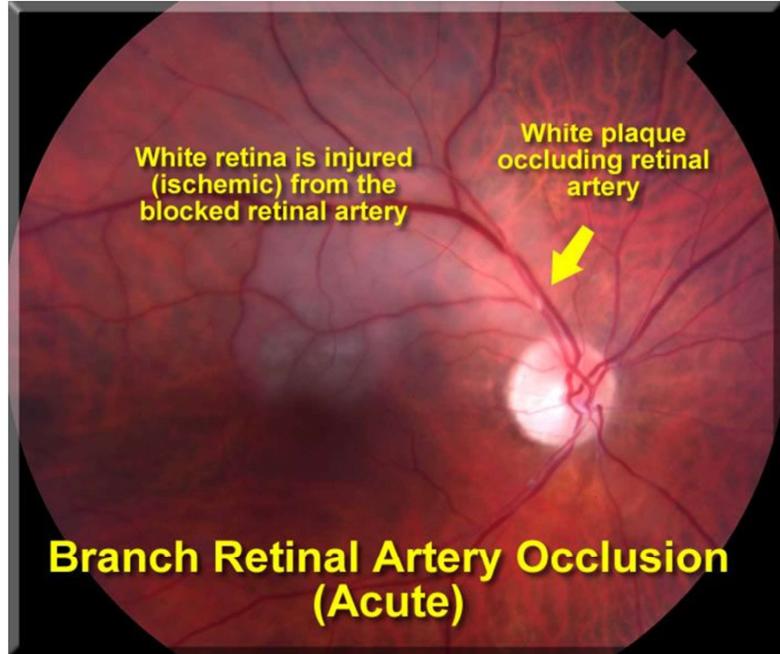
white swelling and thin spot in fovea and can see choroidal circulation = cherry red spot

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Boxcarring is segmental appearance of the arteriole due to sluggish blood flow. Not the best picture of a CRAO since there is some flow in the arterioles, but it does show a cherry red spot, a hollenhorst plaque, and box-carring (superior to fovea)

# BRAO

no cheery red spot  
bc didn't get all the  
way down to retina



# Management of CRAO/BRAO

- Emergent Ophthalmology consultation\*\*
- Stroke equivalent
- If confirmed, requires systemic evaluation
  - Auscultation of the heart and carotid arteries
  - Neurologic assessment
  - Anyone over 65 - labs for ESR, CRP, platelets to look for giant cell arteritis
  - Schedule carotid ultrasound and echocardiogram
- Long term Ophthalmology follow-up for complications

if a month ago not as emergent but still need to do in a timely fashion

# Retinal Vein Occlusion

- Risk factors similar to those of DVT
  - Older age, HTN, DM, CAD, hypercoagulability
- **Central retinal vein occlusion (CRVO)**
  - “Blood and thunder appearance”
    - Dilated, tortuous venules and diffuse intraretinal hemorrhages
- **Branch retinal vein occlusion (BRVO)**
  - Occlusion occurs at **site of arteriole/venule crossing**, as they share common vascular sheath

# Central Retinal Vein Occlusion

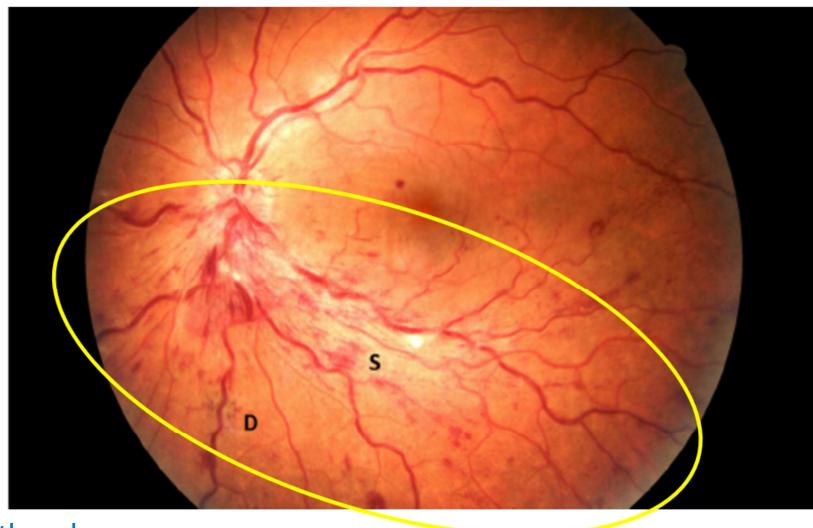
widespread flame hemorrhages



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Presence of many dot-and-blot and/or flame-shaped hemorrhages diffusely and venous thickening (blood and thunder appearance).

