EyeFlyMD

The EYE GUIDE

V.MAY.22

Chapters 1-2

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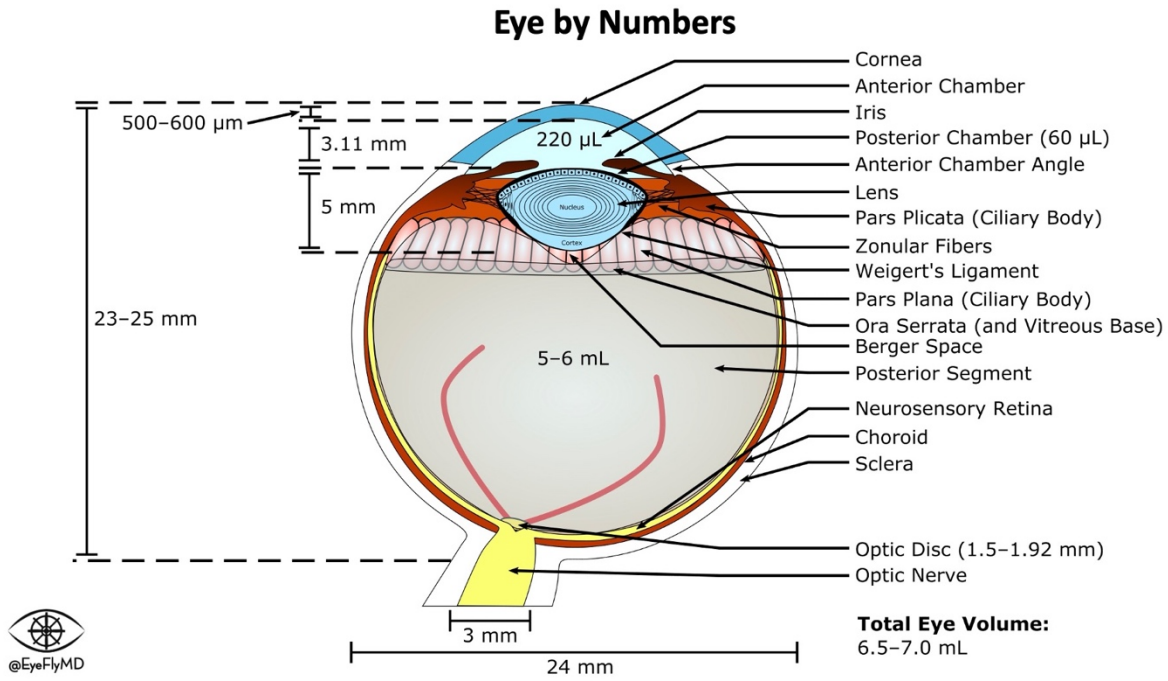
Disclaimer: This writing (and most of the figures) is my own and based on 4 resources: The BCSC, two ophthalmology question banks, and primary literature for some of the refractive sections (because I find it interesting) and areas where I felt the BCSC was contradictory or incomplete. This is not medical advice and is intended for the purposes of ophthalmology education. I created the majority of the figures and included as many original photos as possible. I don't cite anything here because it's too much work but you can always refer to the BCSC. Don't take my word for it.

Anatomy, Terms, and Basic Concepts

Anatomy Overview



- **Conjunctiva** – Clear, thin epithelial membrane that covers the inner eyelids (palpebral conjunctiva) and surface of the eye (bulbar conjunctiva) continuously to the limbus (sclera-cornea interface). Because of the conjunctiva it is impossible for contact lenses to "get lost" behind the globe.
- **Cornea** – The clear portion in the front of the eye. The limbus delineates the cornea from the surrounding sclera. Responsible for 2/3 (~43D) of the eye's refractive power.
- **Iris** – Pigmented structure that controls amount of light entering the eye.
- **Lens** – Transparent, biconvex structure. Responsible for 1/3 (~20D) of refractive power.
- **Zonular fibers** – Connect the lens to ciliary body, support the lens and help it accommodate.
- **Ciliary body** – There are two portions of the ciliary body. The pars plicata is the anterior portion with the ciliary muscle and ciliary processes. It contracts to assist in accommodation and the ciliary processes (outpouchings) produces aqueous humor by active secretion at a rate of 2-3µL/min. There are around 80 ciliary processes. The pars plana portion is the posterior, flat (planar) part that meets the retina. Because there is no retina in this area it is where trocars are placed for a Pars Plana Vitrectomy (PPV).
- **Anterior Chamber** – Aqueous filled space between the corneal endothelium and iris. Volume: ~200 µL.
- **Posterior Chamber** – Aqueous humor-filled space between the iris and the anterior lens. Volume: ~60 µL.
- **Anterior Segment** – The Anterior Chamber + Posterior Chamber.
- **Posterior Segment** – Essentially everything behind the lens including the vitreous and retina.
- **Uvea** – The structure composed of the iris, choroid, and ciliary body.
- **Vitreous** – The vitreous is 99% water. The rest of the composition is collagen and hyaluronic acid which gives it twice the viscosity of water. The vitreous occupies ~ 4 mL of the total 5–6 mL of the posterior segment.
- **Neurosensory Retina** – Light-sensing tissue of the eye. The term "Retina" by itself technically refers to the neurosensory retina *and* the retina pigment epithelium (RPE).
- **Retina Pigment Epithelium (RPE)** – The pigmented structure that lies outer to and apex-to-apex with the neurosensory retina. It has many functions including absorbing light energy, dissipating heat, supporting the neurosensory retina metabolically, and forming the outer blood-retina barrier.

- **Choroid** – Vascularized layer just inner to the sclera and blood supply for the outer retina. The inner retina is supplied by the central retina artery.
- **Optic Disc** – The area where the axons from the retina come together to form the optic nerve and where the fibers subsequently exit the eye. The optic disc is the most anterior portion of the optic nerve and is ~1.5 mm wide and extends ~1 mm into the eye.
- **Sclera** – The collagenous (mostly type I), avascular tissue encapsulating the eye. The three layers from outer to inner are the episclera, stroma, and lamina fusca.
- **OD** – "Oculus Dexter" or "Right Eye"; note this does not apply to lids (i.e., Say "Right Upper Lid" not "Upper Lid OD".
- **OS** – "Oculus Sinister" or "Left Eye"
- **OU** – "Oculus Uterque" or "Both Eyes"

Anatomy of the Cornea

The 5 layers of the cornea are the epithelium, Bowman's layer (not a true membrane mostly type I and V collagen), the corneal stroma (Type I collagen comprises 70% of dry weight), Descemet's Membrane (the basement membrane of the endothelium; type IV collagen), and the endothelium. The corneal stroma maintains a water content of 78%.

Different substances deposit in different layers. For example, iron lines are deposited in the epithelium, calcium (band keratopathy) is deposited in Bowman layer, and copper (Kaiser-Fleischer Ring) is deposited in Descemet Membrane. Additionally, the epithelium is lipophilic and the stroma is hydrophilic so these are important considerations for pharmaceuticals.

The adult cornea measures 11–12 mm horizontally and 10–11 mm vertically. The average central corneal thickness (CCT) is 540 – 550 µm. The peripheral cornea is thicker and flatter with maximal cornea thickness of 1 mm at the limbus. The cornea is thinnest inferotemporal to center. The cornea can become edematous, and this is usually a result of endothelial pump failure. Signs of corneal edema include epithelial haze, stromal thickening, *Waite-Beetham lines* (wrinkles deep in the stroma), and folds in Descemet membrane. Descemet folds appear when corneal thickness increases by $\geq$ 10%. Of the layers of the cornea, vision loss is most affected with changes in the epithelium. Epithelial edema occurs when CCT $\geq$ 700 µm. CCT is also important for other reasons. A thin CCT can cause artificially low IOP readings and is also an independent risk factor for glaucoma (it may reflect the integrity of the eye wall). A CCT $\geq$ 640 µm is a risk factor for symptomatic edema after cataract surgery.

We are born with a central endothelial cell density of ~ 5,000 cells/mm^2. The cell density decreases ~ 0.6% per year with landmarks of 3,400 cells/mm^2 at age 15 and 2,300 cells/mm^2 at 85. At any stage of life 2,000 – 3,000 cells/mm^2 is a reasonable normal. < 500 cells/mm^2 risks corneal edema as the important pumping function of the endothelial layer that maintains the proper corneal hydration diminishes.

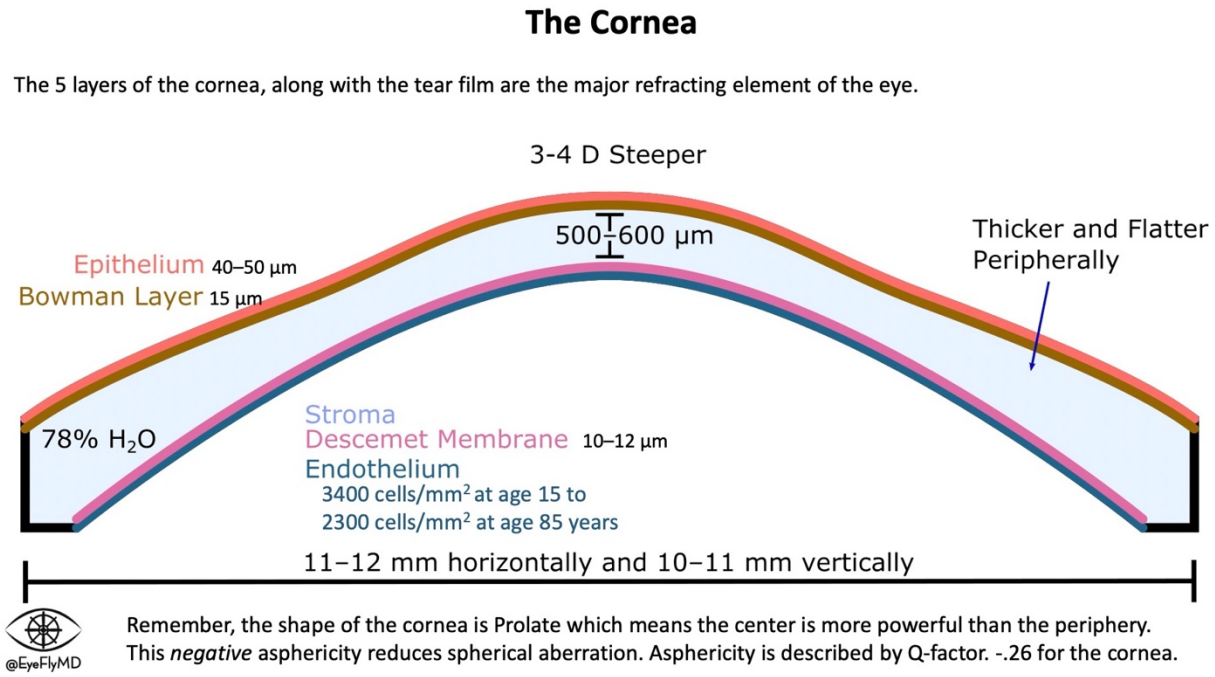
The ***Palisades of Vogt*** are located at the superior and inferior limbus (transition zone between the cornea and sclera) are where the limbal stem cells reside for wound healing. They are deep to avoid mutagenesis from UV exposure. Endothelial cells have limited proliferative potential. 25-33% of stem cells are required to maintain a normal ocular surface.

Light begins is refractive journey at the cornea, specifically, the air-tear interface. Think back to Snell's Law ($n_1 \sin\theta_1 = n_2 \sin\theta_2$). Light changes direction when entering a material of a different refractive index (n). Refractive index can be thought of as "optical viscosity." A refractive index is a dimensionless value and the equation for determining it is: $\frac{speed\ of\ light\ in\ vacuum}{speed\ of\ light\ in\ material} = refractive\ index\ (n)$.

The average tear lake is 7-10 µL and the average drop from bottle is 50 µL for perspective.

The refractive index of the cornea is a tough subject. The average refractive index of the cornea is 1.376 and varies from 1.40 anteriorly to 1.37 posteriorly. For IOL calculation it is often reported as 1.34 (1.3375) though when using standardized corneal radius-of-curvature and dioptric power measurements. Newer IOL calculations use 1.3333 though because 1.34 was overestimating true corneal power.

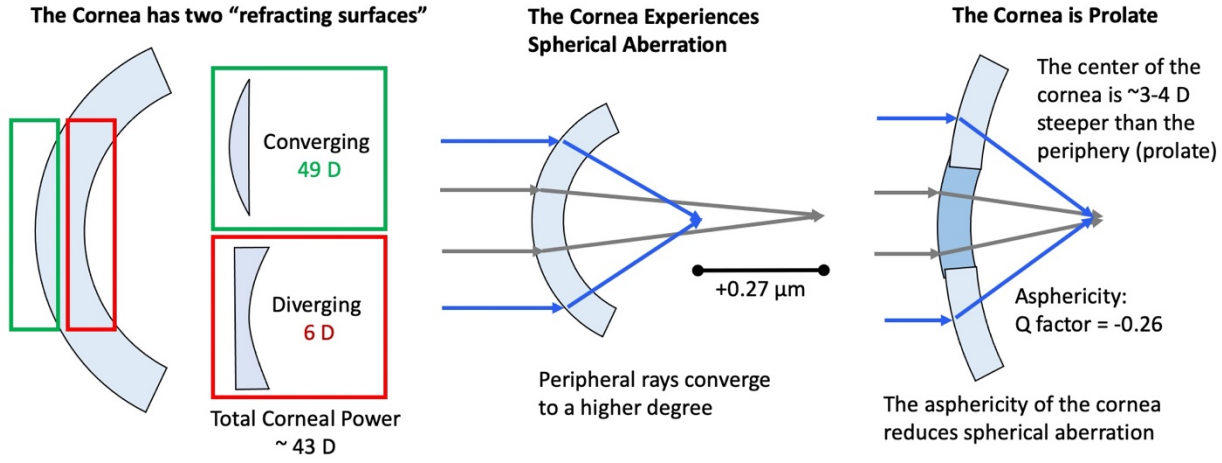
The greatest change in refractive indices occurs when light crosses from air (n ~1.00) to the tear film (n ~ 1.34) so that is where most of the refracting magic happens.



The cornea also has some other important optics. The total refractive power of the eye is ~60 D and the cornea contributes 2/3 to this. The front of the cornea adds about 49 D of convergence to the system and the posterior cornea adds about 6 D of divergence for a total power of ~43 D. Near the periphery of a spherical system light rays with no vergence enter strike the refracting surface at a sharper angle and thus are refracted more. This results in *spherical aberration*. The average eye has +0.27 µm of spherical aberration. This means the peripheral light rays focus ~ 0.27 µm in front of the central rays. To counter this, the cornea is 3-4 D steeper centrally than peripherally to help better focus the light to a single point. This means the cornea is *aspheric*, not sphere-shaped. This amount of asphericity is described by the dimensionless *Q factor.*

The term *prolate* describes when optical surfaces are steeper centrally than peripherally (or flatter peripherally than centrally). This is negative asphericity and is indicated by negative Q factors. An *oblate* cornea is flatter centrally than peripherally (e.g., after myopic refractive surgery) and this positive asphericity is indicated by a positive Q factor. A Q factor of 0 would indicate a spherical surface and this never really happens with respect to the cornea. The middle ~3 mm of the cornea is roughly spherical.

Optics of the Cornea



- The dimensionless "Q factor" describes asphericity, negative indicates steeper centrally. The cornea is -0.26.
- To eliminate the effect of spherical aberration the cornea would require a Q Value of -0.52.
- When we're young, the lens has a Q value of -0.25 so we actually get pretty close eliminating spherical aberration.
- The Q Value of the lens becomes less negative with age, but the pupil also becomes more miotic, eliminating the contribution of the peripheral rays to spherical aberration.

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Anatomy of the Angle and Normal Aqueous Outflow

The angle of the eye refers to the anatomy between the posterior cornea and iris that runs 360° around the eye. The trabecular meshwork (TM) then drains the aqueous humor into Schlemm's Canal, the collector channels, the aqueous veins, and eventually the episcleral veins to the superior ophthalmic vein and cavernous sinus. For uveoscleral flow, the aqueous flows between the ciliary muscles and enters the supraciliary/suprachoroidal space. It's uncertain where exactly it goes next.

The Angle and Aqueous Dynamics

Conventional (Trabecular) Outflow
- Pressure Dependent
- Majority of outflow

Trabecular Meshwork
Uveal
Corneoscleral
Juxtacannalicular
(greatest resistance)

Schlemm Canal

Collector Channels

Aqueous Veins

Episcleral Venous System

To Anterior Ciliary Veins
Superior Ophthalmic Veins
Cavernous Sinus.

The aqueous is produced by the non-pigmented epithelium of the pars plicata portion of the ciliary body.

- Aqueous is produced at a rate of 2–3 µL/min awake
  - Decreases ~50% during sleep
- The anterior segment volume is 200–300 µL
- Aqueous humor is turned over about every 100 minutes

Unconventional (Uveoscleral) Outflow
- Pressure Independent
- Decreases with Age

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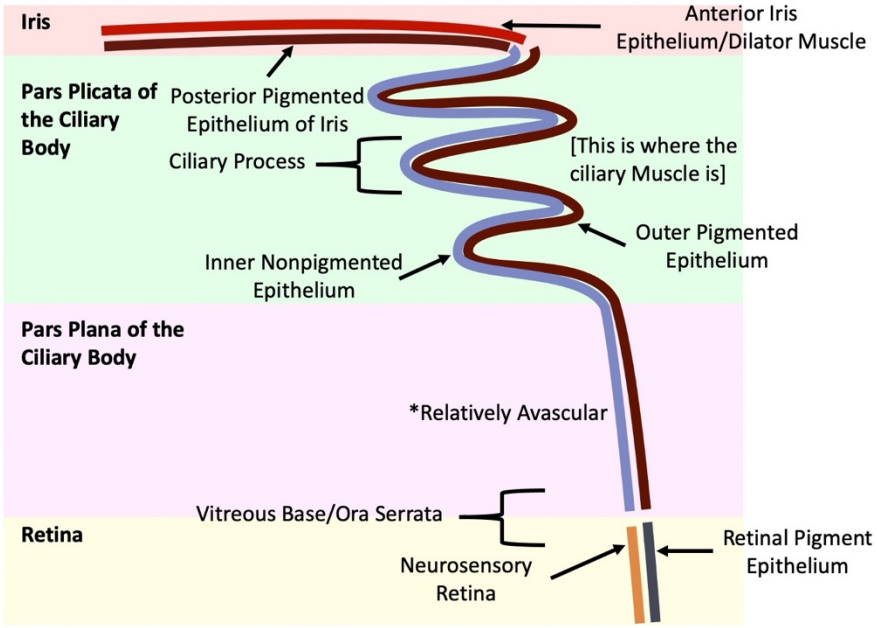
Aqueous is produced and actively diffused into the anterior segment after production by the inner, nonpigmented epithelium of the pars plicata portion of the ciliary body. The outpouchings of ciliary body are called ciliary processes and there are about 80 of them. Remember, the pars plana portion of the ciliary body is the flat area that meets the neurosensory retina. This portion is relatively avascular which makes it the preferred area for intravitreal injections and trocar placement for Pars Plana Vitrectomies.

Continuous Layers of the Retina, Ciliary Body, and Iris

Aqueous is produced by the nonpigmented, inner epithelium of the pars plicata portion of the ciliary body.

There are 70-80 ciliary processes.

Appreciate the the "inner" epithelium looks like the outer portion of the ciliary process but remember "inner" is respective to the eye.



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The **Goldmann Equation** describes the formula for intraocular pressure (IOP). A big takeaway is the 1:1 relationship between IOP and episcleral venous pressure (EVP). Any conditions that raise EVP (e.g., Thyroid Eye Disease) can substantially impact IOP.

The Goldmann Equation

$$P_0 = \frac{F - U}{C} + P_V$$

P_0 – IOP in mmHg
F – Rate of Aqueous Production (µl/min)
U – Rate of Aqueous Uveoscleral Drainage
C – Facility of Trabecular Outflow (µL/min/mm Hg)
P_V – Episcleral Venous Pressure (mmHg)

- Uveoscleral outflow rate cannot be measured noninvasively
- Appreciate the 1:1 ratio between IOP and EVP, this implies that reducing aqueous production or maximizing physiologic outflow can only lower the IOP to EVP which is normally 6–9 mmHg

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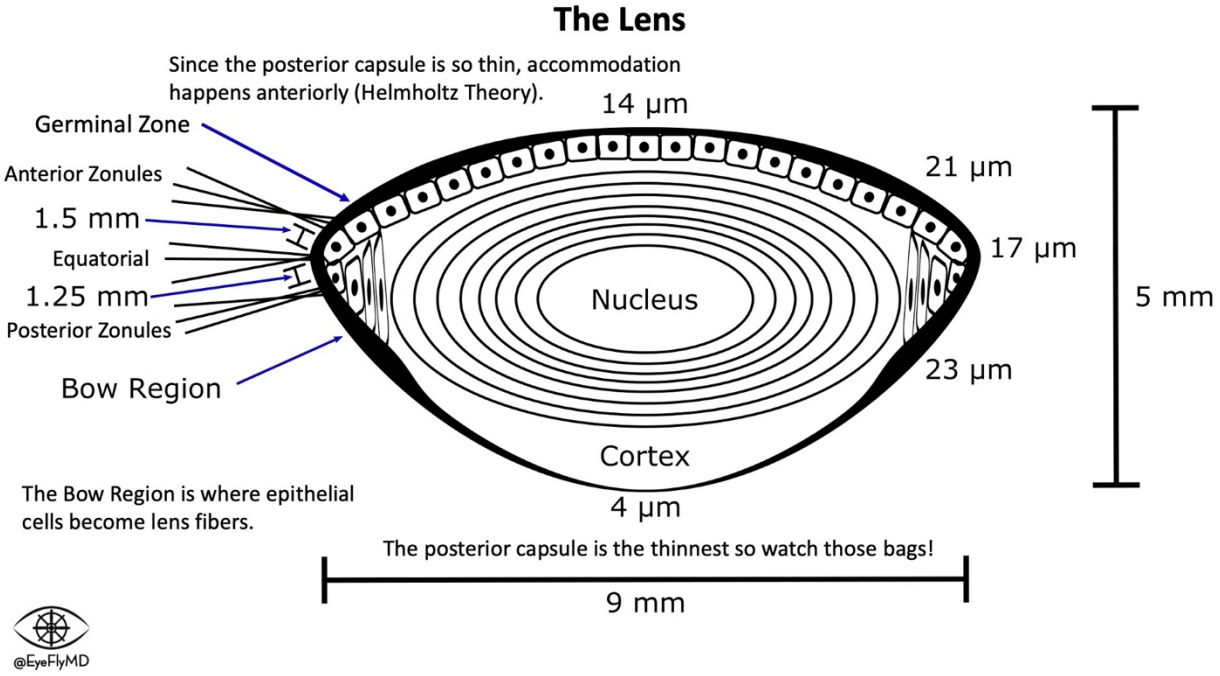
Anatomy of the Lens

The lens is responsible for the other ~20 diopters of the eye's refractive power for a total eye power of ~60 D. It is derived from surface ectoderm. It is surrounded by a capsule (epithelial basement membrane) that is thinnest posteriorly. This means the posterior portion is always distended and most of the change in shape of the lens during accommodation occurs at the anterior portion of the lens (this is called the *Helmholtz Theory of Accommodation*). This also means care must be taken during cataract surgery to avoid rupturing the posterior capsule ("breaking the bag"). The lens has a cortex and nucleus. The epithelial cells proliferate at the *Germinal Zone* anterior to the equator and elongate into lens fibers at the *Bow Region* which is posterior to the equator. The lens can opacify with age and this is what causes cataracts. The lens requires glucose to maintain clarity and its primary metabolism is anaerobic glycolysis. Not too much glucose though or else aldose reductase will convert the excess glucose to sorbitol and precipitate a diabetic cataract.



Anatomy of the Vitreous

The vitreous is the transparent, jelly-like substance that occupies ~ 4 mL of the total 5-6 mL of the posterior segment (aka Vitreous Chamber). It is composed of 99% water and 1% collagen and hyaluronic acid. There are also hyalocytes that help support the vitreous. They derive their oxygen from choroidal diffusion.

The vitreous has high levels of ascorbate (Vitamin C) protect the lens from oxidative damage which is why cataracts are expected shortly after vitrectomy.

The vitreous has five major attachments.

1. **Posterior Lens Capsule (*Ligament of Wieger*)**

The anterior cortical gel (anterior surface of the vitreous) is composed of collagen that attaches to the posterior lens capsule in a ring formation. The posterior lens forms an indentation in the vitreous referred to as the patellar fossa. *The Berger space* refers to the (potential) space in between the posterior lens and anterior cortical gel central to the Weiger Ligament. The attachment to the back of the lens usually persists throughout life except in cases of trauma or disruption to the posterior lens capsule

It's worth mentioning that regarding the Berger space, the BCSC 2020 and 2021 editions are at best vague and at worst incorrect. The phraseology and figure presentation of the BCSC implies it is the donut-shaped potential space *peripheral* to the Weiger ligament. Every single figure online in publication portrays it as the disc-shaped potential space *within* the Weiger ligament between the central posterior lens and anterior vitreous where the *Cloquet Canal* terminates. After a curbside retina consult and reviewing the literature in greater detail, this is the correct location. Berger space is *within/central/bounded by* the Weiger ligament. It's worth knowing this discrepancy.

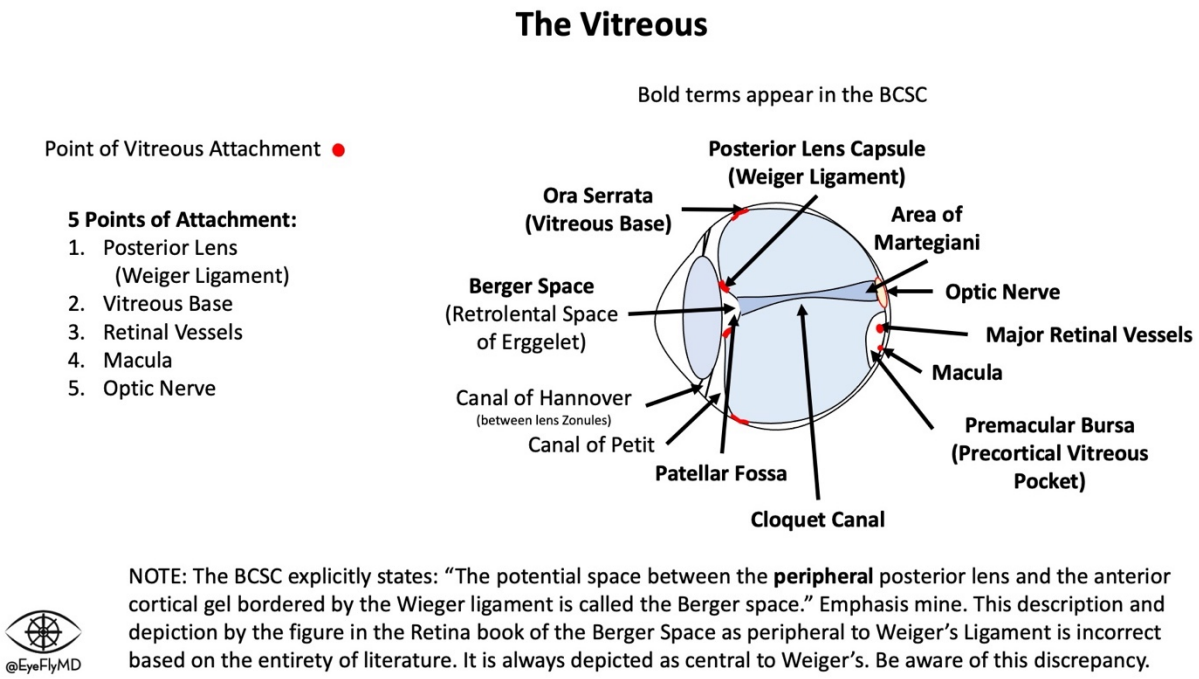
2. **Vitreous Base**

Where the neurosensory retina terminates and meets the ora serrata at the posterior edge of the pars plana portion of the ciliary body there is a firm attachment of the vitreous 2 mm anterior and 3-4 mm posterior straddling the ora serrata in a circumferential fashion. The vitreous base also usually doesn't detach except for cases of trauma.

3. **Retinal Vessels**
4. **Macula**
5. **Optic Nerve**

Posteriorly, the vitreous is attached to the internal limiting membrane of the neurosensory retina at the retinal vessels, optic nerve, and macula by fibronectin and laminin adhesion molecules. Anterior to the macula, there is an area of liquified vitreous called the *premacular bursa* or *precortical vitreous pocket*. The purpose of this area of liquified vitreous is to minimize tractional forces in the area of the macula during regular eye movement (so every motion of the eye doesn't translate to pulling at the macula).

The vitreous is attached to the optic nerve head edge in a circular fashion. The funnel-shaped area without vitreous anterior to the optic nerve head is the called *the area of Martegiani* and is the opening *to the Cloquet canal.* The Cloquet canal is the remnant of the Hyaloid artery, more on this shortly.