# Branch Retinal Vein Occlusion



limited blood and thunder

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“Fundus photograph of an inferior branch retinal vein occlusion in the left eye showing different types of retinal haemorrhages. Superficial retinal haemorrhages (S) are flame shaped; deep retinal haemorrhages (D) are dot-blot.”

Image Credit: [https://www.researchgate.net/figure/Fundus-photograph-of-an-inferior-branch-retinal-vein-occlusion-in-the-left-eye-showing\\_fig2\\_221921783](https://www.researchgate.net/figure/Fundus-photograph-of-an-inferior-branch-retinal-vein-occlusion-in-the-left-eye-showing_fig2_221921783)

# Retinal Vein Occlusion

- Evaluation
  - Ask about thrombotic history (prior PE/DVT), family history of clotting disorders, use of hormones (OCPs = oral contraceptive pills; ERT = estrogen replacement therapy), current pregnancy
  - Check labs to include TSH and coagulation factors
- Treatment
  - **Urgent referral (“See patient A.S.A.P. please”)\*\***, may require laser, injection or IOP lowering, as well as long term monitoring for neovascularization and glaucoma
  - Primary care to address risk factors including HTN, DM, etc.

high risk for neovascularization long term

CRAO true infarct of retina cells are dead so cannot inspire new blood vessels

vein occlusion is ischemic - so ask for help to create new blood vessels

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hypothyroidism → risk factor for atherosclerosis → localized atherosclerosis → retinal vein occlusion

# Giant Cell Arteritis (Temporal Arteritis, Horton disease)

- Is a cause of sudden, typically monocular, **painless** loss of vision
  - Most commonly seen in adults of advanced age (rarely seen in patients < 50 years old)
- Ask about - constitutional symptoms, new-onset **headache** typically located **over the temples**, jaw claudication, proximal muscle/joint aches (association with polymyalgia rheumatica)
- **EMERGENCY! Call Ophthalmology\*\***
- Must treat with high dose steroid to prevent loss of vision in other eye.

Arteritic Anterior Ischemic Optic Neuropathy (AAION) - temporal artery biopsy is indicated; treatment: immediate high-dose systemic glucocorticoids.

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if don't catch it early really high risk for going blind in the other eye

These constitutional symptoms usually present before any loss of vision occurs.

Immediate administration of high-dose glucocorticoids is crucial to lowering the risk of permanent vision loss in patients with giant cell arteritis. [uncomplicated: oral; impaired vision = sign of ischemic organ damage: intravenous for the first few days, followed by oral administration]

There is a disorder of the visual pathway called anterior ischemic optic neuropathy (AION), and the arteritic form is due to giant cell arteritis. In this situation, a temporal artery biopsy is indicated, and immediate high-dose systemic glucocorticoids is required. [The presentation of loss of vision in non arteritic optic neuropathy can be painful, whereas the classic presentation of loss of vision in giant cell arteritis is typically painless.]

# Important Pediatric Ocular Disorders

Infancy

- Nasolacrimal duct obstruction
- Congenital cataracts
- Congenital glaucoma
- Retinopathy of prematurity
- Retinoblastoma

Childhood

- Strabismus
- Amblyopia

# Nasolacrimal Duct Obstruction

- Causes eye discharge and conjunctival irritation from failure of the valve of Hasner to open.
- If no sign of infection, **daily massage** alone is usually sufficient to open the valve if child is under 6 months old  
*ointment will not get in*
- Refer if there is infection
- Older infants should be referred to Ophthalmology for consideration of nasolacrimal duct probing

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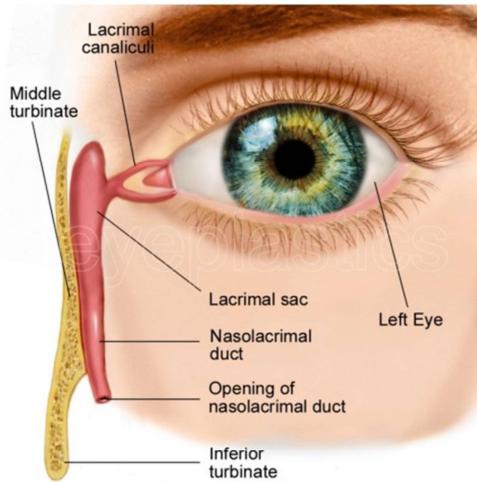
early on is maybe just stenosis

Can also be due to short midface causing kinking of nasolacrimal duct. Probing most successful if done between 12-15 months of age, but most children resolve as mid face grows during first year of life. Other treatments if probing unsuccessful are stenting or DCR

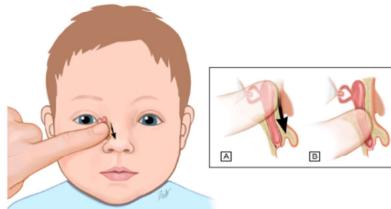
kids don't like bath bc have a rash around eye

most patients resolve on its own

# Lacrimal Sac Massage (Crigler Massage)



- Massage 3-4 times a day over the lacrimal sac with rapid, downward pushing to open the valve of Hasner
- Attempt for 2-3 weeks before considering duct probing [general anesthesia](#)



kinked up like a hose and as child grows and often it will straighten out

# Leukocoria



- Irregular and absent red reflex: ALWAYS REFER!! (semi-emergent in infants)
- Some common etiologies
  - **Cataract**
  - **Retinoblastoma** eye often needs to be enucleated to keep child alive
  - **Retinopathy of prematurity** -infarction of retina
  - **Toxocariasis**
  - **Coats disease**
  - **Persistent fetal vasculature**



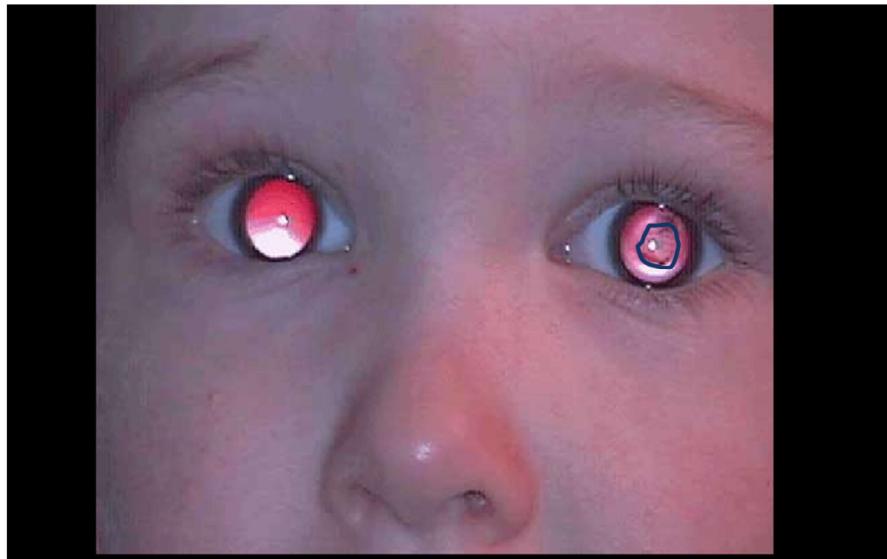
Source: Richard P. Usatine, Mindy Ann Smith, Heidi S. Chumley, Camille Sabella, E.J. Mayeaux, Jr., Elumalai Appachchi; *The Color Atlas of Pediatrics*: www.accesspediatrics.com  
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white red reflex = ophthalmologist!

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boards question - worst possible thing? retinoblast

- **Cataract** = A condition in which the normally clear lens of the eye becomes opaque, which results in decreased or blurry vision.
- **Retinoblastoma** = An intraocular malignancy that typically manifests during childhood with leukocoria (white fundal reflex instead of the usual red) and strabismus. Associated with loss of heterozygosity of the retinoblastoma tumor suppressor gene (RB1).
- Retinopathy of prematurity (ROP) aka retrolental fibroplasia, or Terry syndrome = ROP Disease of the retina with abnormal vessel proliferations that affects preterm, low-weight infants and leads to hemorrhages, fibrosis, and, in severe cases, retinal detachment presenting as leukocoria.
- Toxocariasis = A zoonosis that is caused by the nematodes/helminths *Toxocara canis* and *Toxocara cati*. Typically acquired by ingesting *Toxocara* eggs, which are shed in the feces of infected cats and dogs. Can manifest with acute or chronic endophthalmitis (ocular toxocariasis) and/or visceral larva migrans (visceral toxocariasis).
- Coats disease = A retinal disease characterized by retinal telangiectasias and exudative retinal detachment. Occurs during the first decade of life, predominantly in males, and usually affects only one eye.
- Persistent fetal vasculature, aka persistent hyperplastic primary vitreous = A congenital ocular disease that is due to the failure of both the embryonic primary vitreous and hyaloid vascular system to regress during gestation. Manifests with leukocoria and, if uncorrected, amblyopia.