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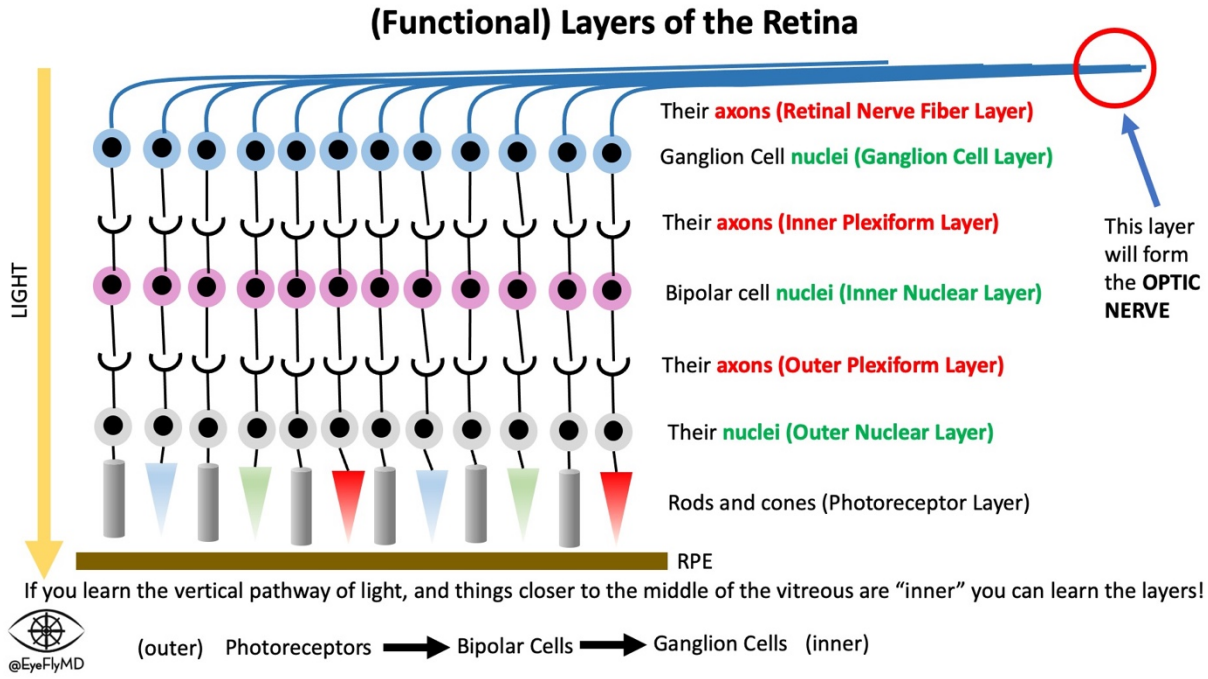
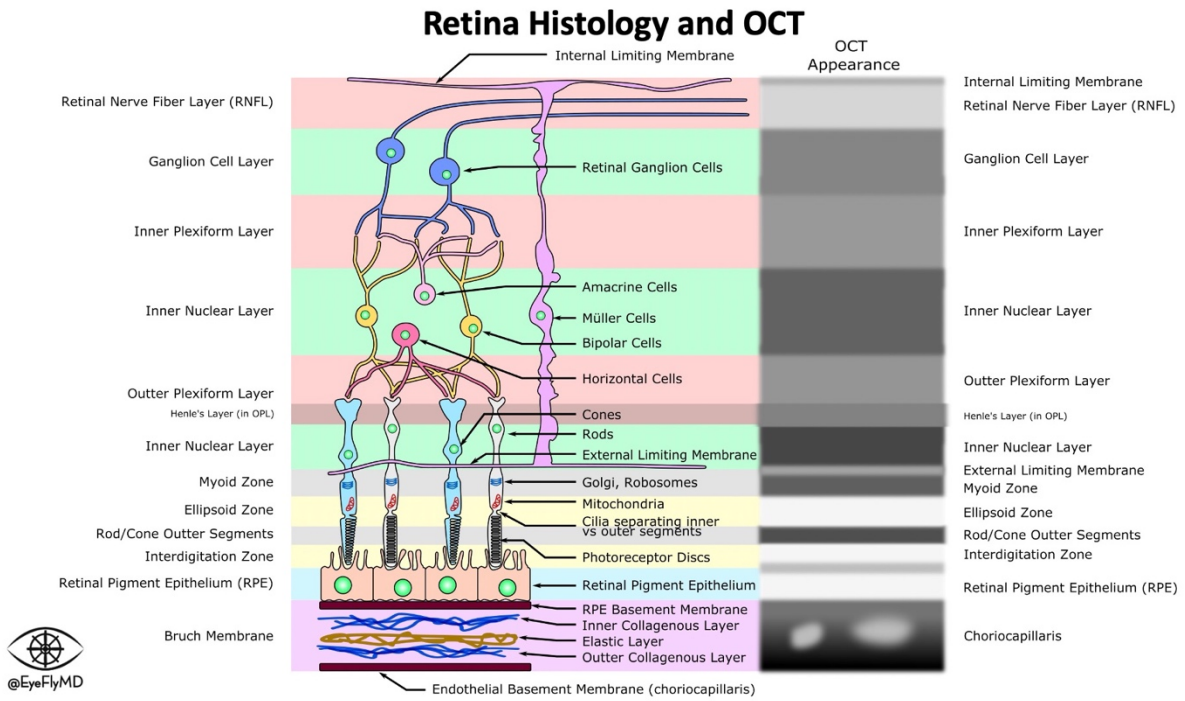
NOTE: The BCSC explicitly states: "The potential space between the **peripheral** posterior lens and the anterior cortical gel bordered by the Wieger ligament is called the Berger space." Emphasis mine. This description and depiction by the figure in the Retina book of the Berger Space as peripheral to Weiger's Ligament is incorrect based on the entirety of literature. It is always depicted as central to Weiger's. Be aware of this discrepancy.

Anatomy of the Retina

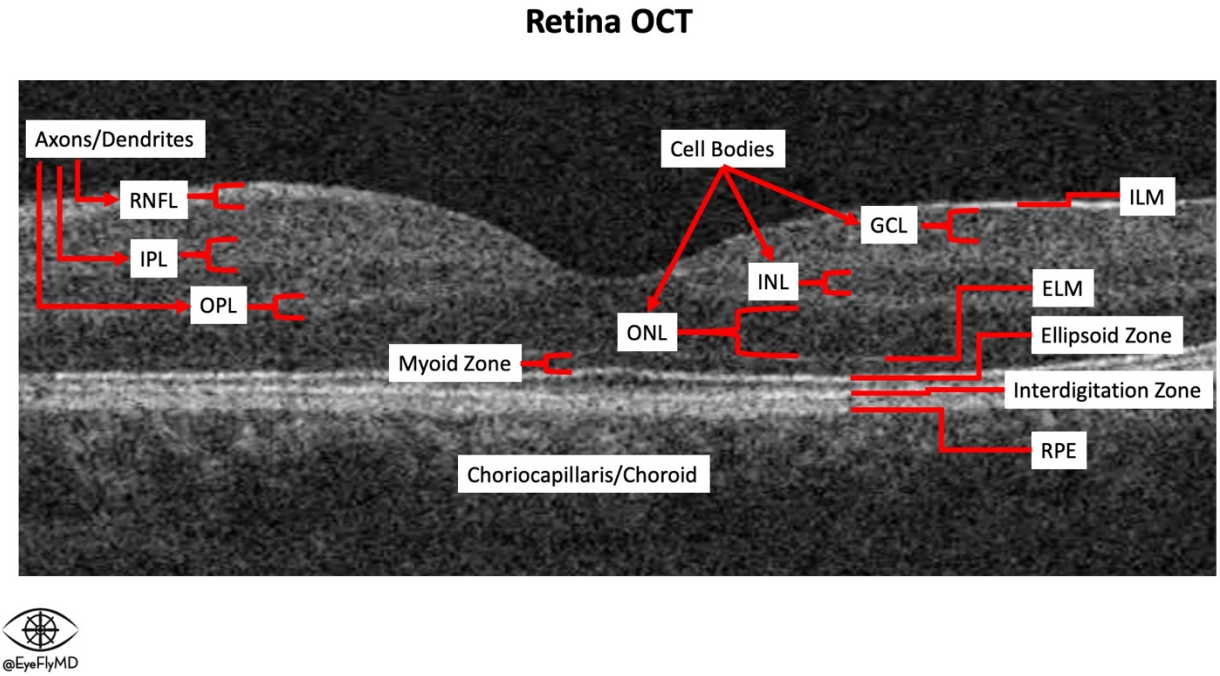
Needless to say, the retina is complex. The best way to memorize the layers of the retina is to first learn the *vertical pathway of light*. Light first begins its physiologic journey to the brain at the rods and cones. The rods and cones do not go all the way to the brain to synapse though, there are "middlemen" called bipolar cells. They modulate the signal and send it along to ganglion cells. It is the ganglion cells whose axons eventually synapse in the lateral geniculate nucleus of the brain (mostly, some make it to the pretectal nuclei of the dorsal midbrain for the pupillary light reflex and some head to the hypothalamus to regulate circadian rhythms). So, the pathway is rods/cones to bipolar cells to ganglion cells. There must be axons/dendrites connecting all these cells. You will notice a "cell body, axons, cell body, axons" pattern emerge. Using the vertical pathway of light and the body/axon pattern you can deduce the retina layers.



Here is a more detailed look at retina histology and how it compares to OCT. You'll notice the inner and outer photoreceptor segments are connected by cilia. You may remember the 9+2 configuration of cilia from medical school, but these cilia are 9+0 as they're nonmotile.

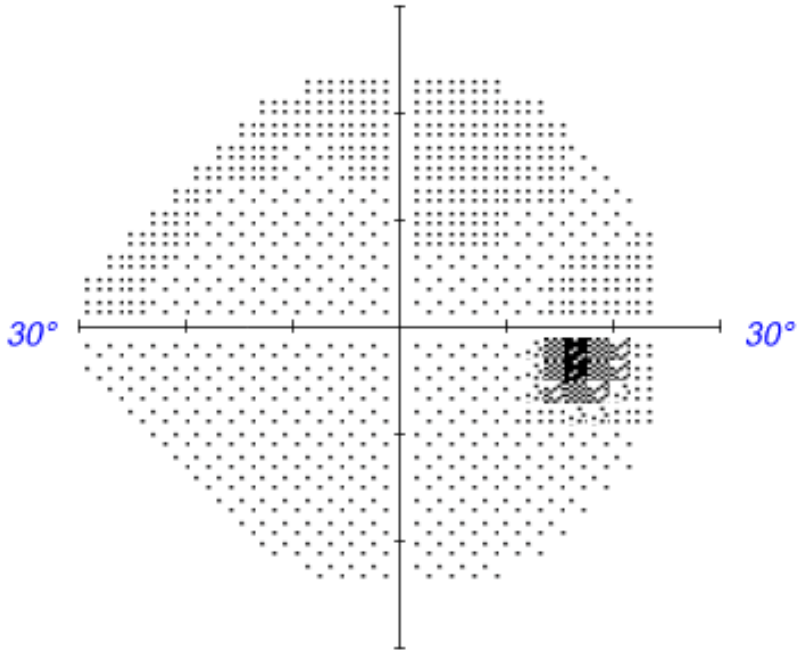


Notice an OCT of the retina (including *my* retina below) follows this pattern. Of course, there's more nuance but if you memorize the big black band is the outer nuclear layer (cell bodies of the rods and cones) you can navigate your way around.



ILM – Internal Limiting Membrane (formed by Müller cells)
RNFL – Retinal Nerve Fiber Layer (axons of Ganglion cells)
GCL – Ganglion Cell Layer
IPL – Inner plexiform layer (axons of INL and dendrites of GCL)
INL – Inner Nuclear Layer (cell bodies of Bipolar cells, Müller cells, Horizontal cells, and Amacrine cells)
OPL – Outer Plexiform layer (axons of ONL and dendrites of INL)
ONL – Outer Nuclear Layer (cell bodies of Rods and Cones)
ELM – External Limiting Membrane (formed by Müller cells)
Myoid Zone – Layer of the Rod/Cone inner segments with endoplasmic reticulum, golgi, and ribosomes
Ellipsoid Zone – Mitochondria-rich layer of the Rod/Cone inner segments
Interdigitation Zone – where the Rods/Cones interact with the RPE
RPE – Retina Pigment Epithelium
Choriocapillaris/Choroid – The vasculature supplying the outer retina

Perhaps you noticed that the neurosensory retina seems "backwards" or "upside down." Why is the light sensitive layer (the rods and cones) underneath the rest of the neurosensory retina? Wouldn't it make more sense if light first hit the rods and cones? It has to do with embryology which we'll discuss a little later. The inverted nature of the retina leads to an interesting phenomenon: the *physiologic blind spot*. Because the axonal component (RNFL) is most interior to the eye, it obviously needs to get out of the eye again to make it to the brain (most fibers will synapse at the lateral geniculate nucleus; the rest will go to the pretectal nuclei of the dorsal midbrain to facilitate the pupillary light reflex and a small portion will head to the hypothalamus to help regulate the circadian rhythm). For these fibers to exit the eye it means they must penetrate the entire thickness of the retina, including the light sensing rods and cones. Since there are no rods or cones where the optic nerve exits the eye there is a natural blind spot there. The nerve inserts nasally so the physiologic blind spot is ~15 degrees temporally and a few degrees inferiorly. A common piece of trivia is the octopus eye does not form the same way as a human and the retina is actually "right side up" so they lack the blind spot (probably, I haven't tried visual field testing on an octopus yet). You can appreciate my (right eye, you can always tell in 24-2s because just picture the cutout for the brow and nose) physiologic blind spot here.



To zoom out a little bit, the center of the retina is the macula. The macula is defined histologically as the area of the retina where the GCL (ganglion cell layer) is ≥ 2 cell layers thick. The macula has 3 common names: area centralis, macula lutea (lutea for the *yellow* xanthophyll pigment there), and posterior pole. The macula is in between the temporal arcades the course from the optic nerve superiorly and inferiorly.

The fovea is usually cited as ~ 3 mm temporal and 0.8 mm inferior to the optic nerve. The foveola lies just within the Foveal Avascular Zone (FAZ). This is where there is no inner/anterior blood supply from the central retina artery. This is also the area where there is no ganglion cell layer or inner nuclear layer, just cones doing their thing.

Anatomy of the Macula

Macula	Between the Arcades	5.5 mm	GCL ≥ 2 cells
Perifovea	Farthest from center	1.5 mm ring	
Parafovea	Rim around fovea	0.5 mm ring	Thickest retina
Fovea	Center of Macula	~ 1 DD (1.5 mm)	All Cones
Foveola	Just within FAZ*	~ 1 cup (.35 mm)	GCL/INL Absent
Umbo	Center of Fovea		

The **PERI**fovea is the furthest from the center because if forms a **PERI**meter.

The fovea contains 10% of the total cone population.

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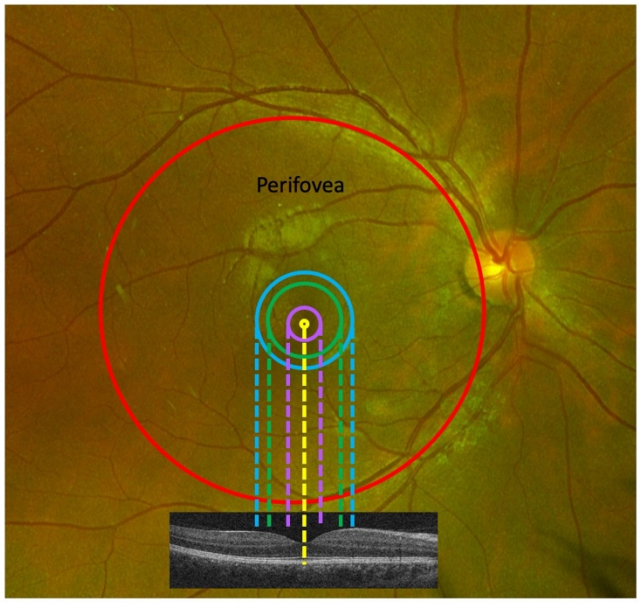
*Foveal avascular Zone, where the inner retina blood supply (from central retinal artery) disappears

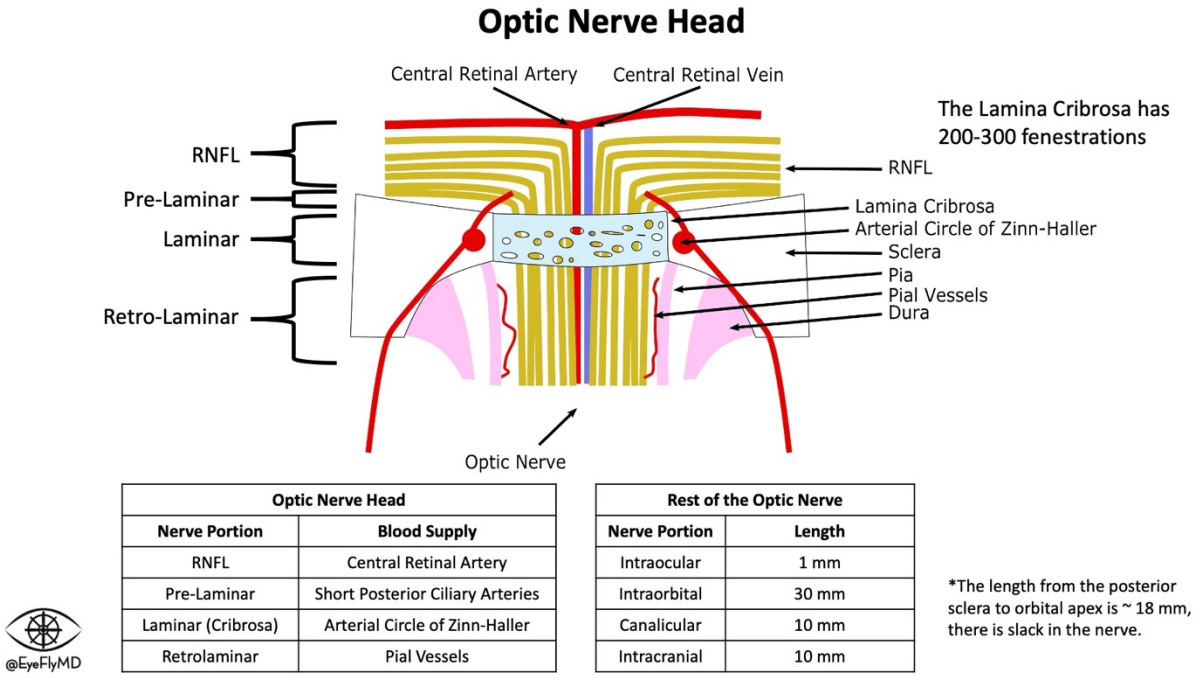


Optic Nerve Anatomy

Once the axons from the retinal ganglion cells coalesce, they form the optic nerve. The optic nerve head extends about 1 mm into the eye and is about 1.50 – 1.92 mm in diameter. Once the axons from the retinal ganglion cells coalesce, they form the optic nerve. The optic nerve head extends about 1 mm into the eye and is about 1.50 – 1.92 mm in diameter with 1.2 – 1.5 million axons. A maximum of 3.7 million axons is achieved by 16 weeks and the final number is reached by 33 weeks. The greatest number of axons the optic nerve ever has is 3.7 million at 16 weeks of gestation before pruning. The lamina cribrosa is the fenestrated portion of the inner 1/3 sclera through which the fibers forming the optic nerve exit. There are 200-300 fenestrations and two large ones allowing the central retinal artery and vein to pass through. After the lamina cribrosa, the optic nerve becomes myelinated and doubles to 3 – 4 mm in diameter. Sometimes the axons of the RNFL can be myelinated within the neurosensory retina and this is called myelinated (or medullated) RNFL. It appears as white patches and presents as visual field defects. The lamina cribrosa is the furthest anteriorly the intracranial pressure can exert its force.

The length of the nerve between the posterior sclera and orbital apex is almost twice as long as the physical space to allow slack for normal ocular movement. During orbital compartment syndromes, when blood or some other space occupying lesion becomes trapped in the orbit, it can cause proptosis and pull the posterior sclera by the optic nerve. This is called “tenting” of the globe.

As discussed earlier, it's hypothesized that CCT is associated with increased risk of glaucoma because it reflects overall integrity of the eye wall including the lamina cribrosa. In glaucoma, when the cup becomes very deep and the tissue becomes very thin it can sometimes be possible to see the fenestrations of the lamina cribrosa. Speaking of the optic cup, this is just the part of the disc where the fibers begin "plunging" into the sclera. A normal cup is about 0.3 of the optic disc (cup-to-disc, or C/D, ratio). More on this later but look at the figure below and picture that as



Optic Nerve Head	
Nerve Portion	**Blood Supply**
RNFL	Central Retinal Artery
Pre-Laminar	Short Posterior Ciliary Arteries
Laminar (Cribrosa)	Arterial Circle of Zinn-Haller
Retrolaminar	Pial Vessels

Rest of the Optic Nerve	
Nerve Portion	**Length**
Intraocular	1 mm
Intraorbital	30 mm
Canalicular	10 mm
Intracranial	10 mm

*The length from the posterior sclera to orbital apex is ~ 18 mm, there is slack in the nerve.

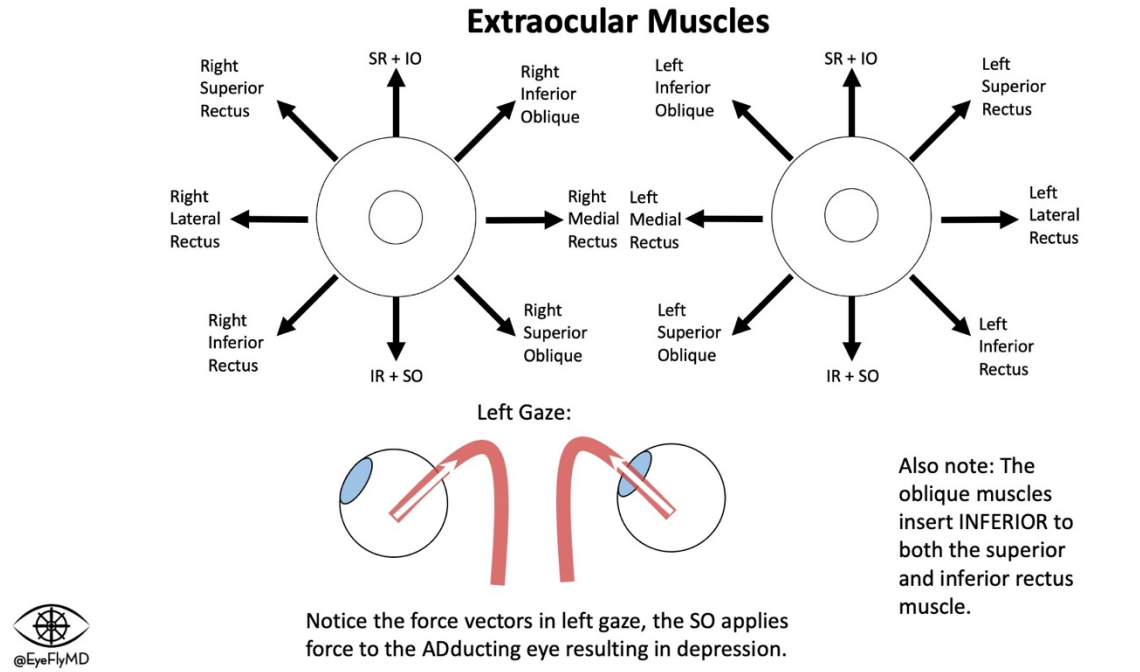
the fibers die, they will appear to "plunge" into the sclera further and further from the center. This will enlarge the C/D which is characteristic of glaucoma.

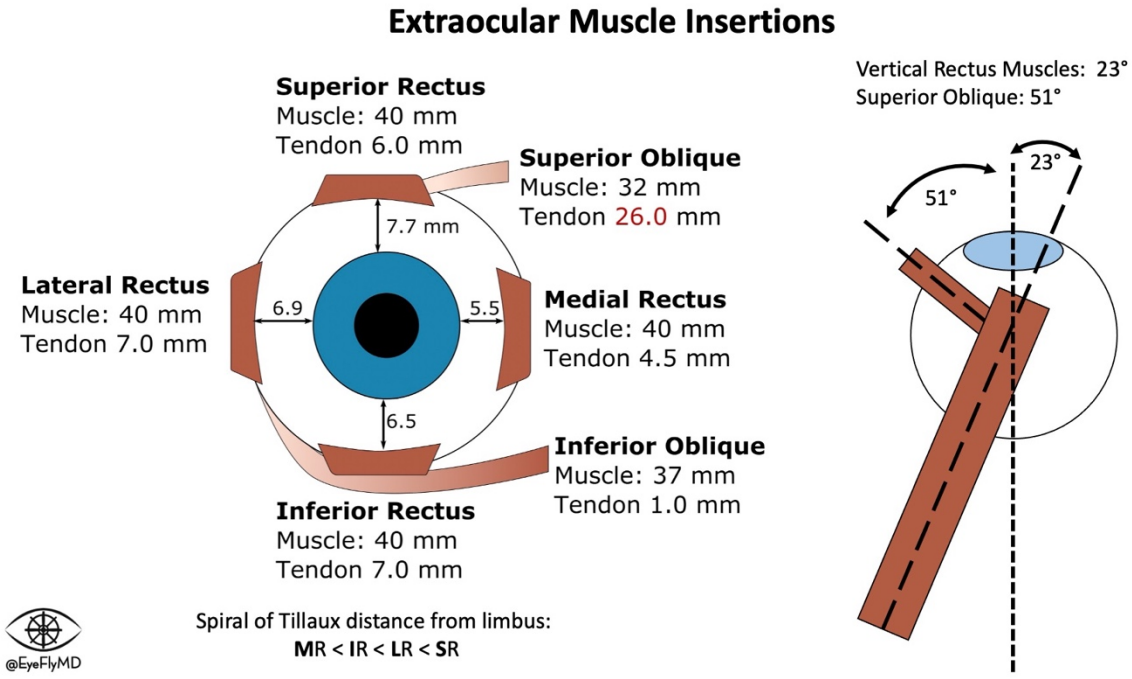
Extraocular Muscles

Let's exit the eye and think about the extraocular muscles. The four rectus muscles originate at the *Annulus of Zinn* (a fibrous ring surrounding the optic canal and a portion of the superior orbital fissure). The superior division of CNIII innervates the superior rectus (and the levator) and the inferior branch of CNIII innervates the medial rectus, inferior rectus, and inferior oblique. The superior oblique is innervated by CNIV and the lateral rectus is innervated by CNVI. The medial rectus is the strongest muscle. There is a condition called "*Duane Retraction Syndrome*" where CNVI does not develop and is innervated by CNIII instead. On attempted ADDuction both the medial and lateral recti contract which can result in retraction of the globe into the orbit.

From the figure below, we can appreciate the simple "up" and "down" gaze require the input of two muscles. This is why having patients follow your finger in an "H" pattern is effective; it isolates each muscle's function. The oblique muscles are the primary cyclotorsional (eye rotating around the pupillary axis) muscles. The superior oblique and superior rectus are responsible for INtorsion and the inferior oblique and inferior rectus are responsible for EXtorsion.

Another fact that comes up often is where the muscles insert on the eye. The four rectus muscles insert in a pattern called the *Spiral of Tillaux*. The inferior rectus inserts near the macula. The sclera is also thinnest just posterior to the muscle insertions (0.3 mm) so the most common locations for globe rupture are at the limbus, posterior to the muscle insertions, and at the equator. During strabismus surgery you might be asked if a dark patch behind the muscle insertion concerning and the answer is no, that's just the choroid showing through the thin sclera.



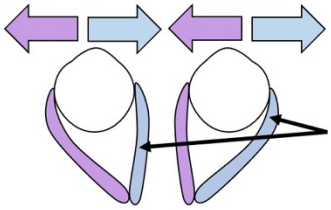


There are two special rules governing extraocular muscle action called ***Herring's Law*** and ***Sherrington's Law***. Herring's Law basically says innervation to conjugate muscles is equal. In other words, innervation to muscles responsible for conjugate gaze between the two eyes is the same to ensure the eyes work together. Sherrington's Law basically says innervation to the yoke muscles is opposite. The yoke muscles are on opposite sides of the globe. In other words, the medial rectus and lateral rectus shouldn't fire at the same time and when one receives an increase in innervation the other should receive a decrease to prevent the globe retracting in the orbit. Does that sound familiar? Duane Retraction Syndrome violates Sherrington's Law.

Motility Laws

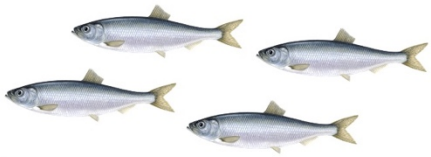
Hering's Law

of Equal Innervation



In right gaze, the RLR and LMR are innervated equally so the eyes move ***together***

EOMs responsible for each eye's movements are innervated equally



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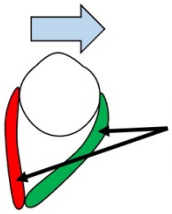
Imagine a school of **Herrings** swimming ***together***

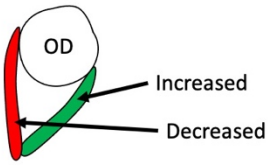
Sherrington's Law

of reciprocal innervation



In right gaze in the right eye, the innervation to the RLR is **increased** and the innervation to the RMR is **decreased**; these muscles are in the ***same eye***

Increased innervation of a given EOM is accompanied by a reciprocal decrease in innervation of its antagonist.



Imagine the muscles of **Sherring**ton's Law ***sharing*** and eye

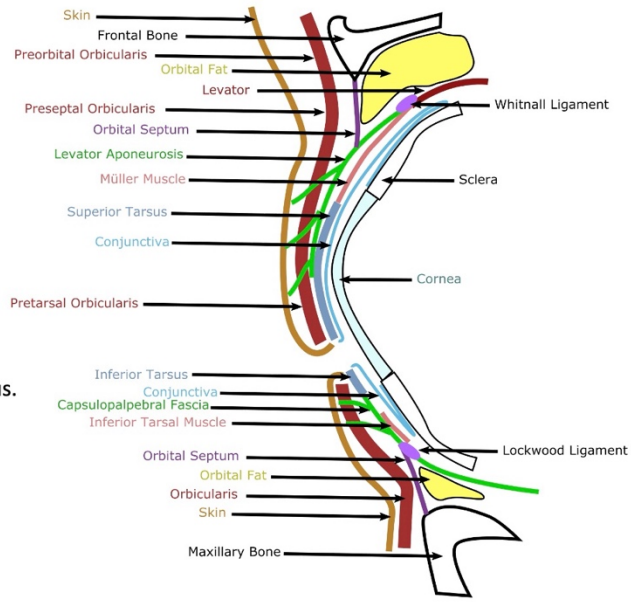
The Eyelids

The eyelids are also deceptively complex. The many layers are outlined below. It's also worth thoroughly understanding the inferior eyelid counterparts to the superior eyelid anatomy. The purpose of *Whitnall* and *Lockwood* ligaments is to redirect the horizontal force applied by the muscles to be vertical so the eyelids open and close rather than retract into the orbit.

Eyelid Anatomy

Superior/Inferior "Siblings"	
Superior	**Inferior**
Tarsus	Tarsus (1/3 as tall)
Müller Muscle	Inferior Tarsal Muscle
Levator Aponeurosis	Capsulopalpebral Fascia
Whitnall Ligament	Lockwood Ligament
Levator Palpebrae Superioris	Capsulopalpebral Head (arises from terminal muscle fibers of the inferior rectus muscle)

Note: The eyelid crease is due to the levator aponeurosis-pretarsal orbicularis muscle/skin attachment above the tarsus. In Asian eyes, the attachment is lower and there are less aponeurotic attachments to the skin which is why the eyelid crease is less pronounced.



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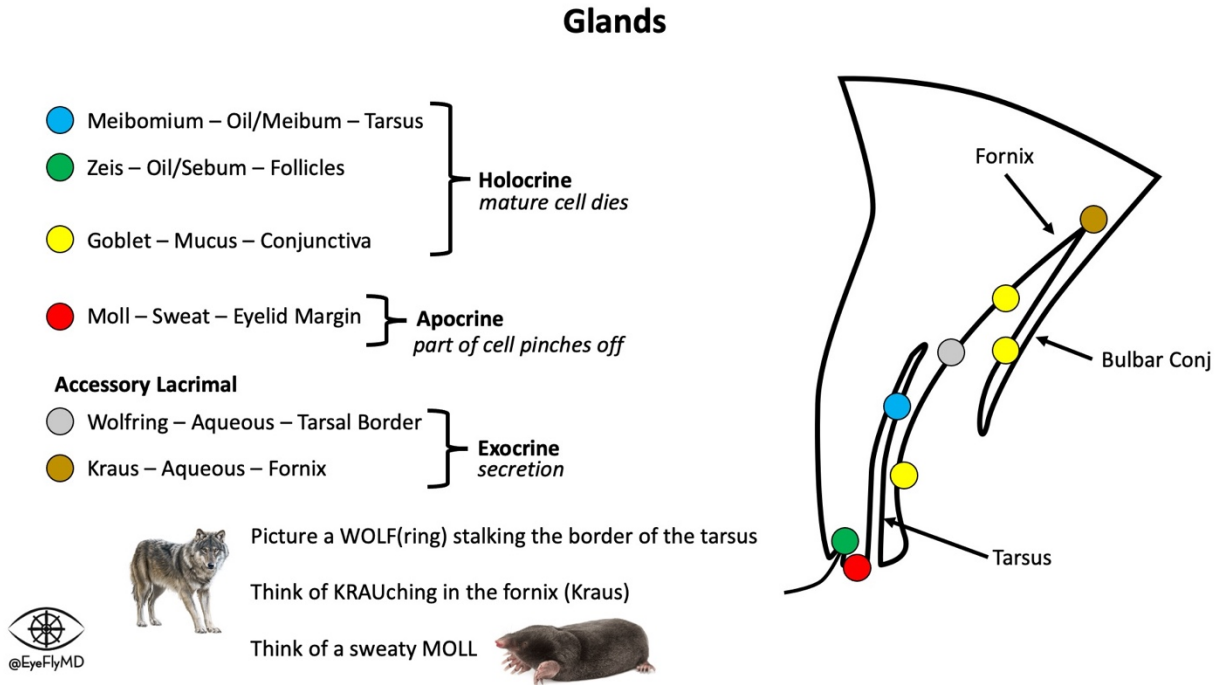
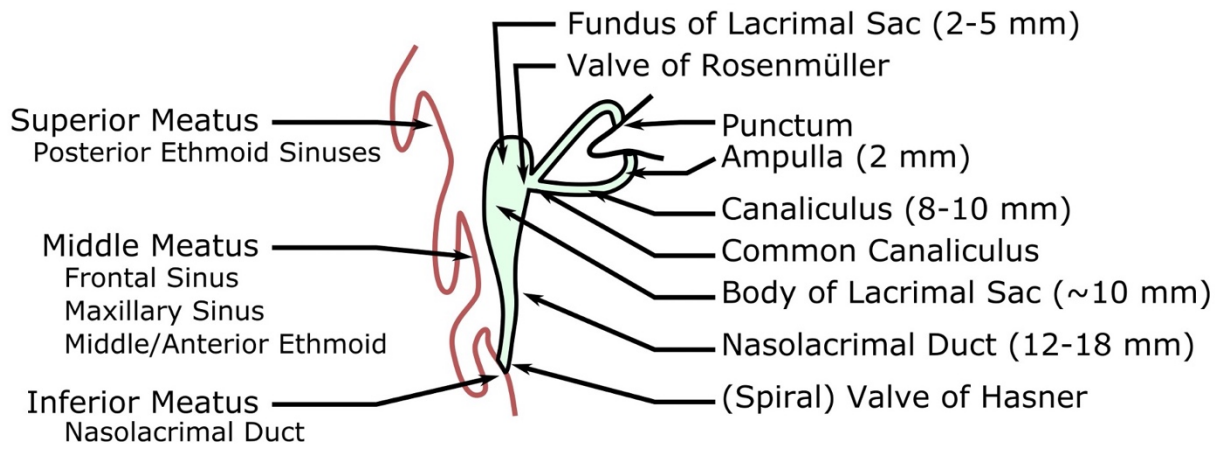
There are also several glands that are part of the eyelids that help to maintain a healthy tear film. In addition to the main lacrimal gland, there are *accessory lacrimal glands*, the glands of *Wolfring* (near the tarsal border) and *Kraus* (in the fornix) that also secrete the aqueous portion of the tear film. The oil/sebaceous portion is secreted by the Meibomian (tarsal) glands in the tarsal plate and the glands of *Zeis* in the hair follicles. There are 30-40 meibomian glands of the upper eyelid and 20-30 of the lower. Goblet cells scattered throughout the conjunctiva and secret mucus. The sweat glands of the eye are the glands of *Moll*, located at the eyelid margin.



Part of the complexity of the eyelids is the nasolacrimal system. There is a superior and inferior punctum that can both be occluded with plugs as a treatment for Dry Eye Disease (DED). The puncta open to an ampulla and then a canaliculus which drains into the lacrimal sac via the *Spiral Valve of Hasner*. If this valve experiences a membranous obstruction at birth, dacryocystitis (inflammation/infection of the nasolacrimal system) can occur. As many as 50% of newborns have an obstruction but the valve of Hasner self-perforates in the majority by 6 weeks which is about the time significant tear production begins.



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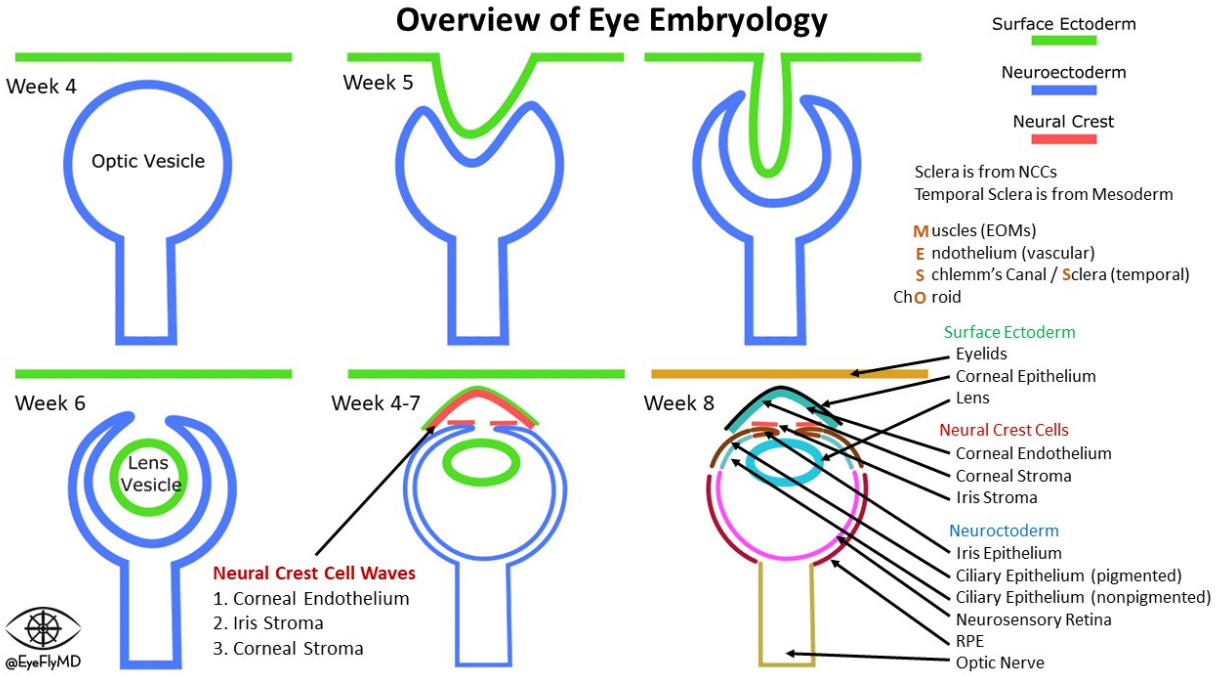
Total Length of Lacrimal Sac: 12-15 mm, fundus is superior to medial canthal tendon
Average Length from Punctum to Nasolacrimal Duct: 23 mm

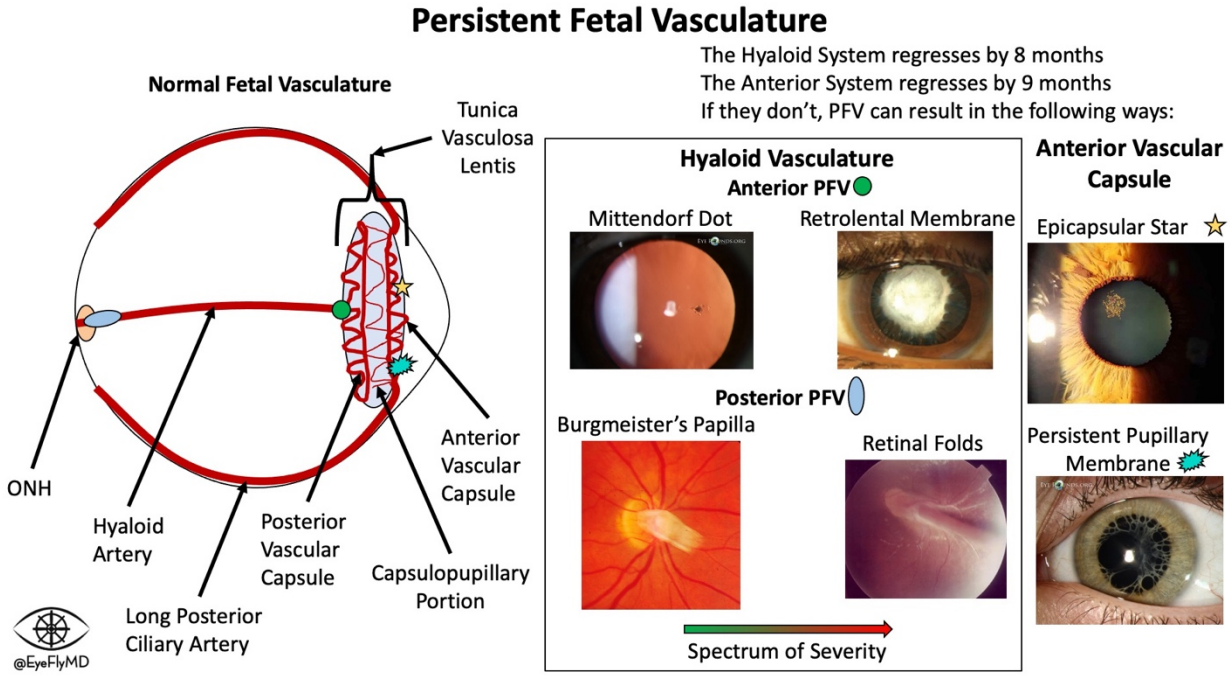
Embryology

Where did all this come from? The figure below is an overview from beginning after the optic vesicle has formed from an outpouching of the neural tube. In the most basic sense, the retina/RPE/optic nerve and everything continuous with those structures (ciliary body epithelium and posterior iris epithelium) derive from neuroectoderm. The anterior chamber mostly derives from neural crest cells. The lens, corneal epithelium, and eyelids derive from surface ectoderm. See the figure below for details and exceptions.



One more note on embryology. The developing eye and lens require nutrients and a vascular supply. The **hyaloid artery** extends from the optic nerve head to the posterior lens and supplies the developing retina and posterior lens. The long posterior ciliary arteries supply the anterior portion of the lens and an anastomosis between the two vascular supplies is called the capsulopupillary portion. Together, this all forms the ***tunica vasculosa lentis*****.** The hyaloid system normally regresses by 8 months and the anterior system by 9 months. This is important because failure of the hyaloid system to regress results in **Persistent Fetal Vasculature (PFV)**. The hyaloid artery normally leaves a space through the vitreous called the *Cloquet canal* referenced earlier. Anterior PFV involves the retrolental space and can present on a spectrum from a small inferonasal remnant of the attachment to the back of the lens (***Mittendorf Dot***) to a fibrous, vascularized membrane that threatens the eye. The eye is usually microophthalmic in these cases with a shallow anterior chamber and a small, cataractous lens with visible elongated ciliary processes. Posterior PFV involves the optic disc and exists on a spectrum from a small tuft of glial tissue (**Burgmeister's Papilla**) to persistent branches from the hyaloid artery or a retinal fold. The eye is usually microphthalmic in these cases.

Anterior PFV is still failure of the hyaloid system to regress. Don't get this confused with the anterior fetal blood supply. Failure of the anterior blood supply (the long posterior ciliary arteries) to regress results in either an *epicapsular star* (sometimes described as chicken feet) on the anterior capsule or a *persistent pupillary membrane* (small strands of iris in the anterior chamber). Both of these are rarely clinically significant.



Basic Optics and Refractive Principles

Important Refractive Concepts

Visual Acuity (VA)

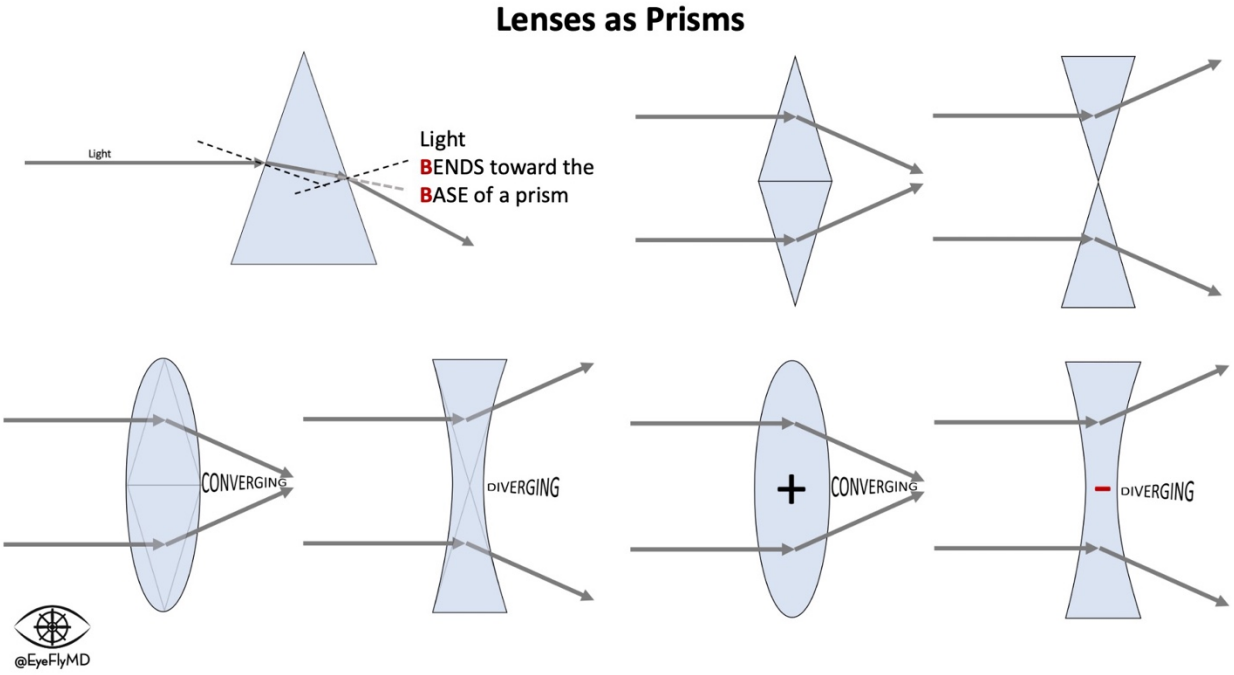
Visual Acuity is a measure of the vision clarity. There are different ways to measure visual acuity and the most common way in clinic is with the Snellen chart. There are different type of visual acuity tests and *minimum legible threshold* is the most common. As it sounds, this tests the ability to discern smaller and smaller optotypes (letters). The Snellen chart is the most common method. It is reported as the distance patient could read a set of optotypes (letters) divided by the distance for a person with normal vision. So, 20/60 means the patient can see at 20 feet what a normal person could see at 60. 20/15 means the patient can see at 20 feet what a normal person can see at 15. The technical definition of 20/20 vision is the ability to resolve a separation of one minute of arc on the retina.

20/20 is “normal” vision, around 20/60 driving becomes questionable, and 20/200 is legal blindness. 20/800 is where walking around becomes challenging.

Lenses (As Prisms) and Vergence

If you can remember what a prism does to light you will never forget what plus or minus lenses do to light ever again. Due to Snell’s law, light bends towards the base of a prism. The dotted lines on the prism below represent the *normal*, the imaginary line perpendicular to the optical surface. When light enters a substance with a greater refractive index it bends towards the normal. When light enters a substance with a lesser refractive index it bends away from the normal. The n of air is 1.00. Other important ns include the cornea (1.376 as discussed before), aqueous (1.336), vitreous (1.337), water (1.333), and crown glass (1.517).

Spherical lenses can be thought of as two prisms either apex to apex or base to base. Light has 3 options, it can either converge, diverge, or have zero vergence (be parallel). Plus lenses (two prisms base to base) are converging lenses and minus lenses (two prisms apex to apex) are diverging lenses.



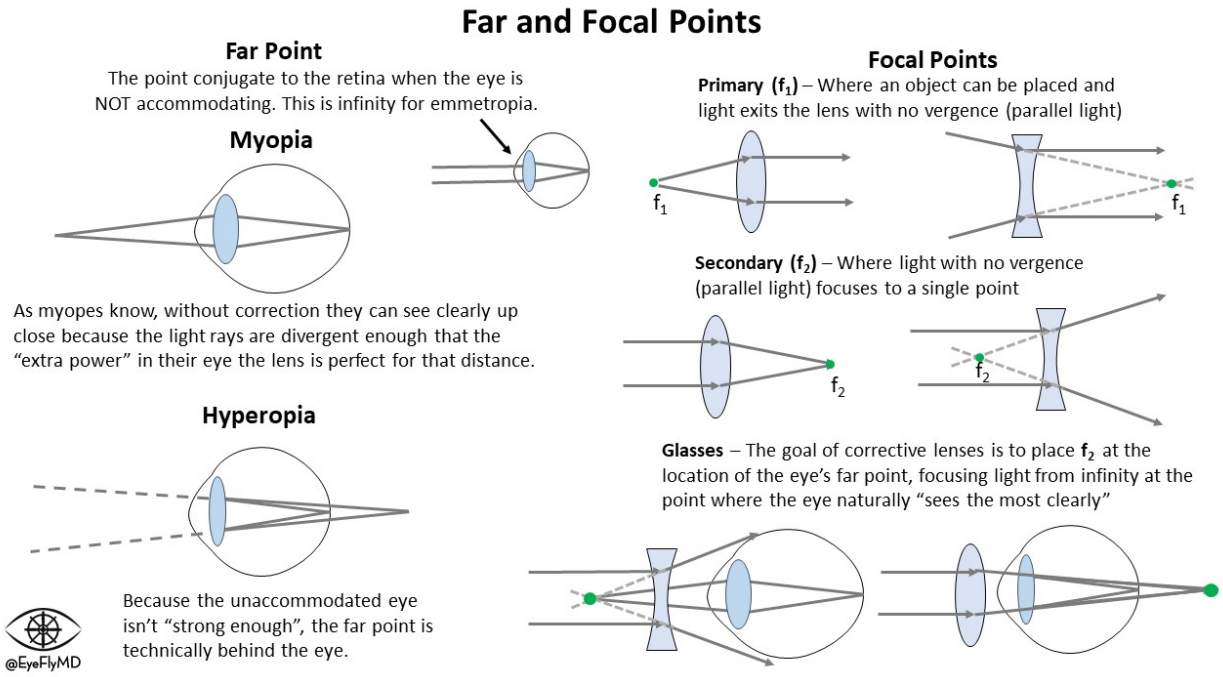
Far Points and Focal Points

The eye must take light with no vergence (remember, parallel light from infinity) and focus it to a single point on the retina over a distance of about 24 mm. This requires quite a bit of refracting power and the average eye is ~ + 60 D. The eye must be plus naturally because the light must be converged onto the retina. The cornea (especially the air-tear interface where the biggest refractive index change occurs) contributes ~2/3 of this refractive power and the lens does the rest.

Eyes have a **Far Point**. This is the point conjugate to the retina when the eye is not accommodating. Put another way, this is the point where an object could be placed and the emitted/reflected light rays would focus onto the fovea after passing through the eye's optical system. For myopes, either the lens is "too powerful", or the axial length is too long and light rays with no vergence will focus in the vitreous. For hyperopes, the lens is either "too weak" or the eye is too short and the incoming light would focus behind the retina but strikes the retina as a blurry circle.

Lenses have **Focal Points**. A primary focal point (f_1) is a point where an object could be placed and the light rays coming from that object would leave the lens system with zero vergence. A secondary focal point (f_2) is where light with zero vergence would focus to a single point. For minus, concave lenses, the focal points are the locations where the light rays "appear" to be going or coming from because the light rays in a concave system may never actually cross to form a single point. This is indicated with dotted lines below.

The goal of corrective lenses in a pair of glasses then is to take the light coming from a distance with zero vergence and focus it to the conjugate point of the unaccommodated eye, the **Far Point**. Remember, the far point is where light will focus on the retina after passing through the unaccommodated optical system of the eye. You can think of a corrective lens then as taking light from infinity and focusing it to the point where the eye sees most clearly. As far as the is concerned then, it is seeing that "image" of infinity since all the light is focused there and the result is clear distance vision.



Refractive Error

Emmetropia

This is term for an eye without refractive error. Light with zero vergence from a distance will pass through the optical system of the unaccommodated eye and focus on the retina.

Myopia

As referenced before, Myopia or "nearsightedness" is a refractive error of the eye characterized by light focusing IN FRONT OF the retina after passing through the optical system of the unaccommodated eye. Myopes can see up close but not at distance.

- Correction: **Concave** lenses that diverge the light (denoted by a - prescription)
    - One way to think of it is that a concave lens will "spread" the light rays or "push" the image back so it falls nicely on the retina.
    - Another way to think about it is that the concave lens will focus light with zero vergence to f_2 which is powered to align with the far point of the eye (in front of the eye in myopes).

Hyperopia

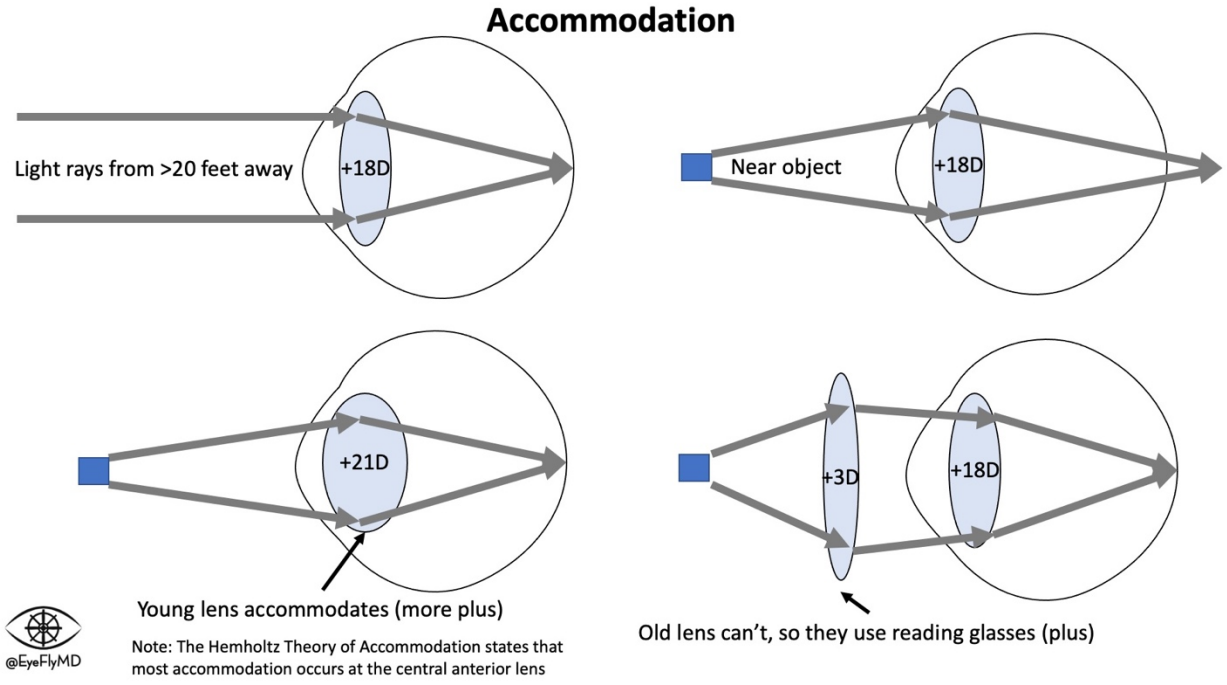
Hyperopia or "farsightedness" is a refractive error of the eye characterized by light focusing in BEHIND the retina after passing through the optical system of the unaccommodated eye. Technically for the *unaccommodated* hyperopic eye no distance (near or far) will result in clear vision but (especially young) hyperopes can simply accommodate, make their lens more convex (plus) and see well at distance.

- Correction: **Convex** lenses that converge the light (denoted by a + prescription)
    - One way to think of it is that a convex lens will more "sharply" focus the light rays or "pull" the image forward, so it falls nicely on the retina.
    - Another way to think about it is that the convex lens will focus light with zero vergence to f_2 which is powered to align with the far point of the eye (behind the eye in hyperopes).

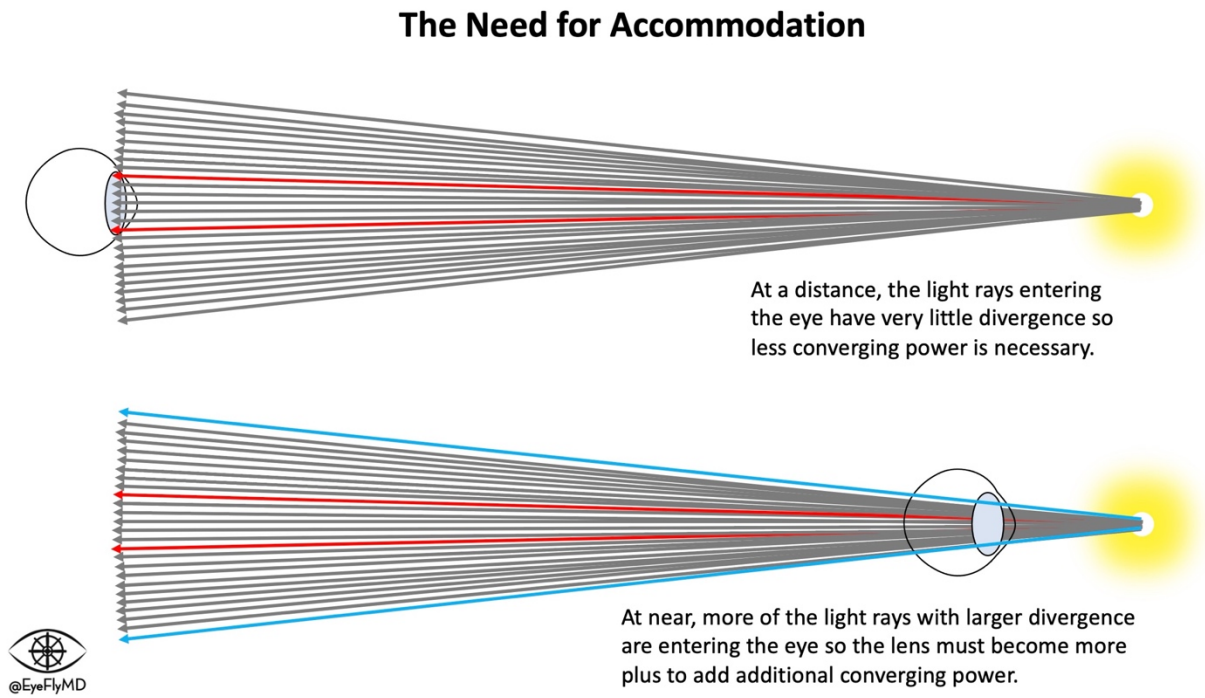
Accommodation

When focusing on near things (within 20 feet) the eye must accommodate to allow the light to keep focus on the retina. The end result of this process is the lens bulging and becoming more convex (positive) to increase the refractive power of the lens and increase its converging power. The process is:

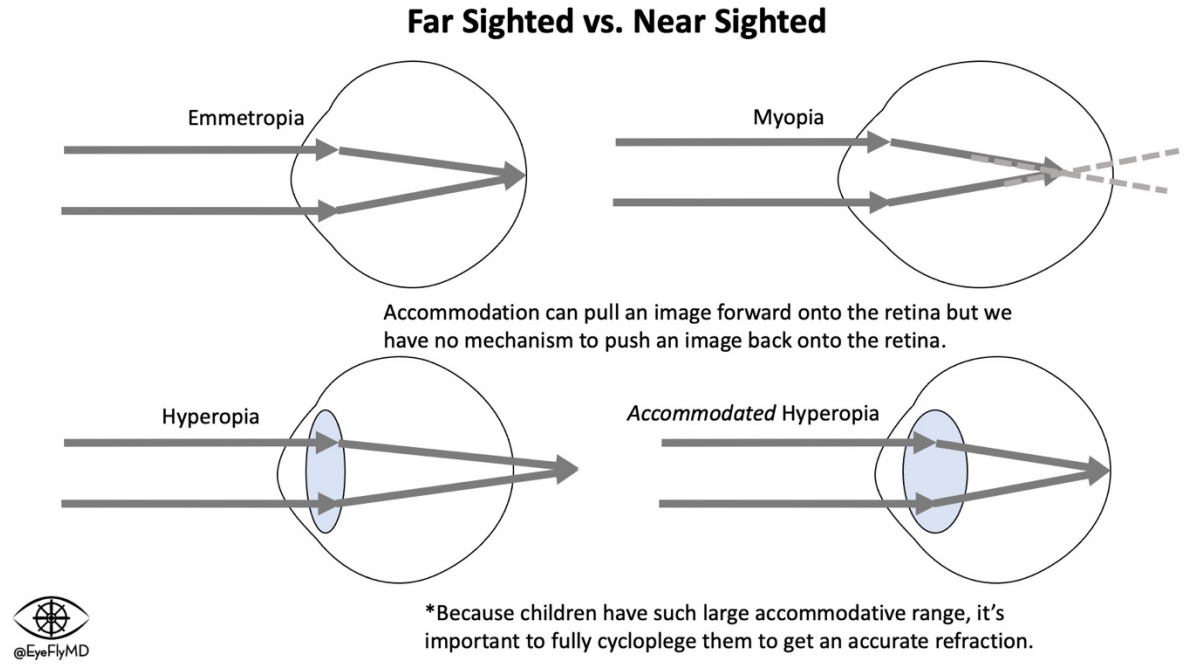
- The ciliary muscles contract
- This loosens the zonular fibers supporting the lens
- The lens bulges becoming more CONVEX (+) and increases converging power



Why is accommodation even necessary? Consider that the closer the eye is to a light source the more divergent the rays of light are. If an eye is infinitely (well, about 20 feet) far from a light source the only rays of light making it in the pupil are essentially parallel to each other. A light source held very close to the eye though means more of the diverging light is making it through the pupil so the refracting system of the eye must increase its converging power to compensate for this. This brings us to exam room mirrors…



After that detour it brings us back to our original questions, why can hyperopes see at any distance if the Far Point is behind the eye? Because they can accommodate and increase the power of their refracting system. They can dial in their own prescription to place the image on the retina.