Congenital cataract causing irregular red reflex

eyes and visual system will not develop with a cataract

# Congenital Glaucoma

- Anterior segment dysgenesis
- **Classic Triad:**
  - Epiphora
  - Photophobia
  - Blepharospasm **constant blinking**
- Buphthalmos from pressure-induced enlargement in eyes with high elasticity
- **Urgent referral\*\*!**



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Epiphora – watery eyes – overflow of tears onto face

Photophobia – “fear” of light

Blepharospasm - The involuntary, forcible closure of the eyelids. The first symptoms may be uncontrollable blinking. Only one eye may be affected initially, but eventually both eyes are usually involved.

Buphthalmos - Buphthalmos is enlargement of the eyeball and is most commonly seen in infants and young children. It is sometimes referred to as buphthalmia. It usually appears in the newborn period or the first 3 months of life. and in most cases indicates the presence of congenital glaucoma, which is a disorder in which elevated pressures within the eye lead to structural eye damage and vision loss. Corneal enlargement can continue until age 3. normal infant corneal diameter 9.5-10.5mm, up to 11.5 mm by age 1. over 12mm before age 1 very abnormal, over 13 mm at any age very abnormal.

[measure thumbnail and compare it to child eyes to get an idea of how large cornea is limbis to limbis](#)

# Strabismus



- Esotropia: **inward deviation**
  - Congenital esotropia is seen prior to age 6 months: likely needs surgery
  - Accommodative esotropia commonly recognized between 2 & 4 years of age; may only require spectacle correction
- Exotropia: **outward deviation**
  - May require surgery if more than intermittent but may be treated with spectacles +/- prisms before considering surgery
- **Must refer for treatment**
- Goal is to use both eyes in order to stimulate binocular visual pathway development and prevent permanent visual loss (amblyopia)

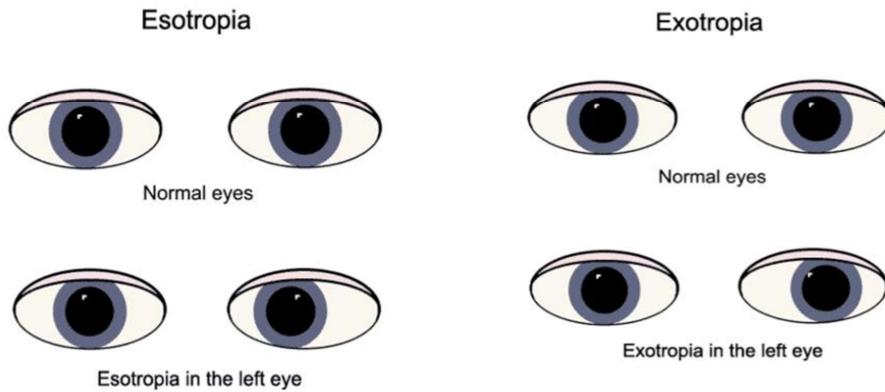
thick glasses to stop accommodating, as eye grows need to accommodate  
take away accommodative need

# Strabismus: Treatment

- Full spectacle correction for esotropia, possible over-minus correction for intermittent exotropia, may use prism glasses for either form of strabismus
- Muscle surgery for uncorrectable deviation
- Patching of good eye to stimulate usage of amblyopic eye
- Amblyopia reversal is MOST successful at younger ages (<8 years old)