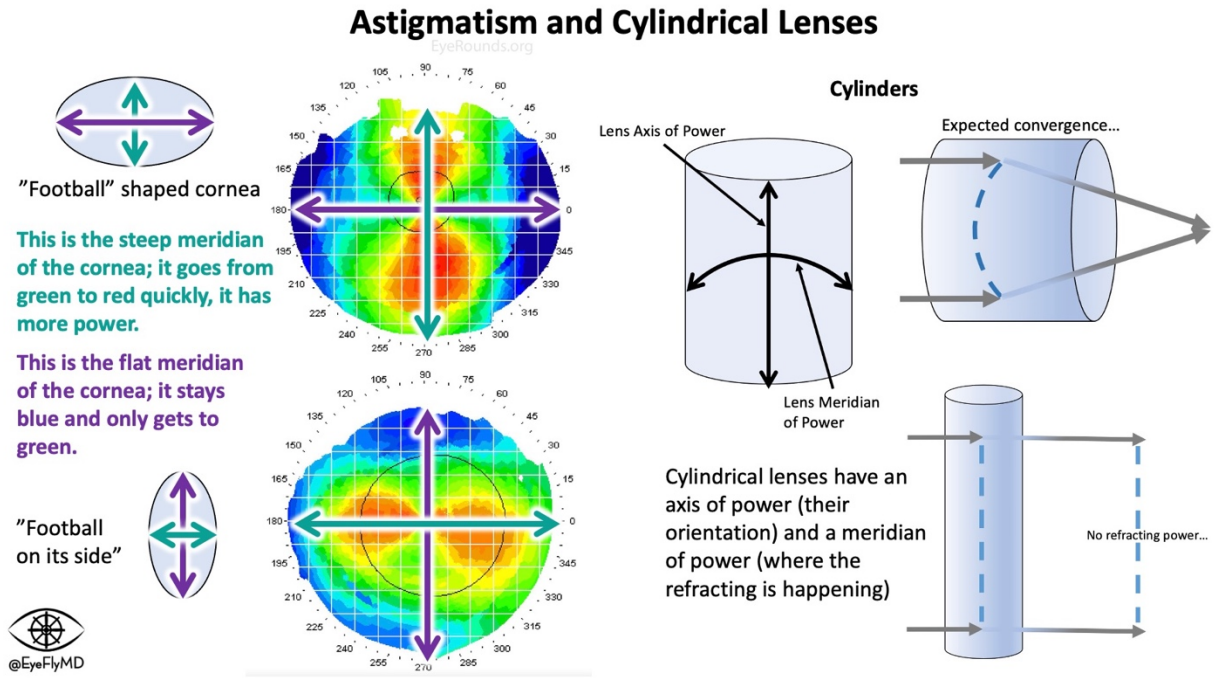


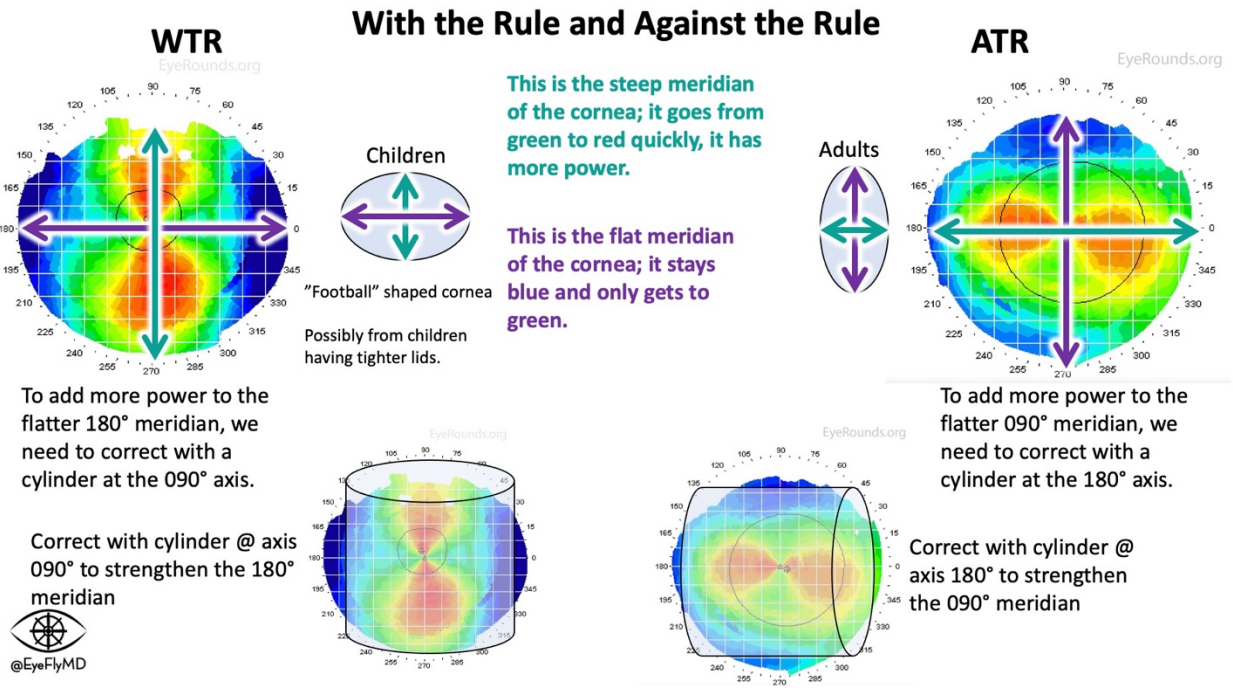
Do you think a patient would prefer if their prescription is too minus or too plus? Think about if their prescription was too plus first. Too much convex, converging power will cause the image to fall closer to the lens, thus in the vitreous cavity and in front of the retina. Does the eye have a mechanism to "push" images back onto the retina? NO. There is no way for the eye to effectively "weaken" (make more minus) it's refracting power beyond relaxing the ciliary body and letting the les assume it's naturally least powerful configuration. The does though have a way to make itself more powerful (more plus) and that's the whole principle of accommodation. If a prescription is too minus the image will fall behind the retina. The (young) eye can just increase its power, become more plus, and increase the convergence of the light so the image falls nicely on the retina. This is the reason hyperopes don't really need glasses, especially when they're young and can dial in their own prescription. Constant accommodation can cause a headache and if a young child is in the +4.00 range the amount of accommodation can cause esotropia due to the near triad (miosis, accommodation, and **convergence**).

Astigmatism

So far, we've only discussed spherical lenses and prescriptions for round corneas. What if the cornea (or lens) is not round? Consider a football-shaped cornea. It has two radii of curvature, one along the stitching and one you'd wrap your hand around. The radius along the stitching is bigger and the surface is flatter. The radius you'd wrap your hand around is smaller and the surface is steeper. Corneas can do this too and this is called astigmatism. The cornea then is not focusing light to a single point and each of these axes require their own prescription to pull the two focal planes together.

How do we control two axes individually? With **cylindrical lenses**. Imagine a glass cylinder like pictured below. A cylinder has an axis of power running lengthwise along the cylinder and a meridian of power over the actual curved surface. The axis of power dictates its location but the refracting magic happens along the meridian of power. As light passes through the cylinder light striking various points along the meridian of power converge as we would expect but along the axis of power light passes through the cylinder uninterrupted. This means can individual control the power of the corrective lenses based on the astigmatism of the cornea.

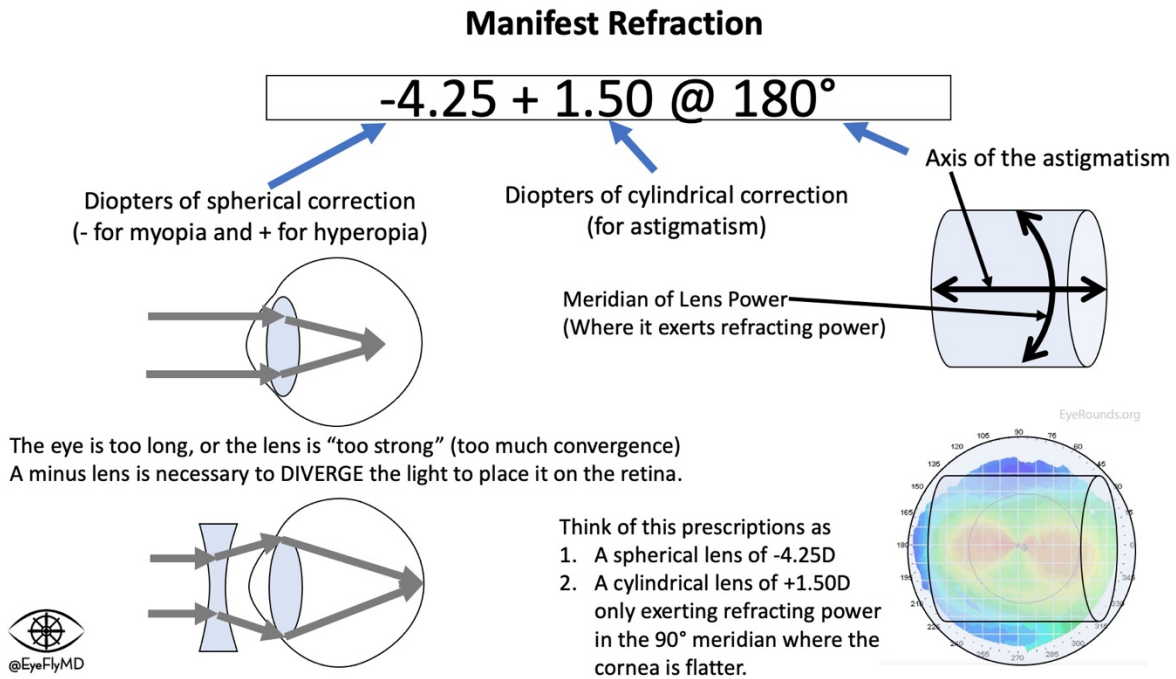


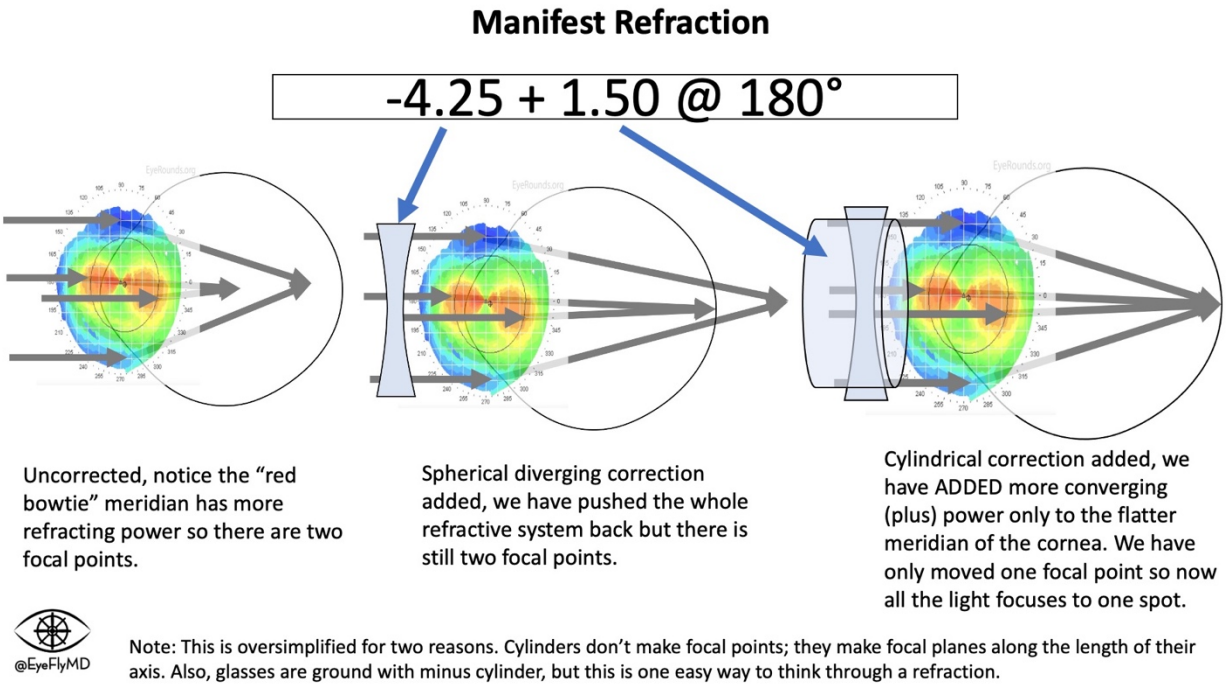


Manifest Refraction

This is when we see what lenses at what powers are needed to correct a patient's vision using a phoropter (the big thing with all the dials in the exam rooms). The first number is the sphere and the second number is the cylinder with its axis (to dictate the orientation of the cylinder). Notice for ophthalmologists the cylinder is always positive. Be aware that optometrists (and lensmakers) use negative cylinder because this is how glasses are actually constructed. This is also where the whole "with the rule" "against the rule" thing comes from. Because ophthalmology and optometry are working in opposite directions, the axes are going to be reversed as well. Using the simple "rule" language helps simplify things and facilitate communication. Let's ignore that entirely and live in the simple world of ophthalmology though.

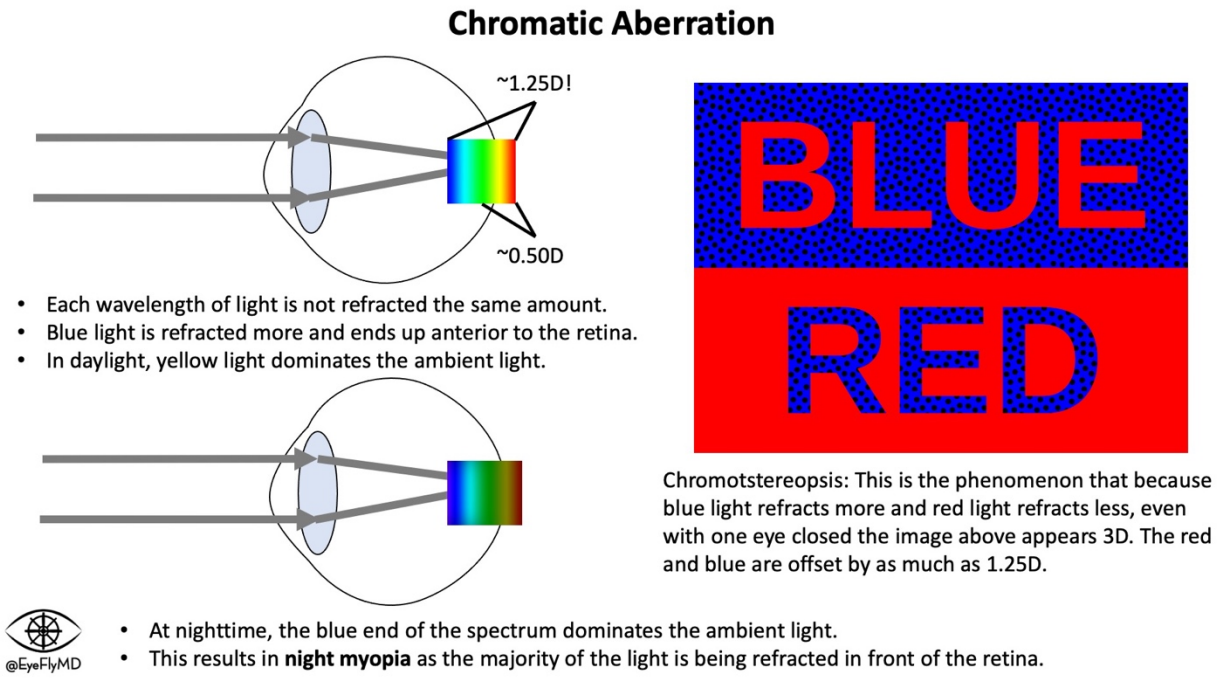
Even though it is entirely fictitious, it is helpful to think of glasses as a base spherical lens with a positive cylinder on top of it. Again, this is the opposite of reality but it's how ophthalmology refracts and operates. This means we can picture a cornea with astigmatism belonging to a hyperope. This astigmatic cornea has two different radii of curvature that require two different powers to place an image on the retina because one is "further back" than the other. Picture using a normal convex, plus, spherical lens to put the farther forward picture on the retina. We're halfway there but we need to individually move the other axis to put it on the retina as well. We need a little more converging power only on that axis so we can use a cylindrical lens to "help out" where the cornea is flatter and contributing less converging power.





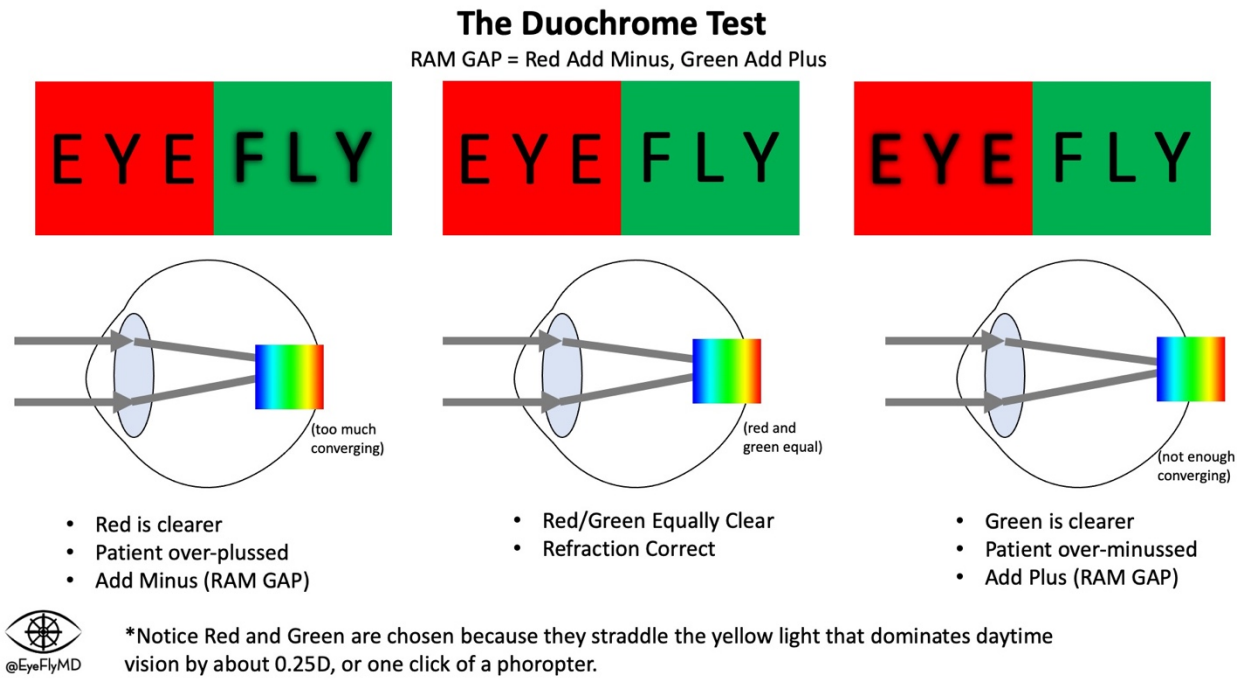
Chromatic Aberration

You also might notice eye charts that have a Red and Green split background or the Snellen chart in the exam rooms include a split red and green filter. What would this be used for? Well, first we have to understand the concept of **chromatic aberration**. Not all light experiences refraction the same way. Shorter wavelengths are slowed more by optically dense substances. The visible color spectrum ranges from ~400-700 nm and there is about a 1.25 D difference between the blue (~400 nm) and red (~700 nm) range of the spectrum. If you look at the BLUE/RED image below, even with one eye closed it will look 3D because the blue is landing slightly in the vitreous relative to the red and therefore the red looks closer and the blue looks farther away.

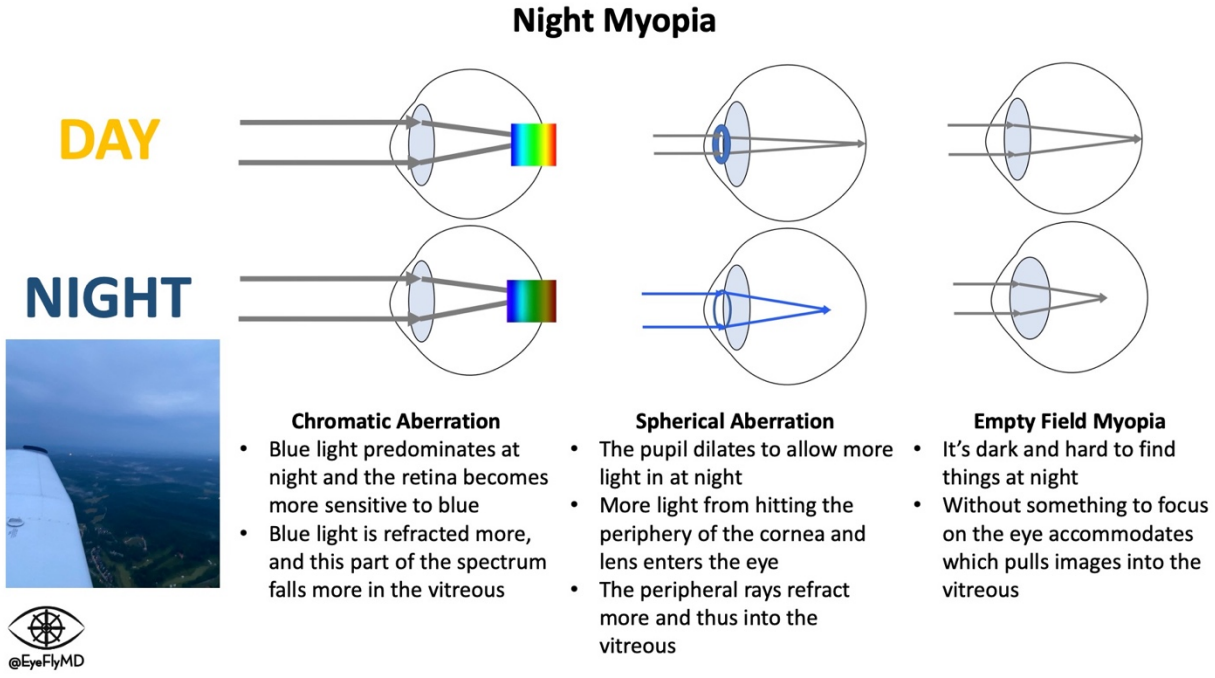


If you had to pick one color to fall on your retina the best which would you choose? During the day, yellow light tends to dominate the ambient spectrum so this is the color for which we try to optimize vision. How are we positive though that the yellow spectrum is the one falling on the retina? Remember earlier that people will generally prefer to go a little more minus than plus because (especially young) eyes can dial in their own accommodation and pull the image forward onto the retina. To avoid allowing someone to "eat up minus" we can perform the **Duochrome Test**. This involves overlying the Snellen (or other) chart over a red and green background. Why red and green though? Well, we just said yellow light is dominates the daytime atmosphere and red and green tend to straddle the yellow spectrum by about 0.25 D on either side.

The mnemonic **RAM GAP** can help us remember what to do depending on what the patient reports seeing. If the red side is clear but the green side is blurry then this means the red end of the spectrum is falling on the retina and there is too much converging power (plus) so we need to "add minus." In the opposite circumstance where the green side is clearer than the red side then there is too much diverging power (minus) so we need to "add plus." Hence, RAM GAP = Red Add Minus, Green Add Plus. The duochrome test is not effective is best corrected visual acuity is less than 20/30 in each eye as it then becomes difficult to distinguish the small difference caused by the chromatic aberration.

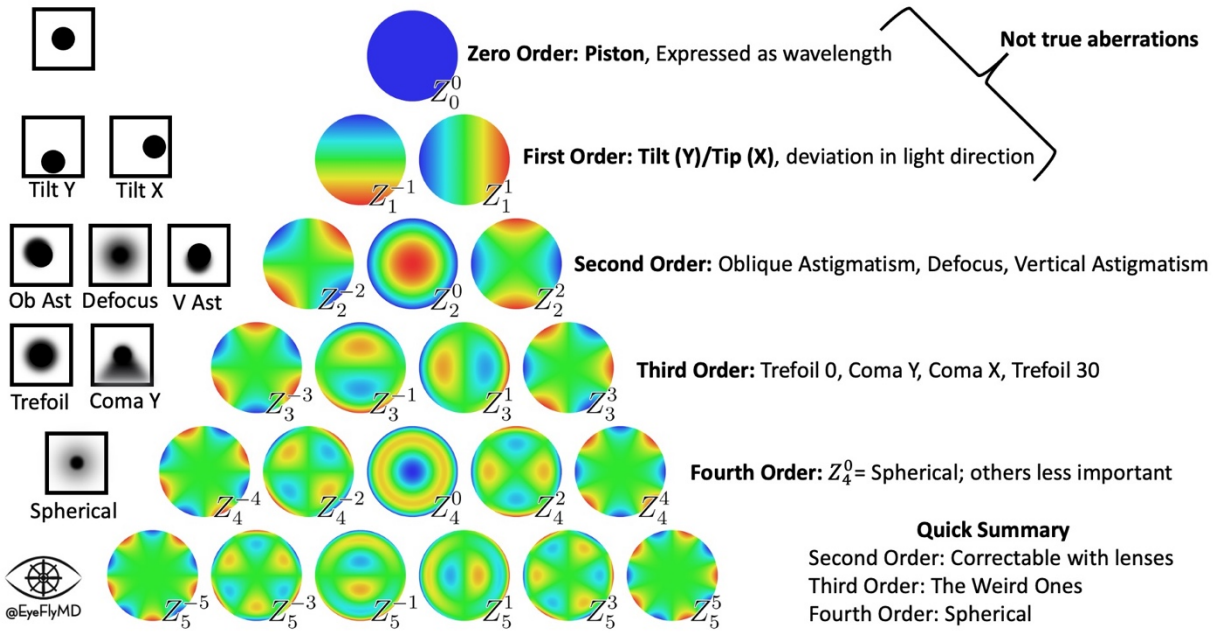


As mentioned above, this chromatic aberration is a cause of night myopia but it's not the only contributing factor. Another cause is spherical aberration referenced before in the discussion of the cornea. Lastly, the eye experiences something called "empty field myopia" where at night when the eye can't find something to focus on the ciliary body dials in accommodation and this also contributes to myopia.



You'll notice we've discussed chromatic and spherical aberration so far. There are other aberrations that degrade vision. These are expressed mathematically as Zernike polynomials. This is an advanced concept but being aware of this at least is very important for refractive surgery. A decentered LASIK surgery can cause coma for example.

Zernike Polynomials



Ophthalmic Testing

Cornea and Refractive

IOL Master

This is a test that measures the length and corneal refractive power of a patient's eye so the right lens can be inserted during cataract surgery.

Keratometry

This is most often automatically done as part of the IOL master, it measures the central corneal power using an image reflected off the tear film.

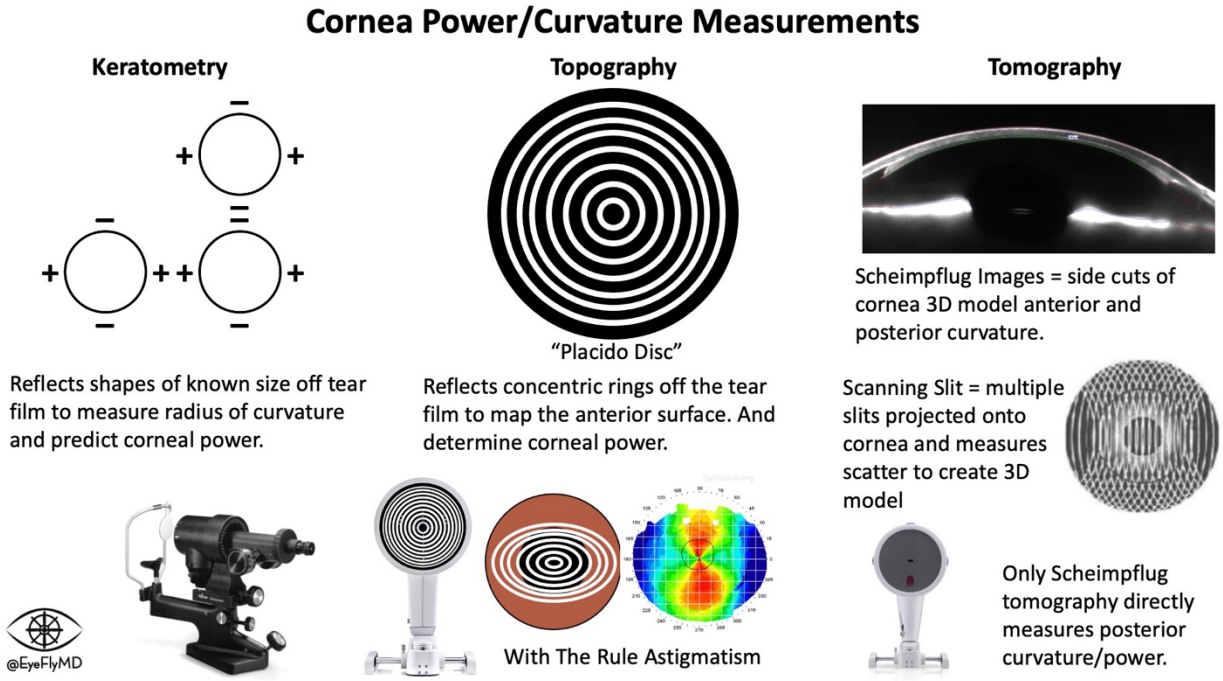
Corneal Topography

Imaging technique using concentric rings to map the anterior corneal curvature and provide information on astigmatism.

Corneal Tomography

Two varieties, the Oculus Pentacam uses Scheimpflug imaging to create a 3D rendering of the anterior and posterior corneal surface. With Scheimpflug imaging a measure of posterior corneal power is possible. The alternative is scanning slit technology which can only map the anterior cornea.

Note: Topography and Tomography are NOT the same thing



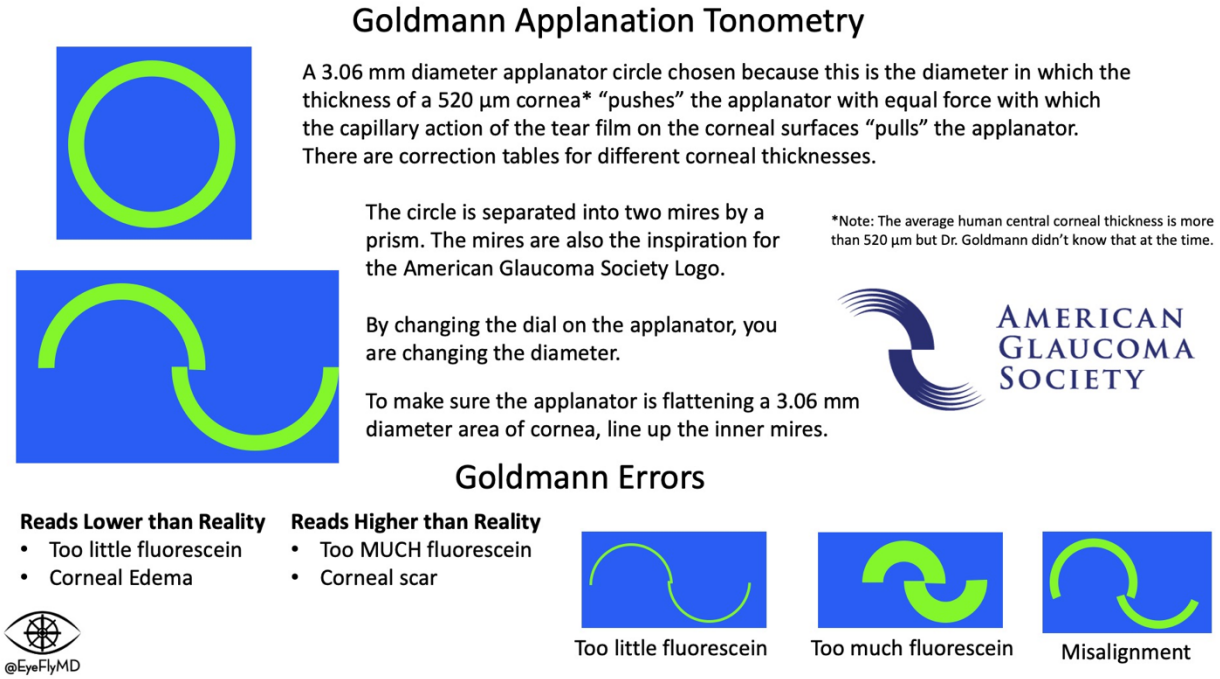
Confocal Microscopy

Measures the number or density of the cells that make up the endothelium and their shape. Polymegathism refers to cell size variability and pleomorphism refers to cell shape variation.

Glaucoma

Goldmann Applanation

IOP measurement using a tonometer with a 3.06mm disc that is considered the gold standard for IOP measurement.

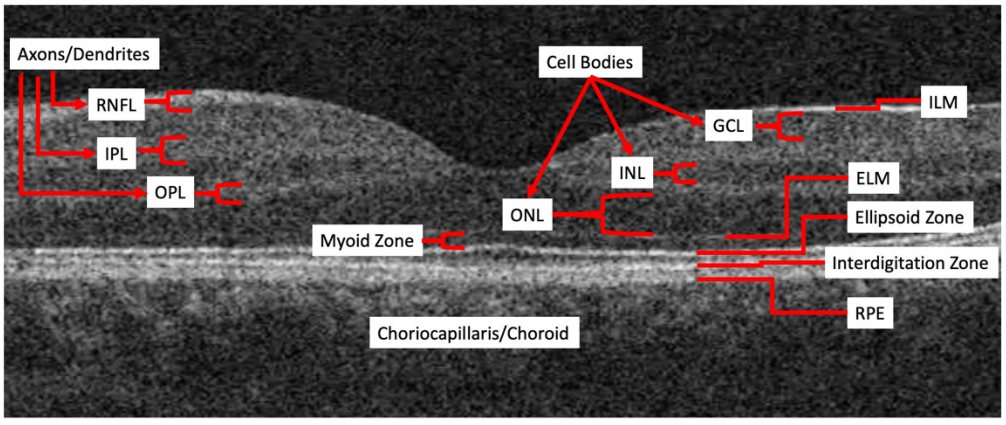


Optical Coherence Tomography (OCT)

This is a noninvasive method to image retinal structures in vivo. Cross-sectional images are produced that display the anatomic layers of the retina and measurements of retinal thickness. The optic nerve head OCT can provide information about the health of the RNFL. An OCT of the macula can provide information about retina architecture, thickness, or swelling. Recall this slide:

	OD	OS
Average RNFL Thickness	62 µm	58 µm
RNFL Symmetry	66%	
Rim Area	0.82 mm²	0.67 mm²
Disc Area	1.43 mm²	1.46 mm²
Average C/D Ratio	0.65	0.78
Vertical C/D Ratio	0.72	0.87
Cup Volume	0.184 mm³	0.375 mm³

Retina OCT



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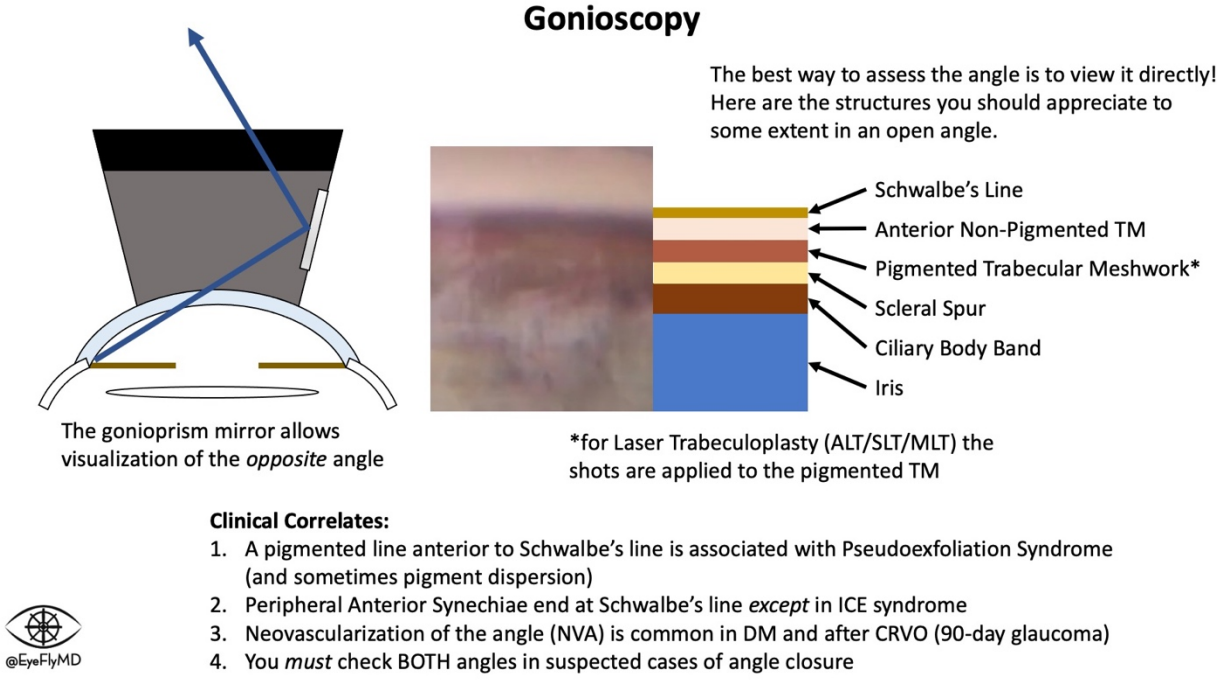
Visual Field Testing

The patient fixates on a target and a computer shines light according to an algorithm and the patient responds by clicking a button if they see the light. This provides information about peripheral vision which is what glaucoma mainly affects. A 24-2 Humphrey Visual Field (HVF) tests the central 48° of vision with points 6° apart. A 10-2 HVF tests the central 20° with points 2° apart.

Gonioscopy

One of the most important tools in glaucoma is gonioscopy. This is visualization of the angle using a lens. The most common types of gonioprisms use mirrors. The angle can convey a lot of information about glaucoma. It can be hard to see the all the structures even if the angle is wide open but with practice the major structures can be identified.



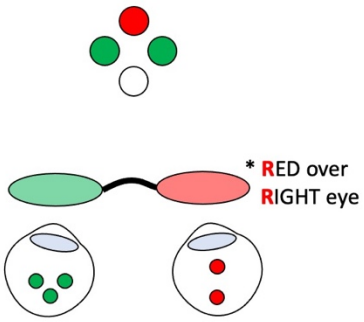
Pediatrics

Worth Four-Dot Test

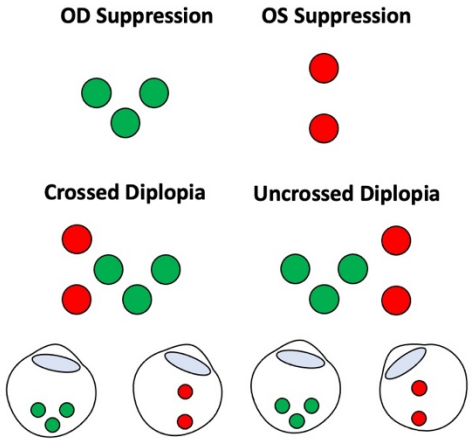
Either hanging in the back of every exam room, sitting in the drawers, or some combination thereof is the Worth Four-Dot Test. This is useful for examining fusion, suppression, and monofixation syndrome. It is composed of a diamond shape with a red dot on top, two green dots in the middle, and a white dot on the bottom. The patient wears red-green glasses, RED over RIGHT eye. The right eye then should see two dots (the top red one and the bottom white one seen as red) and the left eye should see three green dots (the two green and the bottom white one seen as green). The expected results of the tests are below.

The one that may require elaboration is monofixation syndrome. Monofixation syndrome manifests from a small-angle strabismus resulting in a small (< 3º) unilateral scotoma. Essentially, for some reason (small angle strabismus, anisometropia, macular pathology) a small central scotoma develops but binocular fusion is maintained through the peripheral fields which have a larger tolerance for image discrepancy than the high acuity fovea. The eyes will usually not look grossly misaligned. Picture the four dots up close, they are occupying a large portion of the visual field and it is likely the dots will all fall outside of the small central scotoma. Now picture the four dots far away. They are occupying a small central portion of the visual field and now they will fall within the central scotoma so there will be a discrepancy in what the patient sees with the four dots at near and at a distance.

Worth 4-Dot Test

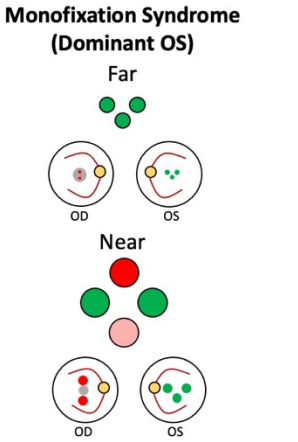


Normal Perception



EXOtropias produce crossed diplopia. The red dots are falling on the temporal retina OS which is the nasal field.

Think **X** for crossed diplopia.



At near, the dots all fall outside of the small central scotoma. At distance, the dots fall inside the scotoma and reveal the monofixation.

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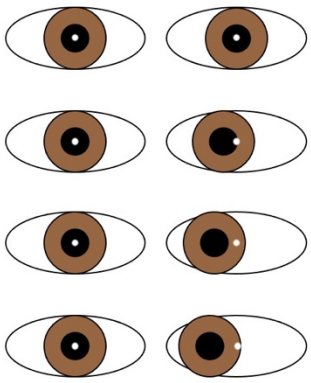
Hirschberg and Krimsky Test

Hirschberg Reflex Approximations



Equal and centered
= Ortho

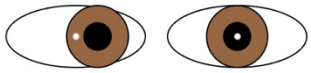
Pupil margin
~ 30 PD

Mid-Iris
~ 60 PD

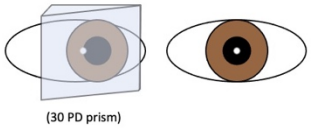
Outer Iris Margin
~ 90 PD

Krimsky Test

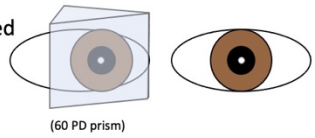
Determine Deviation with Hirschberg (RET in this case)



Use prisms to center the reflex on the pupil (apex toward deviation)



Amount of prism required to center reflex is the degree of deviation



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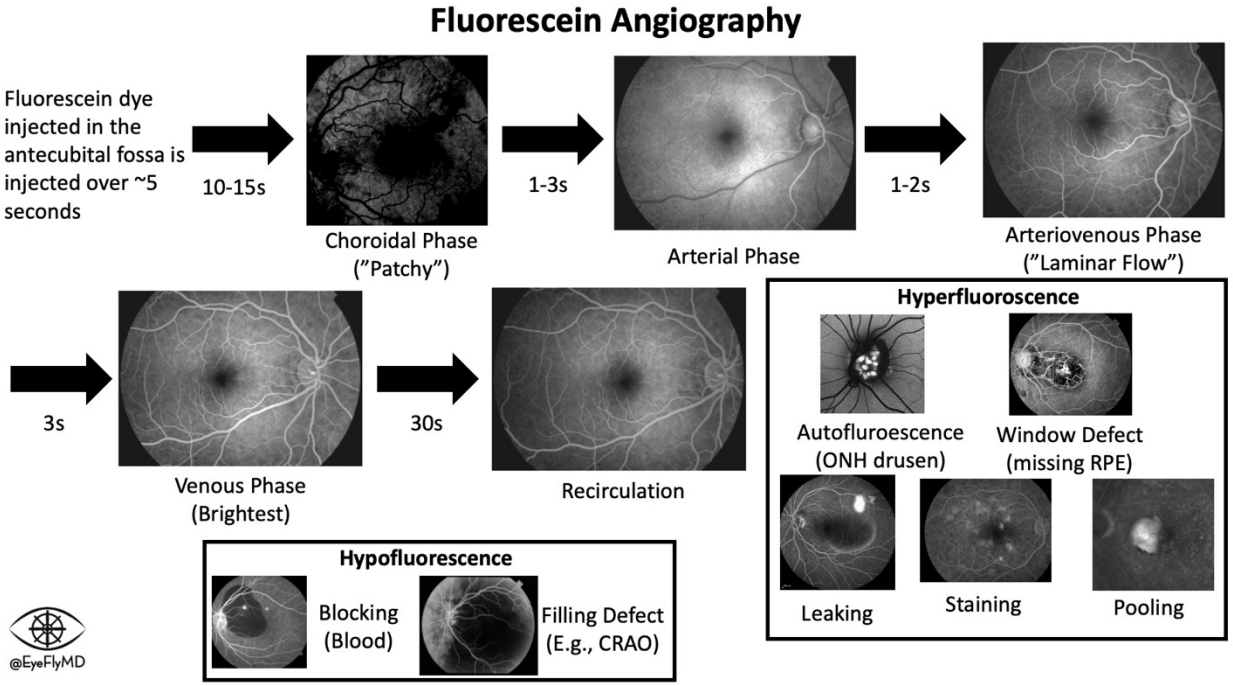
These approximations work well for strabismus cases with poor vision in the deviating eye where a prism alternate cover (PACT) test would be impossible.

Retina

Fluorescein Angiography (FA)

Fluorescein dye (beware green sweat and urine) is injected into the antecubital fossa and images of the retina are taken. A delay in "arm to choroid" time can imply vascular pathology like giant cell arteritis. The choroid fills in a patchy manner typically and then FA proceeds through predictable phases as outlined below. There are two basic abnormalities in FA, too bright (Hyperfluorescence) and too dark (Hypogluorescence). This can tell a LOT about the vasculature of the retina.



Fundus Photography

Machines like the Optos can provide high resolution wide-field retina photography.

A-Scan

Ultrasound biometry in 1 dimension, short for "Amplitude Scan", provides data on the length of the eye and internal reflectivity of suspected masses (e.g., melanomas characteristically have LOW internal reflectivity).

B-Scan

Ultrasonography biometry in 2 dimensions, short for "Brightness Scan", is a diagnostic test used to produce a two-dimensional, cross-sectional view of the eye and the orbit. This is particularly helpful to examine the retina when a clear view is otherwise impossible (e.g., dense cataracts, corneal opacification).

Common Pathology

Cornea/Refractive

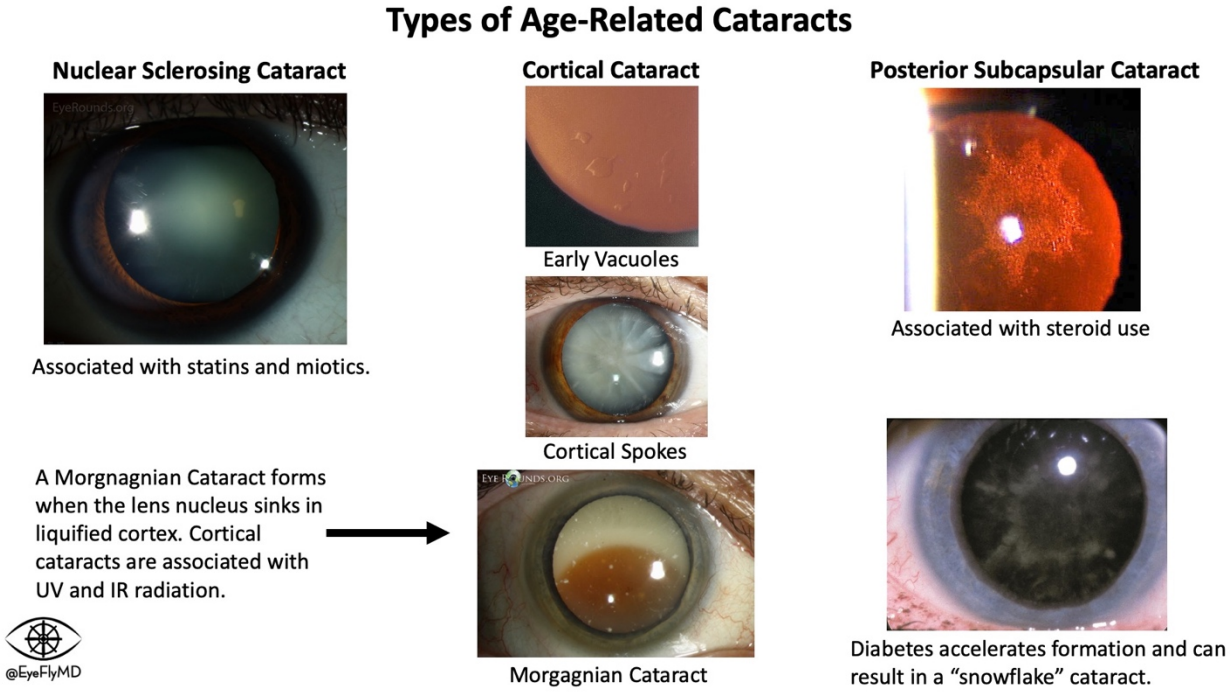
Cataract

A cataract is an opacification of the lens. Lenses may naturally have small opacifications but a clinically significant cataract is one that is suspected to interfere with a patient's visual acuity. Cataracts are also described based on their appearance.

A nuclear cataract affects the nucleus of the lens and presents as a hazy opacity that likely affects vision at night.

A cortical cataract affects the lens cortex and presents first as peripheral vacuoles, then clefts, and eventually the entire cortex can liquify and the nucleus can sink within it (Morgagnian Cataract). This is likely to cause glare and halos for patients.

A posterior subcapsular cataract is associated with steroid use and diabetes and presents as a focal collection of material toward the posterior of the lens. Patients are particularly affected in bright light as the pupil constricts and the available light must travel through the opacity.



Dry Eye Disease

This is what it sounds like and is extremely common. Dry eyes causing burning and tearing. The signs are a reduced tear lake (the fluid resting at the lower lid margin) or increased tear break-up time (TBUT). TBUT is assessed using fluorescein and timing how long the tear film breaks up. Anything <10 seconds is too fast and is abnormal. It is usually caused by the ratio of ingredients in the tear film being abnormal. Aqueous Deficiency is when adequate tears are not being produced. Evaporative Dry Eye is often caused by meibomian gland dysfunction referenced above. If the ingredients are right, the tears will evaporate too fast.

Dystrophies

There are many corneal dystrophies, and they can be difficult to keep straight. They also are tough to identify in clinic. The high-yield information for the dystrophies is presented below. You'll encounter some (Fuch's, Epithelial Basement Membrane) with much greater frequency than others (Congenital Hereditary Endothelial Dystrophy). See below for a high yield reference to those mentioned in the BCSC.

Corneal Dystrophies Review

Category	Dystrophy	Inheritance	Synopsis
Epithelial and Subepithelial Dystrophies	Epithelial Basement Membrane Dystrophy	Sporadic	Maps/dots/fingerprints, distorted epi; REE
	Meesmann Epithelial Corneal Dystrophy	AD	Very young; diffuse microcyts; "peculiar substance in epi; REE
	Lisch Epithelial Corneal Dystrophy	XLD	Childhood; feathery opacities; NO REE
	Gelatinous Droplike Corneal Dystrophy	AR	Childhood; amyloid; Band K or "mulberry" (nodules) presentation
	Epithelial Recurrent Erosion Dystrophies	AD	Not covered in BCS but listed in the table
	Subepithelial Mucinous Corneal Dystrophy	AD	Not covered in BCS but listed in the table
Epithelial-Stromal *TGFBI* (Transforming Growth Factor Beta 1) Dystrophies	Reis-Bückler Corneal Dystrophy	AD (5q31)	Childhood; disrupted Bowman, Geographic opacity, rod fibers
	Thiel-Behnke Corneal Dystrophy	AD (5q31)	Childhood; disrupted Bowman, Honeycomb opacity, curly fibers
	Lattice Dystrophy, Type 1	AD	Childhood; lattice opacities subepithelium/stroma
	Lattice Dystrophy Variant (III, IIIA, I/IIIA, IV)	AD	Childhood; lattice opacities subepithelium/stroma
	Granular Type 1	AD	Young; granules centrally, REE; hyaline; rod fibers
	Granular Type 2	AD	Adult; starry granules; EE; hyaline; rod fibers
Stromal Dystrophies	Macular Corneal Dystrophy	AR	Childhood; opacities with haze in between; rare REE
	Schnyder Corneal Dystrophy	AD	Very young; lipid disorder; central opacity ten arcus; Oil Red O
	Congenital Stromal Corneal Dystrophy	AD	Birth; diffuse haze; stromal thickening; lamellae separation
	Fleck Corneal Dystrophy	AD	Very young; dandruff like opacities in stroma only
	Posterior Amorphous Corneal Dystrophy	AD	Infancy; deep stroma; thin and flat cornea (hyperopic)
	Pre-Descemet Corneal Dystrophy	Unknown	Adults; fine opacities just anterior to Descemet's
Endothelial Dystrophies	Fuchs Endothelial Corneal Dystrophy	Unknown	Adults; Guttata; "Beaten Bronze"; polymegathism; polymorphism
	Posterior Polymorphous Corneal Dystrophy	AD	Teens; vesicles, snail tracks, geographic; multiple endo layers
	Congenital Hereditary Endothelial Dystrophy	AR	Birth; 2-3x thickened cornea; milky/hazy; thick Descemet's

The Big Ones
Marilyn
Meesman
Monroe
Mucopolysaccharide
Always
Alcian Blue

Gets
Granular
Her
Hyalin
Man
Masson trichrome

In

L
Lattice
A
Amyloid
County
Congo Red

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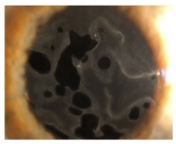
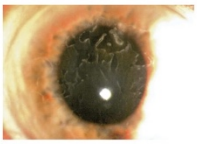
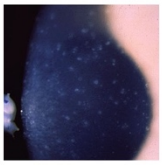
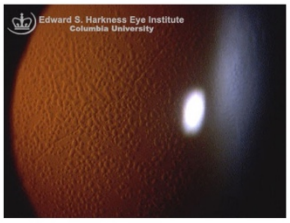
Epithelial and Subepithelial Dystrophies

Epithelial Basement Membrane Dystrophy
Aka Map-Dot-Fingerprint Dystrophy
Aka Cogan Microcystic Dystrophy
Aka Anterior Basement Membrane Dystrophy

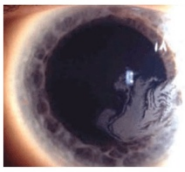
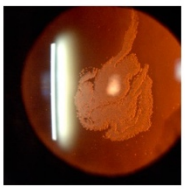
- Maps/dots/fingerprints
- Distorted Epithelium
- Recurrent Epithelial Erosions (REE)

Meesmann Epithelial Corneal Dystrophy

- Very young
- Diffuse microcysts
- "peculiar substance" in epithelium
- Recurrent Epithelial Erosions (REE)

Lisch Epithelial Corneal Dystrophy

- Childhood
- Feathery Opacities
- NO REE

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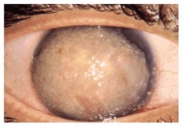
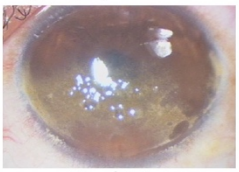
Gelatinous Droplike Corneal Dystrophy


Mulberry Type

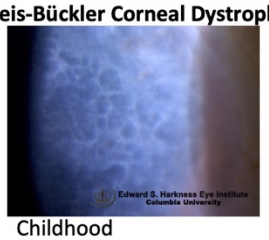

Band K type

- Childhood
- Amyloid
- Band K or "mulberry" (nodules) presentation

Epithelial Stromal Dystrophies

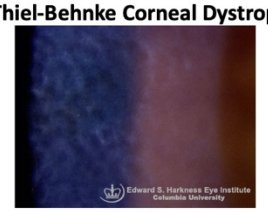
Primarily Bowman's Involving

Reis-Bückler Corneal Dystrophy



- Childhood
- Geographic opacities
- Rod shaped fibers in Bowman

Thiel-Behnke Corneal Dystrophy

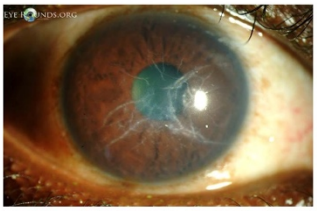


- Childhood
- Honeycomb opacities
- Curly fibers in Bowman

Lattice Types

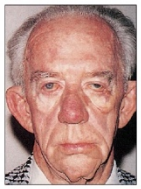
Lattice Dystrophy Type 1

Lattice Dystrophy, Variants



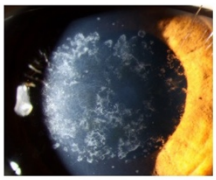
- Childhood
- Amyloid
- REE

Remember Gelsolin Type Lattice is associated with Meretoja Syndrome



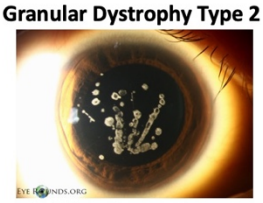
Granular Types

Granular Dystrophy Type 1



- Childhood
- Hyaline
- Rod Shaped Fibers

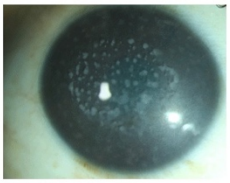
Granular Dystrophy Type 2



- Childhood
- Amyloid AND Hyaline
- Rod Shaped Fibers

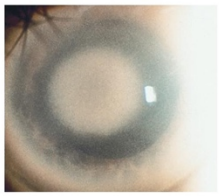
Stromal Corneal Dystrophies

Macular Corneal Dystrophy



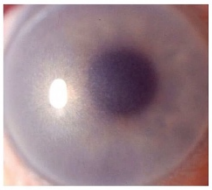
- Childhood
- Opacities with haze in between
- Rare REE

Schnyder Corneal Dystrophy



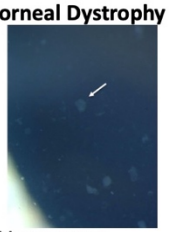
- Very young
- Lipid disorder
- Central opacity then arcus
- Oil Red O

Congenital Stromal Corneal Dystrophy



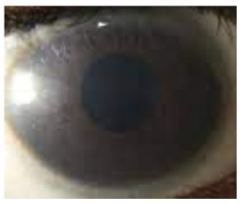
- Birth
- Diffuse haze
- Stromal thickening
- Lamellae separation

Fleck Corneal Dystrophy



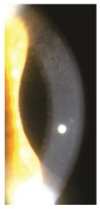
- Very young
- Dandruff stromal opacities

Posterior Amorphous Corneal Dystrophy



- Infancy
- Deep stroma
- Thin and flat cornea (hyperopic)

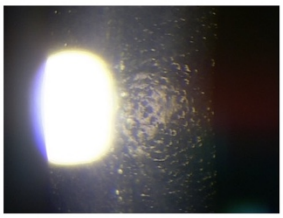
Pre-Descemet Corneal Dystrophy

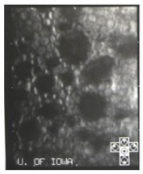


- Adults
- Fine opacities just anterior to Descemet's

Endothelial Corneal Dystrophies

Fuchs Endothelial Corneal Dystrophy





- Adults
- Guttata
- "Beaten Bronze"
- Polymegathism; polymorphism

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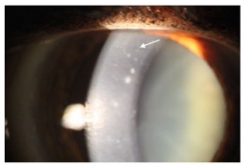
Posterior Polymorphous Corneal Dystrophy

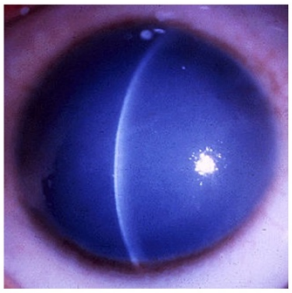
Snail Tracks



Vesicles

- Teens
- Vesicles
- Snail tracks
- Geographic
- Multiple endothelial layers

Congenital Hereditary Endothelial Dystrophy



- Birth
- 2-3x thickened cornea
- Milky/hazy
- Thick Descemet's (use this to help distinguish from CHSD)

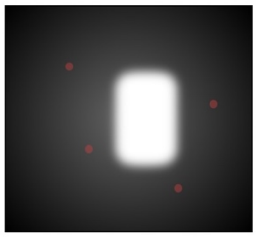
Hyphema

Hyphema is the term for blood in the anterior chamber. This usually follows blunt trauma of some kind. A microhyphema is when there are blood cells floating around the anterior chamber and a layered hyphema is when pooled or clotted blood is visible. The main priority of managing a hyphema is preventing re-bleed by enforcing bed rest and limiting straining or Valsalva. If the hyphema re-bleeds the chances of complications related to IOP are increased. Among the other priorities are controlling IOP, limiting inflammation that may cause Peripheral Anterior Synechiae (PAS, connections between the peripheral iris and cornea than can obstruct the angle and cause elevated IOP), and preventing corneal bloodstaining. Corneal bloodstaining occurs when keratocytes take up hemosiderin. This is especially concerning for younger children as it can cause amblyopia by opacifying the cornea. The RBCs in the AC need to be removed surgically at first sign of bloodstaining.