# Esotropia and Exotropia

single light source



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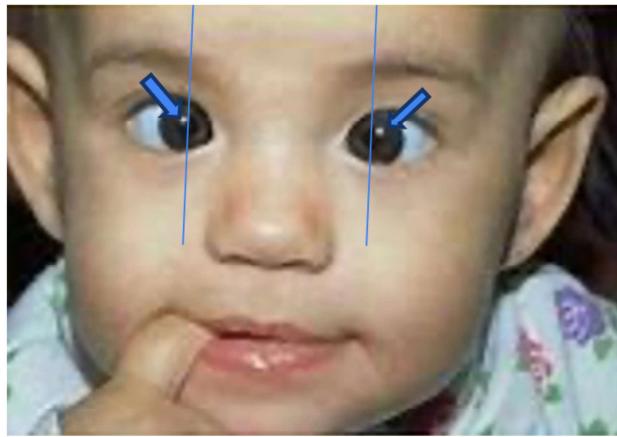
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The corneal light reflection in the “normal” eyes are symmetrical between the right and left eye. Not the best example, since the light is being held slightly off center. Easiest to see if light is central.

In the esotropia example, since the left eye is turned inwards, the light reflection is now temporal in both eyes (mirrored with the light reflex of the right eye rather than symmetrical)

In the exotropia example, the left eye is turned outwards so the corneal light reflection is now further nasal from the visual axis than expected compared to the right eye

# Correction with Glasses



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Look at the corneal light reflection before glasses (bitemporal despite a central light source— shows the eyes are both turning inwards) compared to with glasses (now light reflection is central in both eyes)

# Pseudoesotropia



folds blocking view of normal caruncle

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A type of pseudostrabismus characterized by the appearance of eye crossing (i.e., strabismus) as a result of a wide nasal bridge and/or large epicanthal folds. Most commonly occurs in infants and young children. Note the corneal light reflex is central in both eyes! Compare this to the corneal light reflex in the uncorrected esotropia example on the previous slide.

# Cases

## Case 1

A 72-year-old woman reports that “the vision in my left eye is steadily getting worse.” She has not been able to focus on anything clearly, whether near or far, for at least 1 year. She has worn glasses for “bad vision” ever since age 20. Her last eye exam was 5 years ago because she “got too busy.” She denies eye pain or trauma.

She has a PMH of hypertension on HCTZ and osteoporosis on alendronate (Fosamax). She also takes a “baby aspirin.” [aspirin 81 mg]

Visual acuity: 20/200



white atrophy

# What is the most likely diagnosis?

- A. Atrophic macular degeneration
- B. Cataract *as ddx bc of story but didn't show lens*
- C. Hypertensive retinopathy *no htn problems, no av nicking*
- D. Open-angle glaucoma
- E. Retinitis pigmentosa

## Case 2

A 14-year-old boy presents with his mother for evaluation and management of impaired vision bilaterally. He states that sometimes he will see “halos” or glare around lights. He denies any pain. His mother notes that his vision seems to have slowly worsened over the years. She also reports that he has a PMH of asthma and was originally diagnosed at age 13 months after an episode of bronchiolitis. His current medications include inhaled albuterol 2 puffs q.i.d. p.r.n. as well as 2 puffs b.i.d. of a combined long-acting  $\beta_2$  adrenergic agonist plus corticosteroid. Examination with a slit lamp reveals granular deposits.

# What is the most likely diagnosis?

- A. Acute angle glaucoma no pain, gradual vision loss less likely
- B. Exudative macular degeneration will not see in a 14 y.o.
- C. Functional vision loss need granular deposits so not functional
- D. Posterior subcapsular cataract DM, steroid use**
- E. Strabismus

## Case 3

A 70-year-old man presents to the emergency department for “problems with my vision all of a sudden.” He denies any pain. Examination of his left eye reveals almost complete loss of vision. In performing the swinging flashlight test, the left eye appears to dilate in response to illumination. A cherry red spot at the fovea centralis is appreciated on fundoscopy. No dot-and-blot or flame-shaped hemorrhages are seen.

# What is the most likely diagnosis?

- A. Central retinal artery occlusion    *cherry red spot*
- B. Central retinal vein occlusion
- C. Cytomegalovirus retinitis
- D. Optic neuritis
- E. Retinal detachment

## Case 4

hypercoaguable

A 28-year-old woman has a PMH of Factor V Leiden and a previous episode of DVT. Given this history, she is at increased risk for which of the following?

# Most likely disorder?

- A. Atrophic macular degeneration
- B. Central retinal vein occlusion    hypercoagulable increases risk
- C. Functional vision loss
- D. Hyperopia
- E. Retinitis pigmentosa

# Questions?