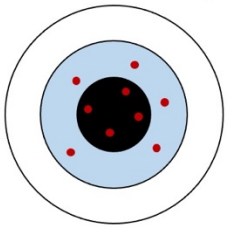
Hyphema

Considerations

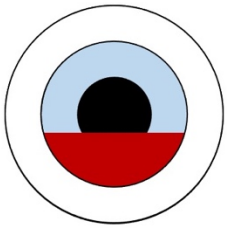
1. Prevent Re-Bleed (most likely in first 3-7 days)
   - Avoid straining and aspirin
2. Prevent high IOP
   - Aqueous Suppressants (α, β, Cs)
   - Hyperosmotic Agents (e.g., mannitol)
3. Prevent Inflammation
   - Steroids and cycloplegia
4. Prevent Corneal Blood Staining
   - AC washout at first sign



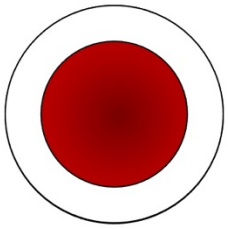
Check for hyphema at the slit lamp by using a small, square beam at an angle aimed at the pupil. Turn the lights off and look for cells floating around in the anterior chamber. It will look like dust in the beam of a movie projector.



Microhyphema
Blood cells visible in the anterior chamber



Layered Hyphema
Clear layering of blood



8-Ball Hyphema
Blood filling the entire volume of the anterior chamber

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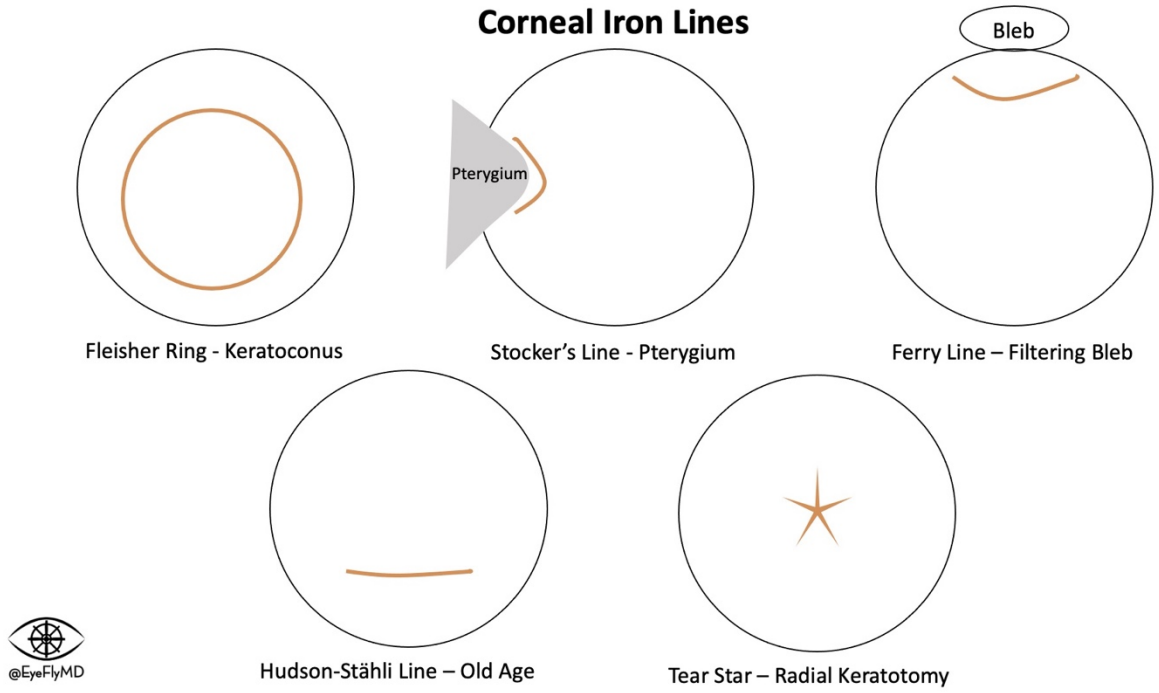
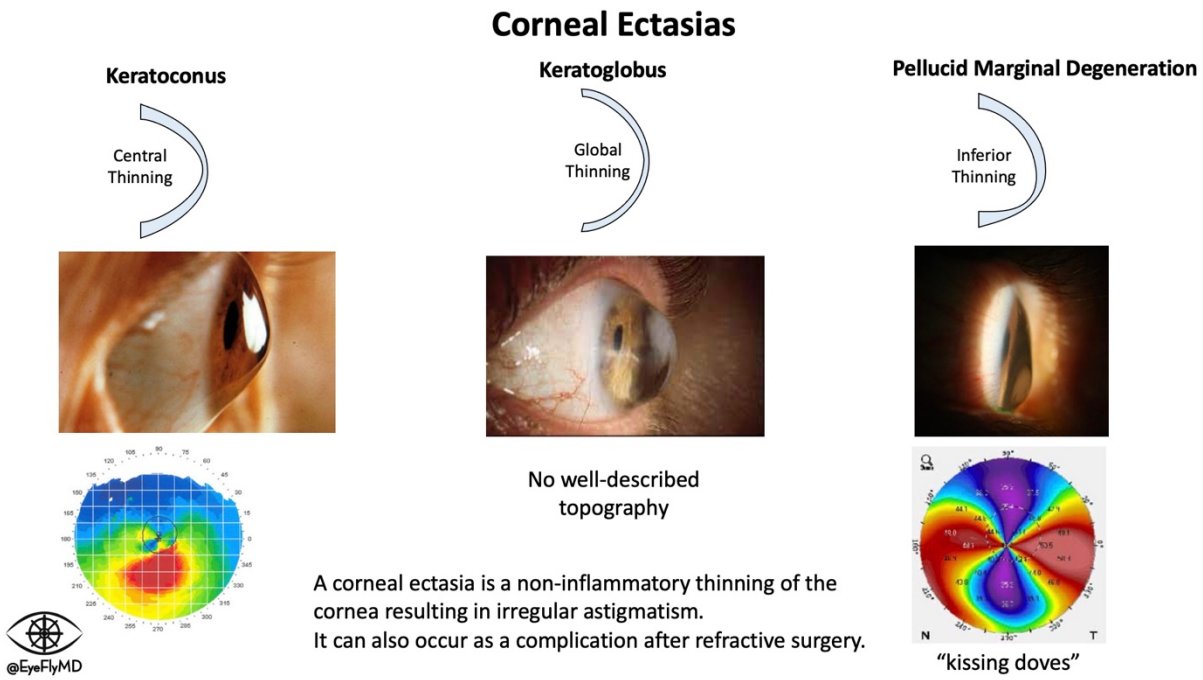
Iron Lines

Many conditions (including just having too many birthdays) can cause iron lines. These are deposits of iron in the corneal epithelium (calcium deposition in Band Keratopathy occurs in Bowman Layer and copper deposition in Wilson Disease is in Descemet's Membrane by the way. Don't confuse a the Fleisher iron ring at the base of keratoconus with the Kayser-Fleischer copper ring of Wilson Disease. Helpful memory aids for the other iron lines are to picture a pterygium STOCKing the cornea (Stocker's Line) and a FERRY floating in a bleb (Ferry Line).



Keratoconus/Corneal Ectasias

A corneal ectasia is a noninflammatory thinning and warpage of the cornea. The main ectasias are keratoconus (cone-shaped warpage of the cornea), Pellucid Marginal Degeneration (area of protrusion above an area of corneal thinning), keratoconus (diffuse protrusion and thinning of the cornea), and ectasias induced by refractive surgery (e.g., after LASIK). If a patient had LASIK or other refractive surgery in the past and is now presenting with worsening visual acuity not correctable with refraction, always consider a topography/tomography to assess for ectasia. A theory is that the majority of the structural integrity of the cornea is in the anterior 1/3 (where LASIK is making most of the changes). SMILE alters the cornea more posteriorly so it was thought the ectasia risk would be lower but new data is showing conflicting reports of this. Stay tuned.

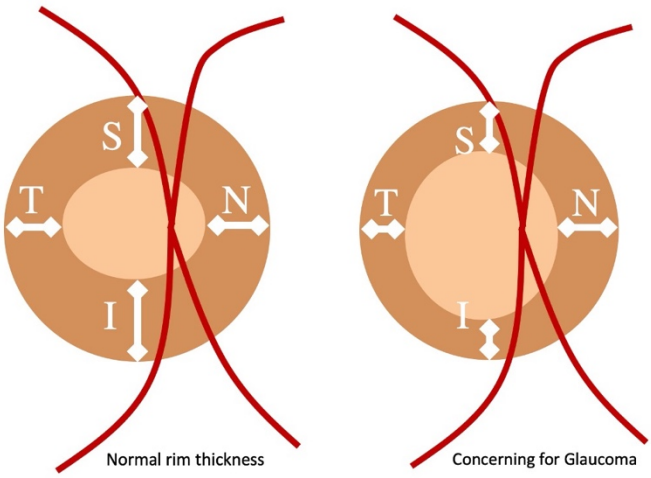


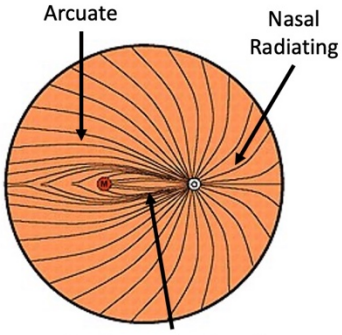
Glaucoma

Glaucoma is a group of progressive optic neuropathies the presents with characteristic optic nerve changes and visual field loss. It is NOT simply elevated intraocular pressure (IOP). Don't let someone trick you into saying "glaucoma is high intraocular pressure." High IOP is called "Ocular Hypertension" without the optic nerve and/or visual field changes that are characteristic of glaucoma or sometimes "Glaucoma Suspect" depending on risk factors. IOP is the most important and only modifiable risk factor for glaucoma and this is why all therapies for glaucoma target reducing IOP in some capacity. The takeaway is you MUST at least look at the optic nerve before diagnosing glaucoma.

The characteristic optic nerve head changes involving cupping (disc excavation). The neuroretinal rim is thickest to thinnest according to the ISNT rule (Inferior > Superior > Nasal > Temporal). Deviations to this rule (e.g. a thin inferior rim relative to temporal) may indicate glaucomotous change. For unknown reasons, glaucoma tends to affect the superior and inferior portion of the disc (so vertical cup to disc ratio is the on we're interested, specifically if it's > 0.3).

The ISNT Rule





Papilomacular Bundle

The axons from a large portion of the retina enter the optic nerve head superiorly and inferiorly which is why the rim is thicker.

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Deviations to the rule may raise concern for glaucoma. This is also why *vertical* cup to disc is the more important measure.

Why glaucoma preferentially prefers these fibers is not yet known.

When facing glaucoma, first consider if the angle is closed or open, which can only be determined by gonioscopy. If the angle is open, consider if the IOP is high (>22) or normal (Normal Tension Glaucoma). If it is high, make sure you rule out the many forms of secondary glaucoma before landing on the very commondiagnosis of "Primary Open Angle Glaucoma."

Below is a flowchart of one way to think about glaucoma. There are many types of glaucoma and while this doesn't cover nearly all of them it's a way to start organizing them in your approach. "Ocular Hypertension" is elevated IOP in the absence of nerve damage or field changes. "Glaucoma Suspect" is a term used for funny looking nerves but no field changes.

The Glaucoma Family Tree

Angle Status

Closed — ALWAYS GONIO — **Open**

Cause

- **Primary**
  - Acute
  - Subacute
  - Chronic
  - Plateau Iris
- **Pupillary Block**
  - **Present**
    - **Len-Induced**
      - Phacomorphic
      - Ectopia lentis
    - **Aphakic**
      - Vitreous face
      - AC IOL
  - **Without**
    - Choroidal
    - Aqueous misdirection
    - NVG/ICE

IOP

- **High >21**
  - **Cause**
    - **None**
      - **Primary Open Angle Glaucoma** (must rule out other causes to call it POAG)
    - **Identified**
      - **Secondary Glaucoma**
        - Lens-induced
        - Inflammatory
          - Posner-Schlossman
          - Fuchs Uveitis Syndrome
        - Trauma
          - Cyclodialysis, Recession
          - Ghost Cell, Hemolytic
        - Pigmentary
        - Pseudoexfoliation
        - Schwartz
        - Elevated EVS
          - CCF
          - SVS
          - AVM
        - Tumor
        - Steroids
- **Normal ≤21**
  - **Normal Tension Glaucoma**

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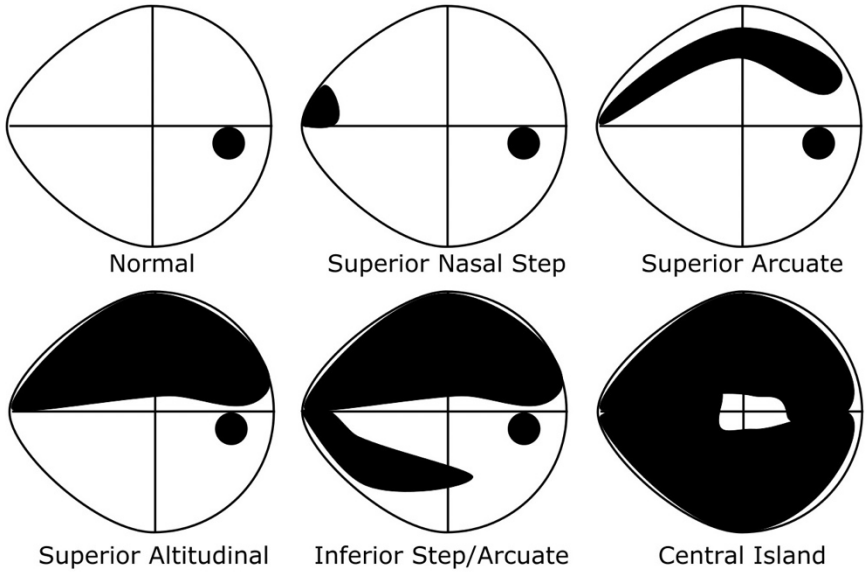
Stages from the AGS:

- Early – HVF "outside normal limits"
- Moderate – < 15 dB within 5° of fixation: Only 1 hemifield
- Severe – <15 dB within 5° of fixation: Both hemifields,

Glaucoma feature progressive and predictable visual field loss as highlighted below. Remember, if the visual field is affected, it actually means the "damage is done" in that the optic nerve is already damaged and this is not reversible. The area of the visual field that is characteristically affected is called Bjerrum's Area.

Glaucomatous Visual Field Progression

The inferotemporal area and the central island are usually the last parts of the visual field to be affected by glaucoma.



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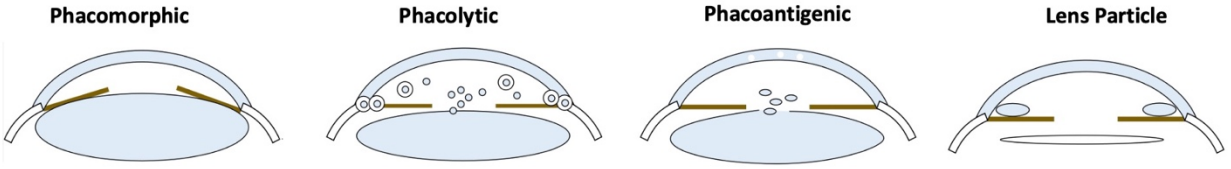
Among the many types glaucoma are the several varieties of secondary glaucoma caused by the lens (these are easy for an attending to ask about). A cataractous lens can become intumescent (swollen) and obstruct the pupil and prevent normal aqueous flow (Phacomorphic Glaucoma), a mature cataract can release denatured lens proteins that are consumed by macrophages that can clog the trabecular meshwork (Phacolytic Glaucoma), the lens capsule can become interrupted and the normal lens protein can lose it's immune privledge and incite a granulomatous reaction (Phacoantigenic Glaucoma), or particles from the lens remaining in the anterior chamber after surgery or trauma can simply obstruct the trabecula meshwork (Lens Particle Glaucoma).

Lens-Induced Glaucoma



Phacomorphic

Intumescent (swollen) cataract pushes the iris narrowing the angle or apposes the pupil margin causing pupillary block.

Phacolytic

A mature cataract leaks **denatured** lens proteins. Macrophages phagocytose these proteins and clog the Trabecular Meshwork.

Phacoantigenic

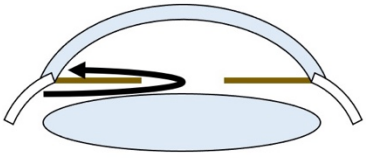
Normal lens proteins leak into the AC after trauma or capsule disruption. A granulomatous reaction to these **normal** proteins ensues.

Lens Particle

Pieces of lens particles (e.g., after cataract surgery) physically obstruct the angle.

Pupillary Block = The anterior lens comes into contact with the pupillary margin and prevents the normal flow of aqueous

Aqueous Humor is produced by the non-pigmented epithelium of the pars plicata of the ciliary body



The majority flows from the posterior chamber to the anterior chamber through the pupil and then through the trabecular meshwork in the angle.

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Angle Closure Glaucoma

This is one of the so-called "true ophthalmic emergencies." Recall from the anatomy above the angle is where the trabecular meshwork resides. If for some reason the iris makes contact with the peripheral cornea and prevents aqueous from draining through the trabecular meshwork then the pressure can rapidly rise. Pressures of 60-80 mmHg are common and can definitely cause fast and permanent optic nerve damage. There are different types of angle closure of course but they are managed by attempting to break the acute attack with a Laser Peripheral Iridotomy (LPI) and pilocarpine (pilocarpine induces miosis to prepare for LPI but also opens the TM), reducing IOP with medications, and examining the fellow eye and prophylactically placing an LPI if it is narrow.

Normal-Tension Glaucoma (Low-Tension Glaucoma)

There is some debate if this should be treated as a separate entity from Primary Open Angle Glaucoma but there are some differences. It is characterized by optic nerve damage and visual field changes without an "elevated IOP". What is a normal IOP anyway? About 10-21. Population studies have shown the mean IOP to be ~ 15.5 with a SD of 2.6 and a positive skew (remember from stats class that's a roughly normal distribution with a tail on the *high* side). Two SDs in either direction give us our "normal range" but remember: "normal" doesn't mean much when the visual field is constricting.

Plastics

Blepharitis

Inflammation or dysfunction of the meibomian glands can result in chronic eyelid swelling, crusting, and/or dry eyes. The meibum produced by the glands can experience an alteration of composition which can clog the glands (and thus interfere with the tear film) and create an environment for bacteria to flourish. Good lid hygiene (baby shampoo or dedicated lid scrubs) and warm compresses can help liquify the meibum and restore the glands to a more natural environment. Artificial Tears can help with dry eye symptoms but be careful using them more than 4 times a day if they have preservatives. You may astutely notice a patient prescribed an antibiotic like doxycyline for blepharitis or dry eyes. This must wipe out the infection and turn things normal again, right? NO. The antibiotic part is minimal, doxycycline has anti-inflammatory properties that makes it useful for blepharitis

Chalazion/Hordeolum ("Stye")

- A Hordeolum is an active infection of the Meibomian gland in the upper or lower lid. It is usually painful and usually presents with a distinct red bump somewhere on the eyelid.
- A Chalazion refers to noninfectious obstruction of the sebaceous glands (e.g., meibomian glands) and there is usually less pain and redness, but a bump is usually obvious.

Dermatochalasis – This refers to the excess skin of the upper eyelid often seen in older adults. It's treated with a blepharoplasty

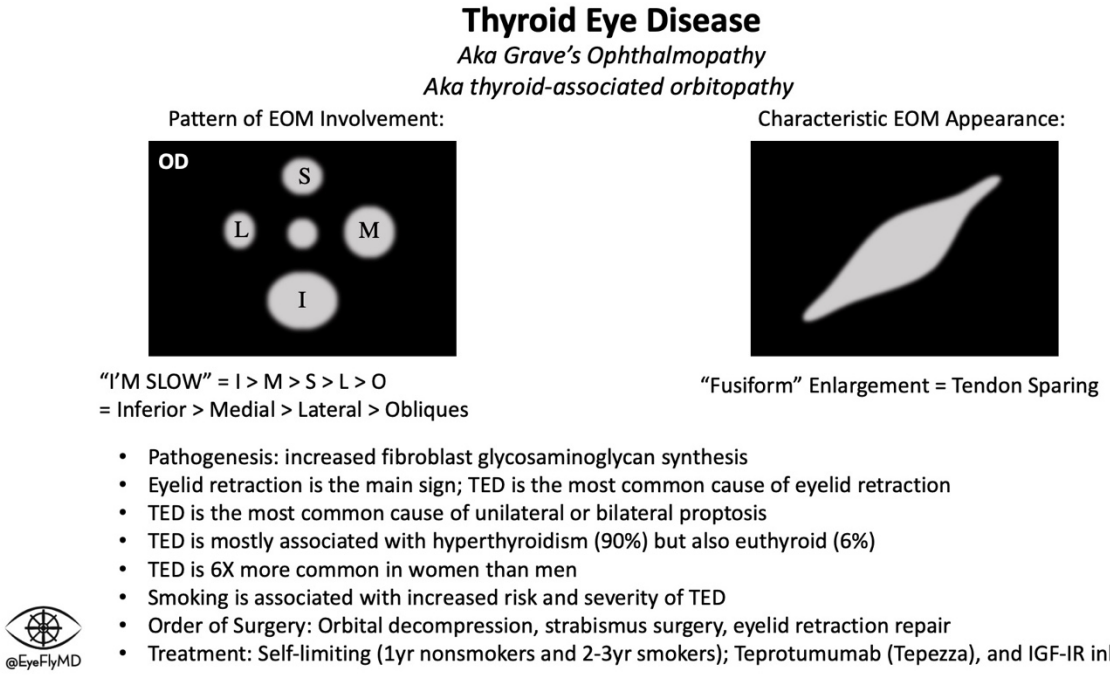
Epiphora – This refers to excessive tearing and can be a symptom of many disease. In very young children be afraid of congenital glaucoma for which the triad is epiphora, blepharospasm, and photophobia. In older kids a nasal-lacrimal duct obstruction (NLDO) can cause tears to run down the face since they can't drain. In adults dry eye syndrome (DES) can paradoxically cause tearing. In older adults changes in the eyelid like ectropion (outward turning of the eyelid margin) or entropion (inward turning of the eyelid margin) can cause tearing.

Ptosis – Technically called "blepharoptosis", this refers to a narrowing of the palpebral fissure (open space between eyelids). It is essentially droopiness of the upper or lower eyelid.

- Aponeurotic ptosis: related to the disinsertion or dehiscence of the levator muscle in the eyelid secondary to age or genetic predisposition
- Mechanical ptosis: related to the weighing down of the lid secondary to extra skin or a mass

Thyroid Eye Disease (TED)

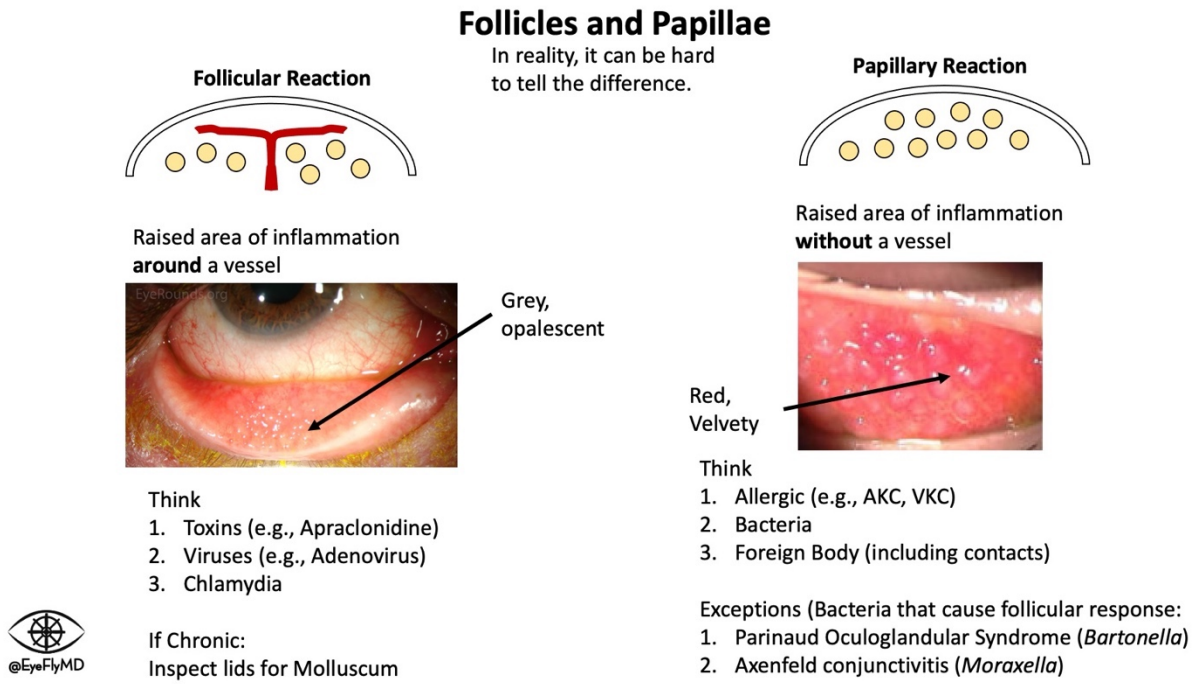
This is the most common cause of eyelid retraction in adults and is usually associated with hyperthyroidism (Graves' Disease) or much less commonly hypothyroidism (e.g., Hashimoto Thyroiditis). It is strongly associated with smoking and being female. The thyroid antibodies stimulate fibroblasts to secrete glycosaminoglycans and this results in proptosis. The muscles are usually enlarged but it spares the tendons, this is called "fusiform." The order of muscles that TED usually affects follows the mnemonic IM SLO: Inferior > medial > superior > lateral > obliques. Steroids can help with the proptosis and there's a new drug on the market called Tepezza (teprotumumab) which target IGF-R1 receptors and can reduce the symptoms of TED (diplopia, pain, swelling, etc.).



Trichiasis – Trichiasis occurs when a misdirected lashes originates from an otherwise normal eyelid and can rub on the cornea causing discomfort. Be careful here, if the eyelid margin is turned in and the lashes are rubbing on the cornea it's technically entropion.

Conjunctivitis

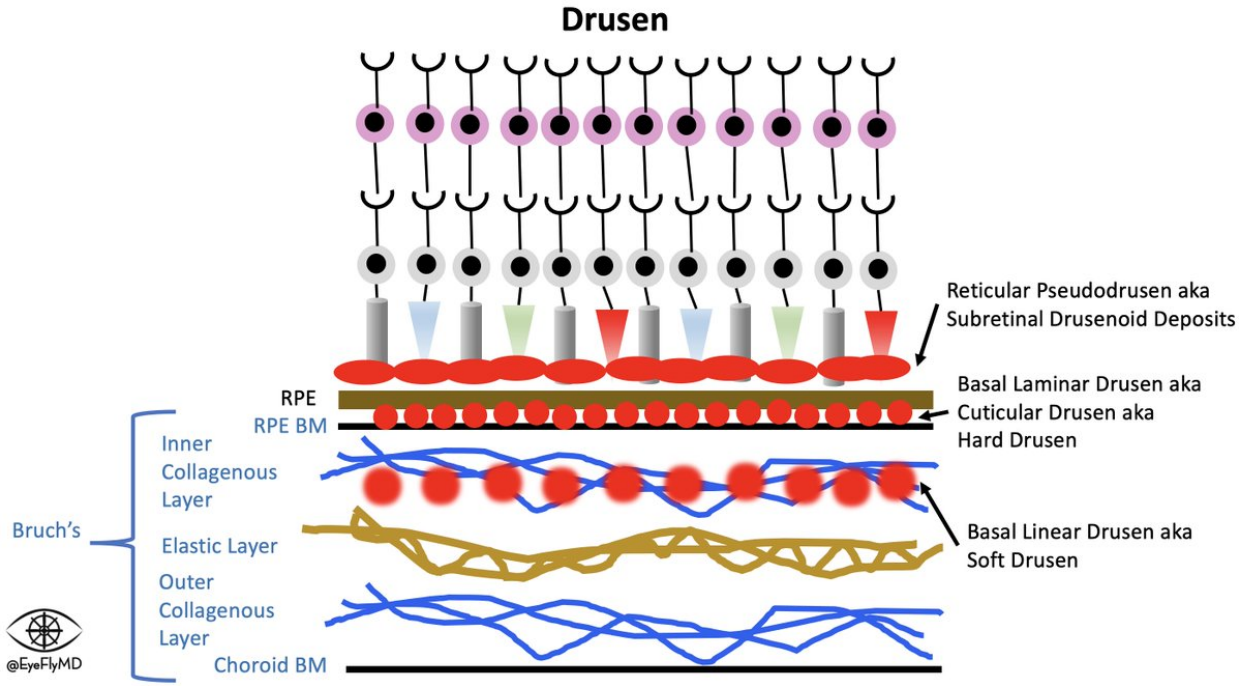
This is a term for inflammation of the conjunctiva. You can usually divide this into follicular or papillary conjunctivitis, and they imply different causes. A follicular reaction is typically caused by Toxins, Viruses, and Chlamydia. Follicular reactions usually involve the lower eyelid. A papillary reaction is usually caused by allergies, bacteria, or foreign bodies (like contact lenses) and typically involves the upper lid. Follicles are caused by inflammation associated with a vessel whereas papillae are caused by areas of inflammation without a vessel.



Retina and Vitreous

Age-Related Macular Degeneration (AMD)

This refers to the deterioration of the macula and the buildup of deposits of proteins and lipids called Drusen. It is also associated with smoking and reduction in central visual acuity. The "Dry" form features the drusen and the "Wet" form implies neovascularization and these new vessels bleed and leak into the retina. There are several types of drusen, typically large and soft drusen are bad news. For the wet form, the usual anti-VEGF injections can be used. You will also hear about the AREDS2 formulation which is a vitamin/mineral cocktail of Vitamin C, Vitamin E, Zinc, Copper, Xeanthin, and Lutein. What happened to AREDS1? It had beta-carotene which was associated with increased risk of lung cancer in smokers.



Diabetic Retinopathy

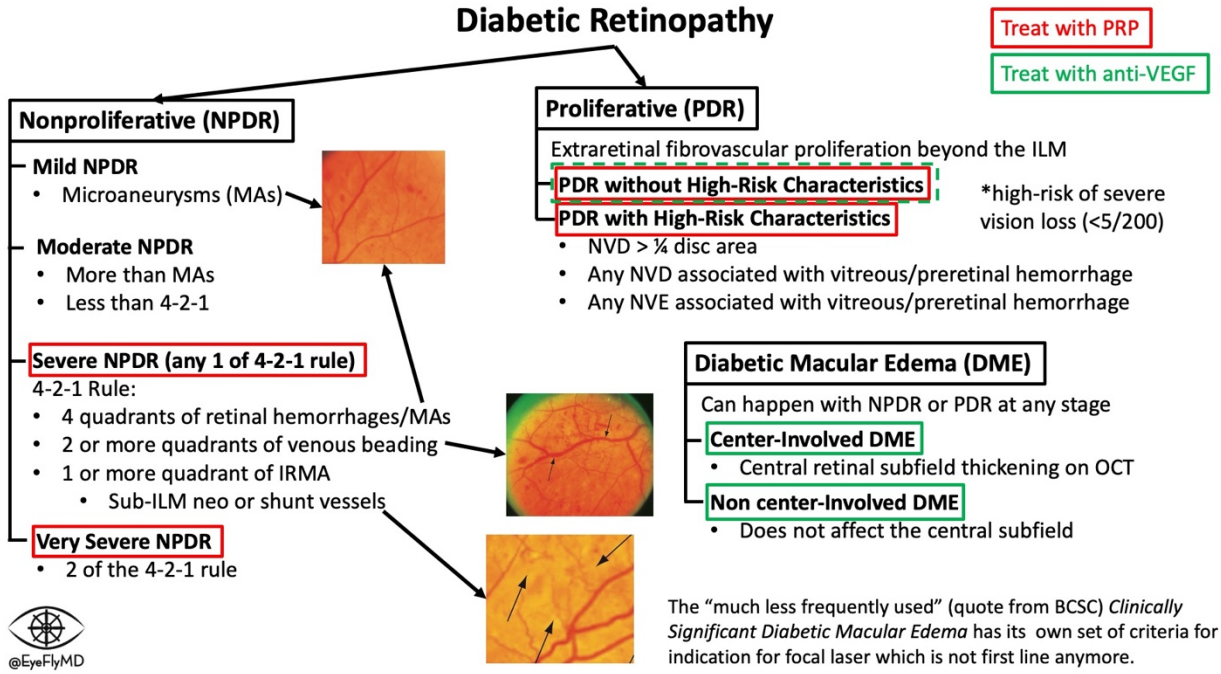
The precise underlying cause of diabetic Retinopathy is no understood but the result of chronic hyperglycemia on retinal vasculature is basement membrane thickening and loss of pericytes. This leads to capillary occlusion and retinal nonperfusion. The hyperglycemia can also cause a breakdown of the blood-retina barrier which can lead to extravasation of fluid and subsequent edema of the retina.

Screening for diabetic retinopathy depends on the type. Screening is usually annually beginning 5 years after diagnosis of Type I diabetes or at the time of diagnosis for Type II diabetes. Screening becomes more frequent during pregnancy depending on the severity.

The two broad categories for diabetic retinopathy are proliferative and nonproliferative. Simply, proliferative diabetic retinopathy is defined as fibrovascular proliferation that penetrates the internal limiting membrane (ILM). Non-proliferative diabetic retinopathy (NPDR) is classified as mild if only microaneurysms (MAs) are present. If there are more than MAs present (e.g., cotton wool spots, exudates) but it doesn't meet the criteria for severe NPDR. Severe NPDR is defined as any one of the 4-2-1 rule presented below. Very Severe NPDR is any 2 of the 4-2-1 rule. Panretinal photocoagulation should be considered for severe NPDR or worse with the goal to place 1200-1500 spots of argon laser burn throughout the peripheral retina to kill the cells, reduce oxygen demand, and thus reduce VEGF secretion. The reduced VEGF in the eye allows for regression of the neovascular vessels.

Proliferative diabetic retinopathy (PDR) occurs when neovascularization breaches the internal limiting membrane. It is classified as without high-risk characteristics that can be treated with anti-VEGF agents under certain circumstances, but PRP should be considered or with high-risk characteristics where PRP should be pursued. "High-risk" is a definition provided by the Diabetic Retinopathy Study: High-risk of Severe Vision Loss (SVL) defined as visual acuity less than 5/200 for at least 4 months at 5 years.

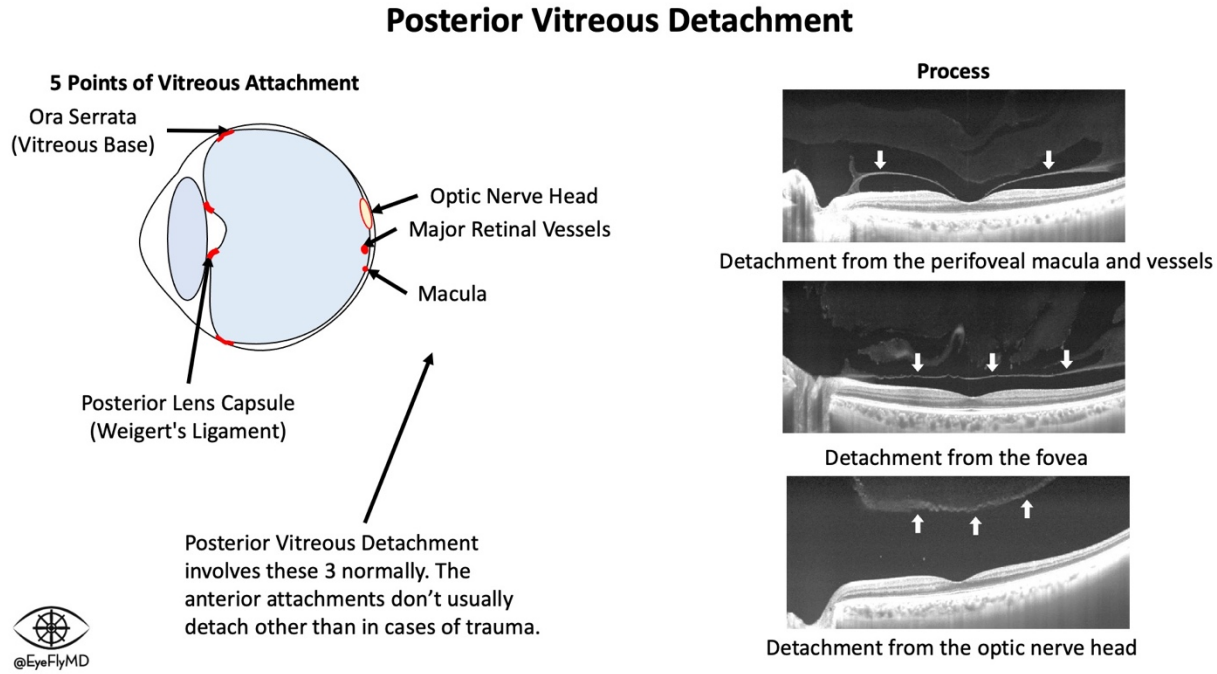
Diabetic macular edema (DME) can be classified by OCT or simple dilated fundus exam (DFE). By OCT, it is classified as "center-involved" if there is thickening of the central retina and "non-center involved" if it spares the central macula. Anti-VEGF agents or intravitreal steroids are first-line treatment options for DME.



Posterior Vitreous Detachment

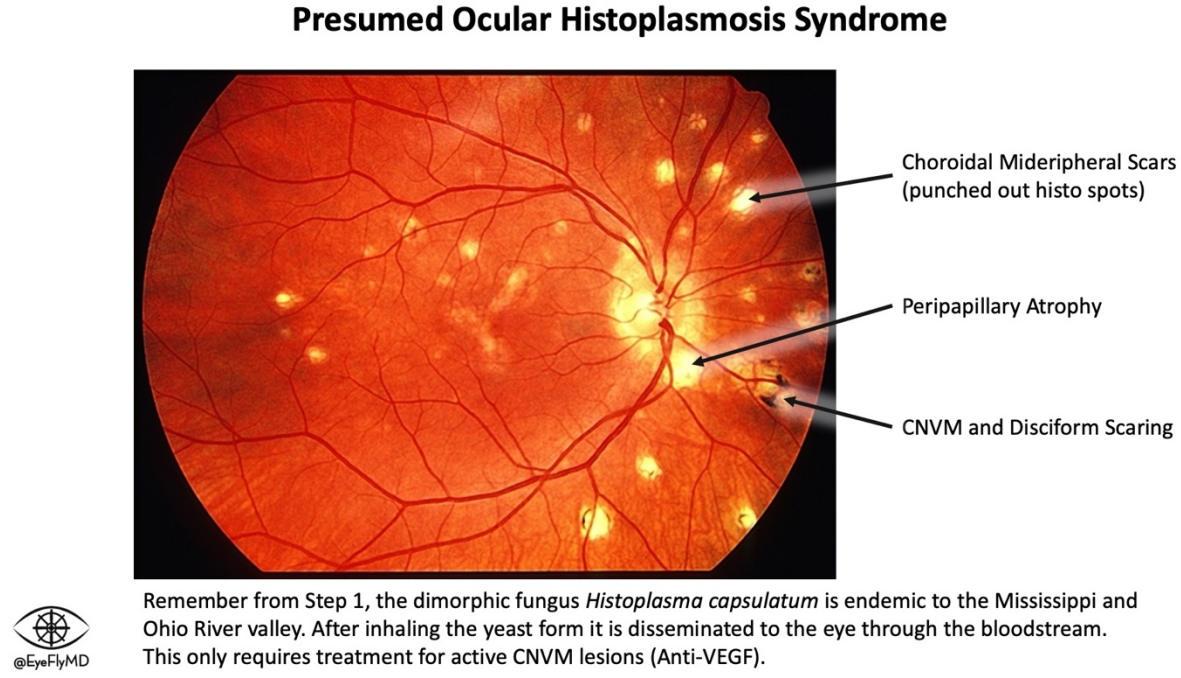
The vitreous is the clear made of water, collagen, and hyaluronic acid that occupies the posterior segment of the eye. It is attached to the retina at 4 major locations: the Ora Serrata, Posterior Lens Capsule, Major Retinal Vessels, Optic Nerve Head, and Macula. During normal aging, the vitreous undergoes **synchysis** (liquefaction) and **syneresis** (collapse).

It separates from the posterior as part of the normal aging process as Posterior Vitreous Detachment. This occurs earlier for myopes. It first separates from the perifoveal macula and vessels, then from the fovea, then from the optic nerve head. This process can be graceful. The process can also result in some retinal pulling manifesting as flashes and floaters. It can sometimes even cause vitreous heme. Things can go really south though if it is firmly attached to the macula. In this case it pulls the macula and results in an **Idiopathic Macular Hole** where it either separates the layers of the neurosensory retina or pulls it from the RPE.



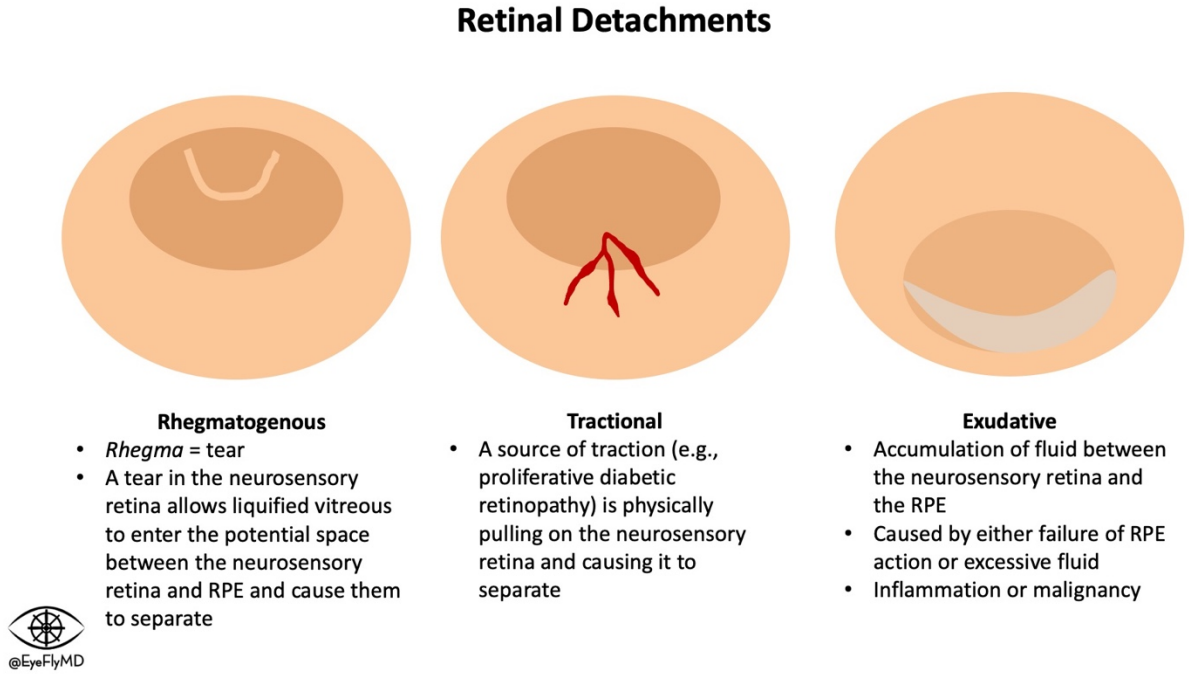
Presumed Ocular Histoplasmosis Syndrome

Remember the dimorphic yeast found in the Ohio and Mississippi river valleys from your Step 1 questions? This can appear in the eye in a variety of ways. It's usually a benign finding but needs to be treated if it starts causing choroidal neovascular membranes (the same way as always, anti-VEGF agents). It's called "presumed" because a causal link has not yet been established.



Retinal Detachment

A retinal detachment specifically refers to separation of the neurosensory retina from the underlying retinal pigment epithelium (RPE). The two broadest varieties are Rhegmatogenous and Nonrhegmatogenous. *Rhegma* means *tear* so these detachments involve holes in the neurosensory retina allowing liquified vitreous to enter and separate the layers. They can also occur from traction (something physcially pulling on the neurosensory retina) or an exudative process (fluid building up inner to the RPE).



CNVI Nerve Palsy

Recall that cranial nerve VI is the abducens nerve which innervates the lateral rectus. Palsy of this nerve is a common entity that presents with sudden onset ABduction defect (because remember CNVI innervates the lateral rectus) and sometimes esotropia in primary gaze. In older people without any other neurological signs this is usually a microvascular ischemic injury, and they can just wait the 3 months for resolution. In younger people (say <50) imaging is a good idea.

Common Surgeries and Procedures

Lasers

Lasers are used by every subspecialty of ophthalmology. The principle of lasers is using light or an electrical current to "excite" the electrons in a material (e.g., crystals or gas) and these electrons move from a lower-energy orbit to a higher-energy orbit around the atom's nucleus. This explains the LASER name: Light Amplification by Stimulated Emission of R

Ophthalmology Lasers and Wavelengths

Increasing Intensity

Tissue Interaction	Mechanism	Example	Relevant Wavelength
Photochemical	Photocatalysis	PDT, Corneal Crosslinking	CXL = UVA
Thermal	Increase in Temperature	PRP	513 nm (Argon Green)
Photoablation	UV Dissociation	LASIK, PRK	193 nm (Excimer)
Plasma-Induced Ablation	Plasma Ionization	SMILE	1053 nm (Femtosecond)
Photodisruption	Shock-Wave Generation	YAG	1064 nm (Nd:YAG)

Other Notable Wavelengths

- The visible spectrum is ~400-700 nm
- Rhodopsin is most sensitive to 510 nm
- Melanin, hemoglobin, and xanthophyll strongly absorb 400 nm - 580 nm
  - Because of this the retina is especially sensitive to blue light
- The natural Crystalline Lens blocks UV-A light (315–400 nm)
- The cornea blocks:
  - UV-B (280–315 nm)
  - UV-C (280 nm and below)
  - IR-B and IR-C (1400 nm to 1 mm)

LASER

Light **A**mplification by **S**timulated **E**mission of **R**adiation

$$Power = \frac{Energy}{Time}$$

$$Fluence = \frac{Energy}{Area}$$

$$\textbf{\textit{Intensity}}\ (Power\ Density) = \frac{Power}{Area\ (cm^2)} = \frac{Energy}{Time \times Area}$$

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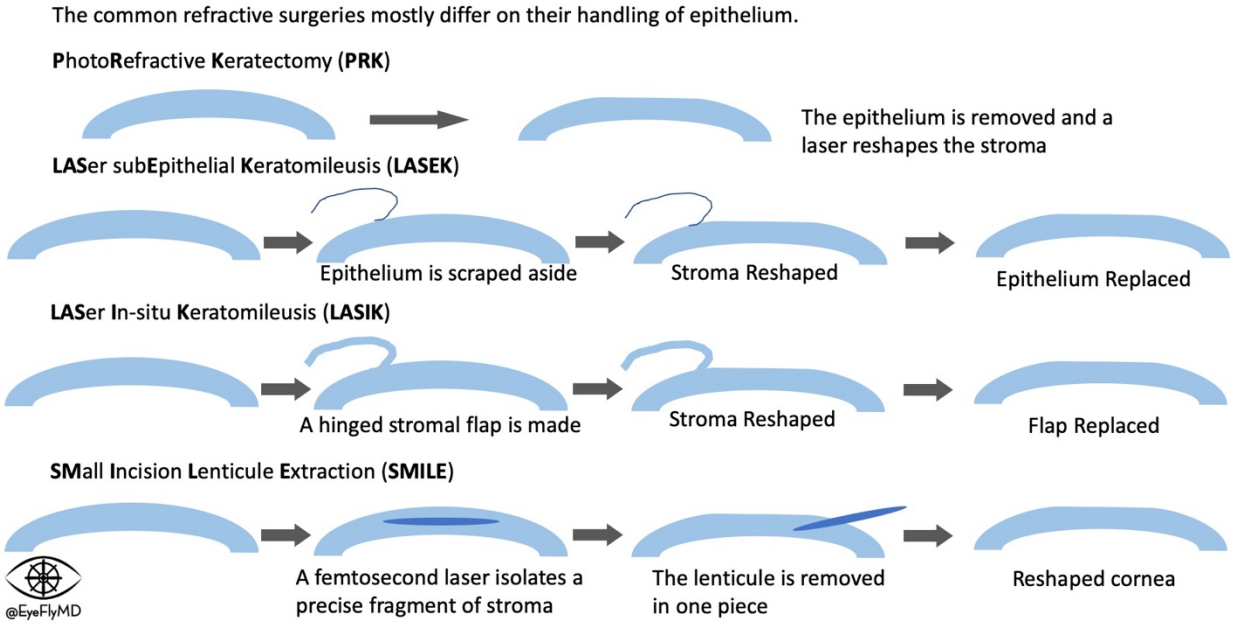
Cornea and Refractive

LASIK/Refractive Surgery

Refractive surgery usually involves changing the shape of the cornea. If an eye is myopic you can think of the eye having too much refracting power. LASIK (LASer In-situ Keratomileusis) is part of the laser-based refractive surgeries. These use the excimer laser with a wavelength of 193 nm to ablate corneal tissue. To reduce the refracting power of the cornea it must be flattened so corneal tissue is removed to make it flatter. If a person is hyperopic, they don't have enough refractive power so their cornea needs to be made "more curved." The refractive surgeries mainly differ on how they ablate the tissue with respect to the cornea. The spectrum is from lasering or scraping off the epithelium (PRK) to leaving it grossly untouched and not going anywhere near it (SMILE). SMILE uses the femtosecond laser (1053 nm) to carve out a lenticule of stromal tissue for removal to flatten the cornea. It can only currently be used to correct myopia with or without astigmatism.

SMILE is a theoretical improvement on LASIK because it has been proposed that the structural integrity of the cornea is mostly in its anterior 1/3, where LASIK is creating a flap. This was thought to minimize the risk of post-operative ectasia (corneal warpage). New data might disagree with this so stay tuned. SMILE is still in its first generation so many surgeons still prefer PRK and LASIK due to the abundance of data and reliability.

Common Refractive Surgery



If corneal tissue is to be removed, it is obviously important to have enough to work with. It is advisable to have a Residual Stromal Bed (left over stroma) of at least 250 µm. How do you tell how much stroma you'll end up with? For MYOPIA ONLY, the **Munnerlyn Formula** will produce approximate ablation depth. The formula is below but one of the main takeaways is the ablation depth is proportional to the *square* of optical zone diameter. Generally, an optical zone of 6.0 or 6.5 mm is best to reduce risk of glare and halos. The shorthand version is that for 6.5 mm optical zones, every diopter of astigmatism will require 14 µm. For 6.0 mm optical zones, every diopter of myopic correction will require 12 µm of ablation. For example, to correct 5.00 D of myopia for a 6.5 mm ablation zone it will require approximately 5 X 14 or 70 µm of corneal tissue to be removed. This means for very thin corneas the residual stroma may be too thin which is a risk factor for iatrogenic corneal ectasia.

Munnerlyn Formula Shortcut

$$\text{Ablation Depth } (\mu m) \approx \frac{\text{Degree of Myopia (D)} \times (\text{Optical Zone Diameter})^2 \text{ (mm)}}{3}$$

OR

Ablation Depth ≈ 14 µm / D for 6.5 mm Optical Zones

AND

Ablation Depth ≈ 12 µm / D for 6.0 mm Optical Zones

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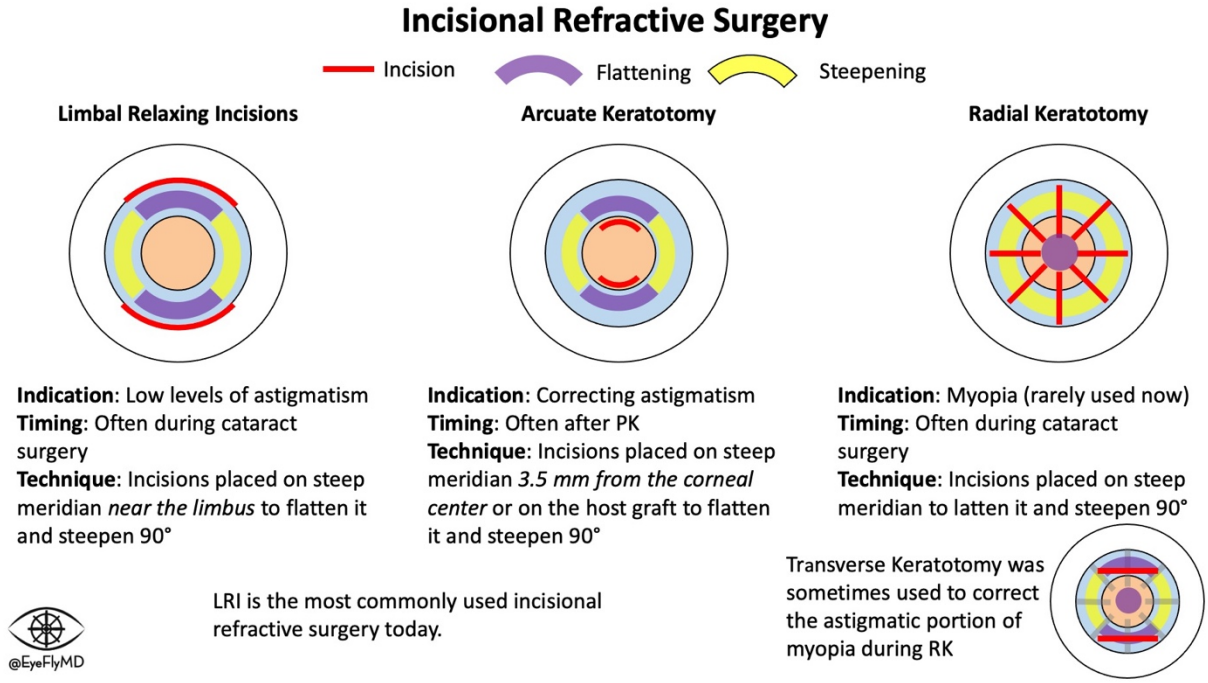
Another method of refractive surgery is **Incisional Refractive Surgery**. The most common variant used today is Limbal Relaxing Incisions (LRI). These are incisions placed in the cornea near the limbus usually at the time of cataract surgery. They're placed on the steep meridian to flatten it and produce a concurrent steepening 90° away. This results in a decrease in astigmatism. Depth and length of incision is determined by calculators, and this generally works well for low levels of cylinder. Arcuate Keratotomy is a technique similar to LRI but much closer to the center of the cornea (3.5 mm from the center). This generally results in greater degrees of astigmatism correction but is used mostly in post-PKP (penetrating keratoplasty) astigmatism. A relic that you might see is the scars from Radial Keratotomy. These were radial incisions placed in the peripheral cornea to correct myopia. The scars would cause the peripheral cornea to bulge and the central corneal would subsequently flatten, reducing the myopia. Patients experienced fluctuation throughout the day and a large portion of patients had the effect regress over time so this is rarely used today.



Phacoemulsification with IOL Implantation

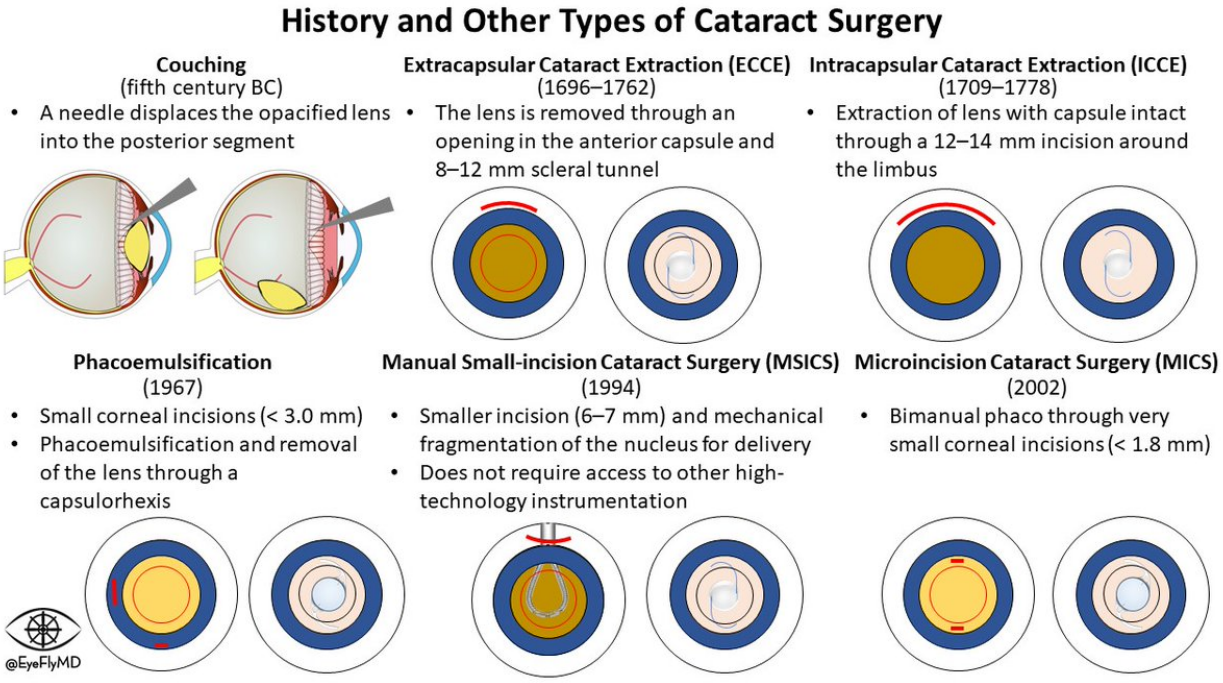
This is cataract surgery; it's also referred to as "Phaco" or "CE/IOL" for "cataract extraction." Phacoemulsification is the process of using ultrasound energy to liquefy the lens with the cataract and aspirate it, leaving the capsule intact. This capsule then receives an IOL (intraocular lens) that also corrects the patient's vision.

Cataract surgery has gone through many iterations beginning in the 5th century BC with a practice called couching. This is where a needle was used to simply disrupt the zonules of a dense cataract so it would fall back into the vitreous cavity.

Extracapsular Cataract Extraction came about in the 1600s and involved opening the anterior capsule and removing the lens through a scleral incision. Intracapsular cataract surgery involved a larger wound and was a more sophisticated form of couching that consisted of removing the lens still inside the capsule.

During WWII, a Royal Air Force fighter pilot who wasn't wearing goggles was shot down and sustained shards of the windscreen in his eye. An observant physician appreciated that this material caused no inflammation and was well tolerated by the eye so he proposed and eventually placed the first acrylic lens in 1949. Before this, patients would require strong plus spectacles ("coke bottle glasses") so the ability to put a lens in the eye was revolutionary.

Manual Small-Incision Cataract Surgery was described in the 1990s as a solution to safely remove dense cataracts especially in resource-poor areas. A phacoemulsification machine is expensive and not all facilities around the world will have them, so this skill is especially useful for global ophthalmology.



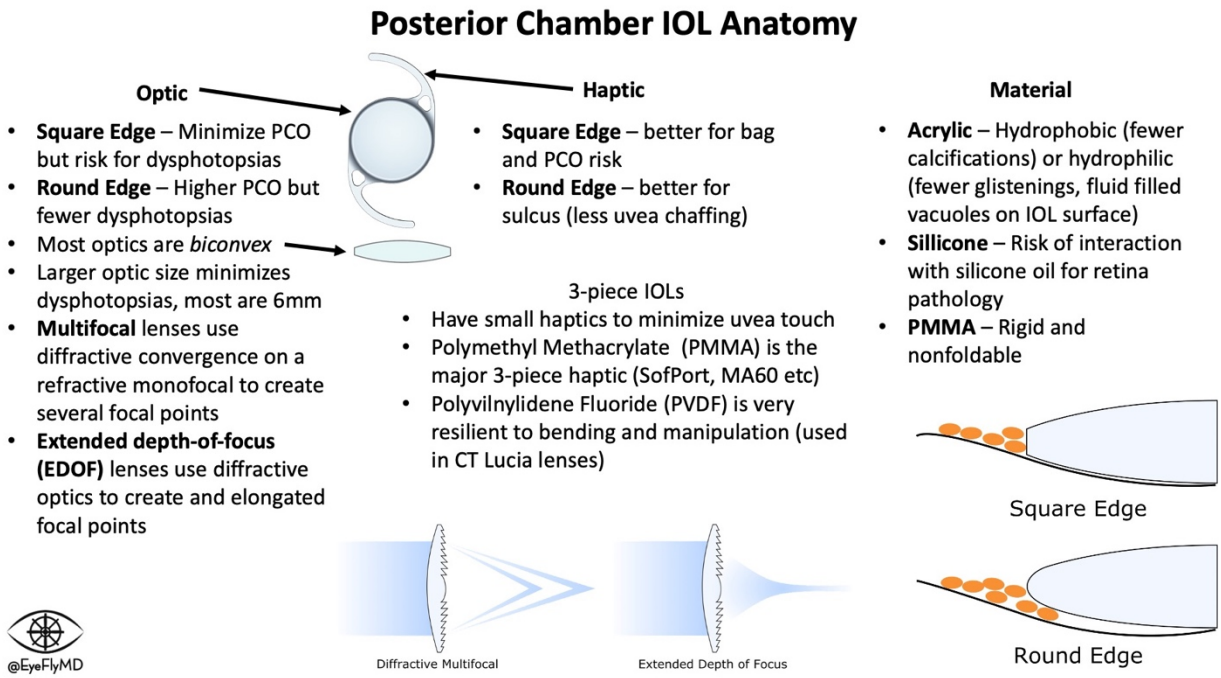
There are multiple types of lenses including:

- **Monofocal IOL** – The standard lens that corrects vision at one distance.
- **Multifocal IOL** – Premium lens (~$2,000) that corrects vision up close and at distance (many people complain of halos, glare, and loss of contrast though).
- **EDOF IOL** – An "Extended Depth of Focus" lens is also a premium lens that uses sophisticated optics to enlarge the distance light is focused on the retina. Patients can still experience halos, glare, and loss of contrast sensitivity.
- **Toric IOL** – A Torus is a donut shape. When you take a bite out of a donut there are two radii: the one dictating the diameter of the entire donut and the one dictating the diameter of the actual dough. These are also premium lenses that are inserted at the angle of the patient's astigmatism to correct this and provide better freedom from glasses.
- **Accommodating IOL** – Theoretically continues to accommodate like a natural lens.

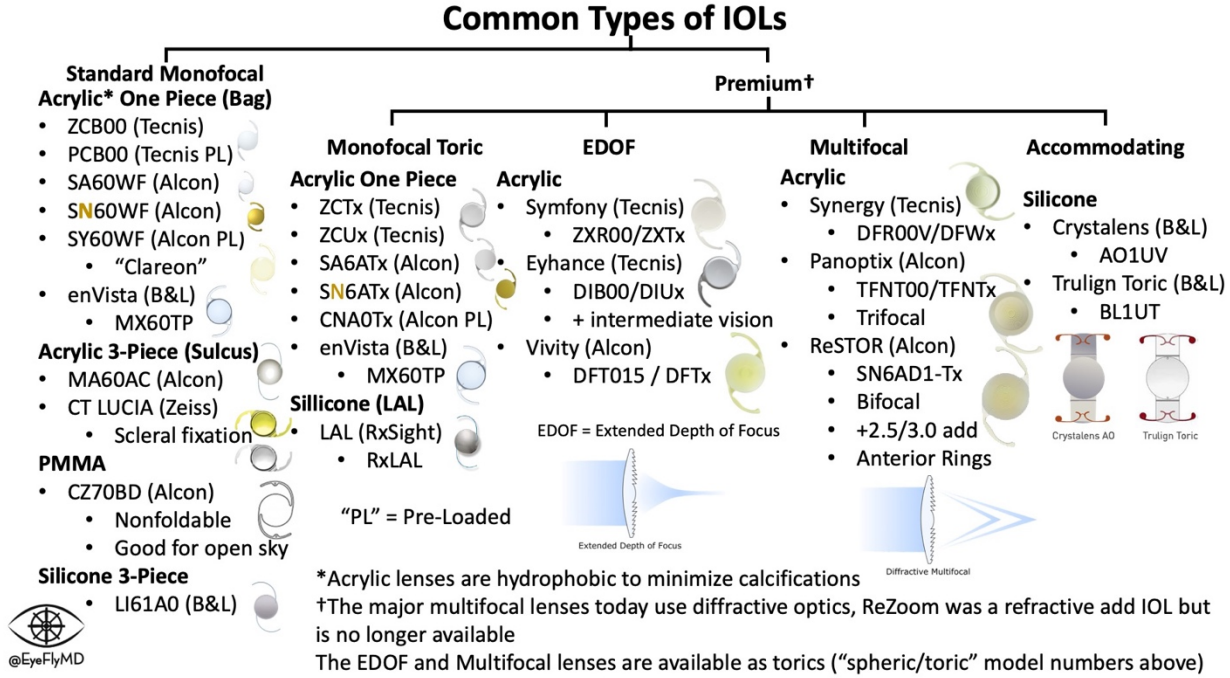
IOLs have a central optic portion and (usually) two haptics that hold the IOL in place in the capsular bag. Keeping the IOL stable in the bag is especially important in toric lenses where rotation would cause reduction in cylinder correction. For every 1° a toric IOL rotates off axis, ~3% of the cylinder power is lost so if the lens rotates 30°, the entire effect of the lens is lost. If it rotates >30°, the lens starts adding cylinder to the system.

Optics are normally 6 mm. The most common types of IOLs are Acrylic, Hydrophobic (to minimize calcifications), foldable, and square edge (to act as a physical barrier for epithelial cell migration and reduce PCO formation).

3-piece IOLs have small, filament haptics because they are typically placed in the ciliary sulcus so contact with the uveal tissues must be minimized.

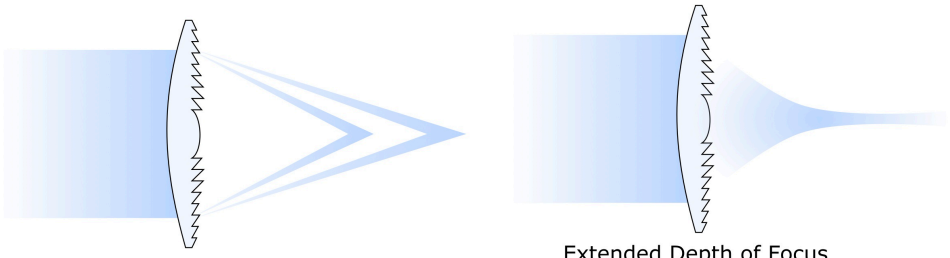


A very brief overview of some of the more common lens options can be found below, for a more comprehensive (but still not nearly all the options) list please visit the website under "Educational Resources". There are new lens options released every couple months (at least minor alterations to existing lenses) so I will try to keep the website updated as new model numbers come out. Below is a small sample of the most common of the most common IOLs.



*Acrylic lenses are hydrophobic to minimize calcifications
†The major multifocal lenses today use diffractive optics, ReZoom was a refractive add IOL but is no longer available
The EDOF and Multifocal lenses are available as torics ("spheric/toric" model numbers above)

Multifocals use a base monofocal and a diffractive element to create an additional focal point. EDOF lenses create an extended focal point by spreading the light. These come at the cost of contrast sensitivity because there is only so much light entering the eye and by using it to spread the light over a larger range or multiple focal points there is inherently a reduction in the amount of photons focusing on each point. Monofocal IOLs still provide the best contrast sensitivity because they focus all available light to a single point (remember no optical system is perfect and some light is always lost).



Diffractive Multifocal

Extended Depth of Focus

Modulation Transfer Function (MTF) charts or Defocus Curves (explained below) highlight this topic well. The cost of multiple focal points or extended focal ranges is contrast sensitivity. Surgeons are usually cautious to place premium (multifocal or EDOF) IOLs in eyes with existing or the potential for retinal or other eye pathology (e.g., AMD or glaucoma) because complicating the optical system further will risk suboptimal vision in the setting of disease.

Modulation Transfer Function (MTF) Charts and IOL Performance

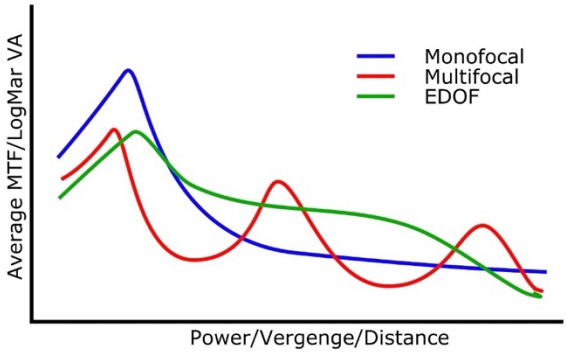
There are multiple ways to objectively compare IOL performances. These are called Modulation Transfer Function (MTF) Charts (sometimes Defocus Curves or Performance Charts). There can be different values on the X and Y axes so it's important to know how to read and interpret the data.

Y-Axis

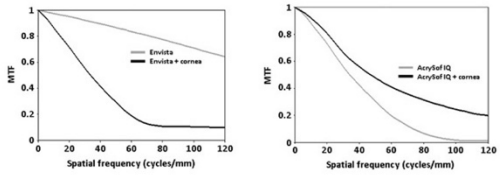
- **Modulation Transfer –** The ability of an optical system to transfer contrast to its image; MTF=Mi/Mo (image/object)
- **LogMar VA –** Visual acuity is scored with reference to the log of the minimum angle of resolution; 20/20 is LogMar 0

X-Axis

- **Spatial Frequency** - measured in line pairs per mm
- **Defocus** - measured in either D or distance



Examples:



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Here are hypothetical performance charts for the different IOL concepts. Notice there's no free lunch and only so much light entering the eye, to achieve multiple focal points or extended ranges there is typically a division of available light and a reduction in contrast.

Before cataract surgery, it's important to obtain accurate measurements of the eye (biometry) to accurately calculate the power of lens to use. Lenses are most often placed in the capsule but can also be placed in the ciliary sulcus or anterior chamber. The most important measurements for IOL calculations are axial length and corneal power. Extremes on either end of axial length or corneal power can impact the accuracy of calculations. The originally lens formula, SRK, is now mostly of historical significance but underscores the importance of accurate measurements. Appreciate that a 1 mm mistake in axial length equates to an average of ~2.5 D of error for the lens!

The key to successful cataract surgery is good IOL calculations. The original way to calculate lens power was called the "SRK" formula. It is mostly of historical significance but highlights some important concepts. Errors in axial length are the most significant

Principles of IOL Calculations

The SRK formula is a first-generation formula for calculating lens power and now mostly only of historical significance.

We have moved to more sophisticated calculations but the principles behind this formula underscore the importance of accurate axial length which is considered the most important variable in Biometry.

$$P = A - 2.5\,L - 0.9\,K$$

P = Lens Power for Emmetropia (D)
A = Specific IOL Constant (usually ~118)
L = Axial Length (mm)
K = Average corneal Power (D)

There are many IOL calculations now and improvements to accuracy are due to improved estimation of "**Effective Lens Position**", the distance between the cornea the principle optical plane of the IOL.

Notice an incorrect axial length of just 1 mm will result in a 2.5 D lens error!

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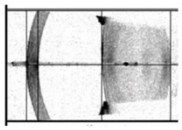
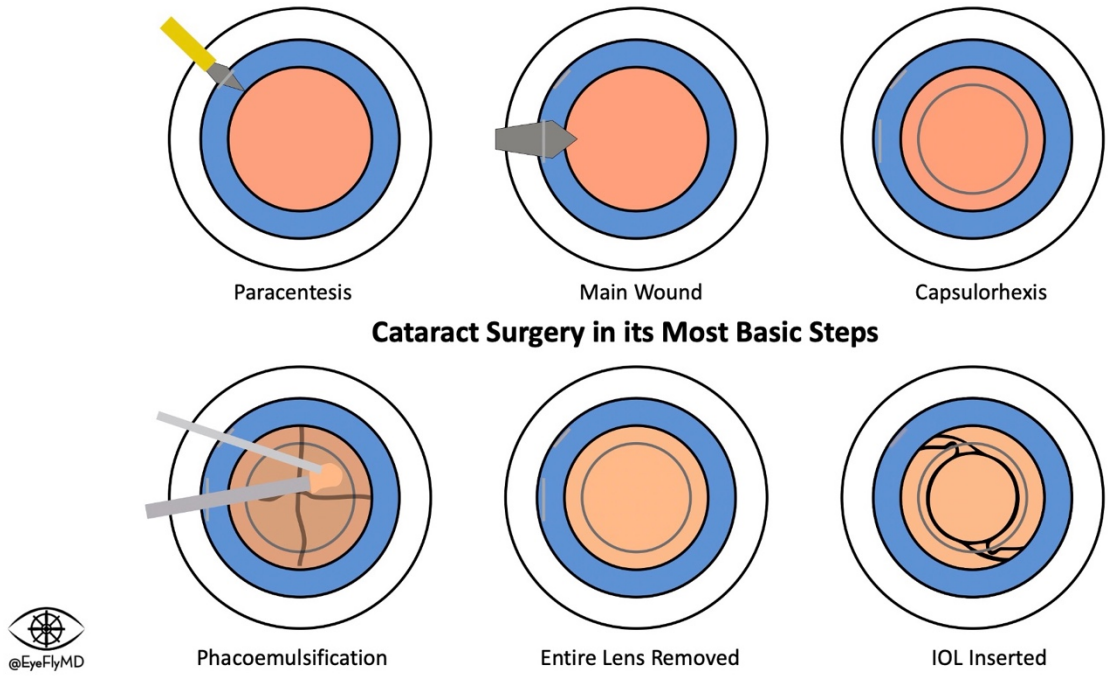
Recall the total refracting power of the eye is ~60 D, 43 D of which is from the cornea. Thus, expect IOL powers ~20 D.



Biometry of my eye!

Cataract Extraction (Phacoemulsification) with Intraocular Lens Insertion (CE/IOL)

1. A 1mm paracentesis (sideport) is made using an MVR blade.
    a. This allows two instruments to be used inside the eye.
2. Phenylephrine with Lidocaine is injected into the anterior chamber.
    a. This assists with pupil dilation and further analgesia. Patients usually feel an ache or burn.
3. Viscoelastic is injected into the eye to preserve the anterior chamber.
    a. This viscous solution contains hyaluronic acid and helps prevent the anterior chamber from collapsing. It also provide pressure on the anterior lens capsule to make the capsulorhexis safer.
4. A 2.4mm (sizes vary) clear (cornea) or near-clear (some limbal vessels) corneal incision is made temporally using a keratome.
    a. "Near-clear" means it is slightly through the vascular portion of the cornea to assist with wound healing.
5. Continuous curvilinear capsulorhexis is achieved using different tools, creating a ~5.5mm opening in the anterior capsule.
    a. The anterior capsule is peeled away in a circular fashion
6. Hydrodissection is achieved by injecting BSS via Chang (or other) cannula immediately underneath the anterior capsule. This will facilitate lens rotation and cortical removal.
    a. This begins the separation of the lens from the capsule.
7. The lens is rotated to ensure complete separation from the capsule.
8. Phacoemulsification – Liquification and removal of the lens.
    a. The lens is liquified using ultrasound and aspirated through the handpiece.
9. The remaining cortical material is removed using I/A handpiece.
    a. The cortex of the lens that's still adhered to the capsule is removed.
10. Cohesive viscoelastic is injected into the capsule prior to pulling out the I/A handpiece.
    a. This makes it easier to insert the new lens because the shape is retained.
11. The foldable IOL is injected into the capsule.
12. The viscoelastic is removed and BSS in injected to reform the chamber.
13. Hydration of both wounds ensure watertight seal.
    a. Creating local edema in the cornea helps seal the wounds



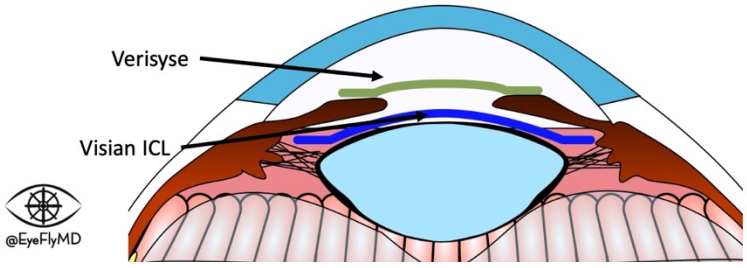
IOLs can also be placed other places in the eye. As discussed earlier, 3-piece IOLs are placed in the ciliary sulcus. IOLs can be placed in phakic eyes as well. This is usually to correct high orders of refractive error where laser surgery (LASIK or SMILE) would not be feasible.

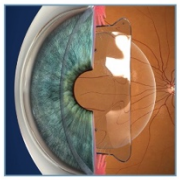
Phakic IOLs

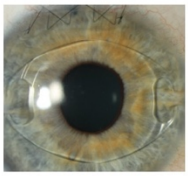
Procedure:
- The Visian ICL (Implantable Collamer Lens) is injected through a small wound and sits in the sulcus between the natural crystalline lens and the iris
  - They are vaulted 1.0 ± 0.5 corneal thicknesses (250-750 µm) over the natural lens to optimize aqueous flow while avoiding pupillary block or iris chafing
- The Verisyse Lens is fixated to the iris (enclavation); the wound is large

Indications:
- Cornea is too thin, flat, or steep
- Refractive error is too high
- Visian ICL:
  - -3.0 to -20.0 D
  - 1 - 4 D of astigmatism
- Verisyse Lens:
  - -5.0 to -20.0 D
  - No Toric version

Complications:
- Anterior subcapsular cataract risk is up to 10% with phakic posterior chamber IOLs
- Endothelial cell loss and dislocation are risks with the anterior chamber lenses




STAAR Visian ICL

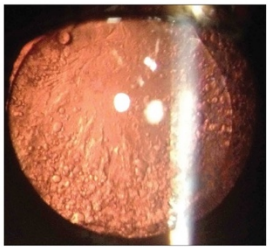

Verisyse Lens

YAG Capsulotomy

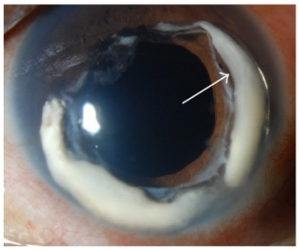
The most common "complication" from cataract surgery is proliferation of remaining posterior lens epithelium and opacification formation. This is called Posterior Capsular Opacification (PCO) This is a laser procedure for when a patient who had cataract surgery tells you the cataract has come back but they actually have a PCO. The cataract can never recur because the lens has been removed but the capsule that is holding the lens can opacify and the patient can experience recurrence of cataract symptoms. This laser just breaks the fibrous bands causing the symptoms.

Posterior Capsular Opacification

- "Secondary cataract"; the most common postoperative complication of cataract surgery
- Residual lens epithelial cells from the anterior capsule undergo proliferation, migration toward the posterior capsule, and normal and abnormal differentiation
- Can feature Elchsnig Pearls (of swollen Bladder/Wedl cells), Soemmering Rings (where anterior and posterior capsule meet), or Capsular WRINKLING
- Occurs in 20-50% of patients within 2 to 5 years of cataract surgery



Elschnig Pearls



Soemmering Ring

The 1064 nm Nd:YAG "YAG" laser uses photodisruption to open the posterior capsule using most commonly a cruciate or circular pattern (sometimes spiraling if lens dislocation is a concern) with a typical power between 0.8 – 3.0 mJ and between 10-20 shots

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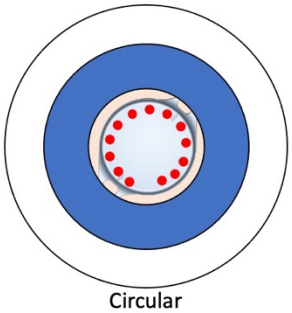
Cruciate



Circular

Retina

Pars Plana Vitrectomy (PPV)

After a retinal detachment, the source of the detachment (e.g., traction from the vitreous) must be eliminated. Going through the Pars Plana portion of the ciliary body (where there is no retinal tissue) ports are placed and the vitreous and ILM are surgically removed. The area around the tear is often lasered to create a chorioretinal scar that will prevent further separation of the neurosensory retina from the retinal pigment epithelium. A gas bubble is frequently placed in the eye to eliminate any currents from disrupting the tear further and the patient must assume a face-down position usually for the gas bubble to be positioned over the tear.

Scleral Buckel

Also frequently following a retinal detachment, a band is placed around the sclera to relieve traction. This is often combined with cryopexy which uses liquid nitrogen to freeze the ocular tissue and create a chorioretinal scare similar to the PPV. Squeezing the eye makes it longer which results in... you guessed it, increased myopia.

Pneumatic Retinopexy

If the tears are small, superior, and close together a gas bubble can be injected in-office. When combined with face-down positioning this can be a way to treat retinal detachments without the complications of more aggressive surgery.

Glaucoma

Micro-invasive glaucoma surgeries (MIGS)

These are a set of newer procedures that allow for intervention earlier in the disease and prevent the progression of optic nerve damage. Most of what Dr. An performs are MIGS.

Kahook Dual Blade (KDB) Goniotomy

The Kahook Dual Blade (KDB) is a surgical knife is designed to excise a strip of trabecular meshwork in a procedure known as excisional goniotomy (side note: "gonio" means "angle" like a "goniometer" in orthopedics measures angles for range of motion. A "gonioprism" allows visualization of the angle, "gonioscopy", and "goniotomy" means "cutting into the angle"). The procedure can be performed at the time of cataract surgery or as a standalone procedure and is effective in mild to advanced primary open angle glaucoma (POAG) and has been performed successfully in secondary glaucomas as well. The instrument has two (dual) blades that excise the trabecular meshwork and a wedge shape that lifts it away for removal.

XEN Gel Stent

XEN gel stent facilitates drainage of aqueous humor into the subconjunctival space (forming a Bleb) to lower IOP via a XEN ab-interno approach using intraoperative gonioscopy. Recently, an ab-externo approach (without the use of gonioscopy) has been gaining popularity for its easier application and better positioning of the stent, potentially leading to less intraoperative complications.

Hydrus

A microstent that scaffolds 90° of Schelmm's canal ensuring consistent access to collector channels.

iStent

The iStent trabecular micro-bypass shunts aqueous across the trabecular meshwork (TM) into Schlemm's canal. The device's label specifies that the procedure be combined with cataract surgery in eyes with mild to moderate open-angle glaucoma (OAG).

MLT

MicroPulse® laser trabeculoplasty (MLT) is an advancement over its older counterpart selective laser trabeculoplasty (SLT). Both of these procedures use a laser to biochemically alter the trabecular meshwork and increase aqueous outflow. MLT allows for tissue cooling in between energy pulses, preventing surrounding tissue damage compared to SLT or even the older argon laser trabeculoplasty (ALT).

MPCPC

A laser ablation technique. Micropulse diode cyclophotocoagulation (MPCPC), used to decrease the inflow of aqueous humor by destroying the ciliary body and reducing aqueous humor production.

Traditional Surgeries

While the above procedures were MIGS, the older procedures include "trabs and tubes" or trabeculectomies and tube placement procedures. Trabeculectomies form tunnels into the anterior chamber to reduce IOP and tubes like Ahmed, Moreno, or Baerveldt form Blebs like XEN but are more complicated surgeries.

Common Medications

Allergies

- **Prescription** – Pataday (olopatadine), Acular (ketorolac), Alrex (loteprednol)
- **Over the Counter** – Alaway (ketotifen), Zaditor (ketotifin)

Antibiotics

- **Antibiotic Drops**– Vigamox (moxifloxacin), Ocuflox (ofloxacin), Tobrex (tobramycin) sol./oint., AzaSite (azithromycin) sol., Polymyxin B Combinations
- **Antibiotic Ointments** –Tobrex (tobramycin), Erythromycin, Bacitracin

Dilating

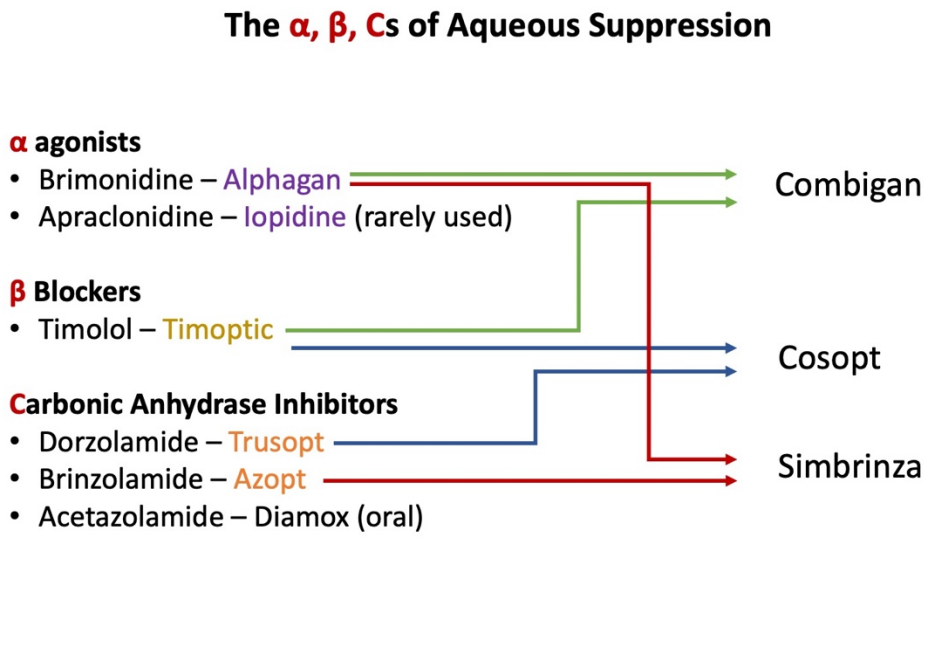
- **Sympathomimetics** – Phenylephrine stimulates the dilating muscles like the "flight or fight" response. Lasts ~6 hours.
- **Anticholinergic** – Tropicamide (~12 hours) and atropine (~2 weeks) relaxes the muscles that constrict the pupil (think of it like preventing the opposite of "flight or fight" so you're left with just "flight or fight." It also prevents the lens from accommodating so patients will have difficulty reading. It is also necessary to adequately cycloplege (paralyze the ciliary body) kids who have a large accommodative amplitude in order to obtain an accurate refraction.
- A common regimen for in-office dilation is proparacaine (to eliminate the pain associated with tropicamide and the TonoPen), tropicamide, and phenylephrine.

Dry Eye/Meibomian Gland Dysfunction

- **Lubricants** – Drops like Refresh Optive Advanced, Refresh Optive Advanced Preservative Free, Systane Balance, Systane Ultra, Systane Ultra Preservative Free have similar composition to human tears. Preservative free causes less irritation.
- **Immunosuppressants/Anti-Inflammatories** – Restasis (cyclosporine) and Xiidra (lifitegrast) prevent inflammation.

Glaucoma

- **α Agonists** – Drops like Alphagan®P (brimonidine), Iopidine® decrease aqueous humor production.
- **β-Blockers** – Drops like Timolol decrease aqueous humor production.
- **Carbonic Anhydrase Inhibitors** – Drops like Trusopt® (dorzolamide), Azopt® (brinzolamide) and pills like Diamox (acetazolamide) decrease aqueous humor production.
- **Prostaglandins** – Drops like Xalatan® (latanoprost), Lumigan® (bimatoprost), Travatan Z® (Travoprost), and Zioptan™ (tafluprost), and Vyzulta™ (latanoprostene bunod) increase uvealscleral outflow by an unknown mechanism.
- **Cholinergics** – Drops like Pilocarpine increase flow through the trabecular meshwork.
- **Combination** – Any drug with the "co-" prefix counts as two medication classes. Examples include Combigan™ (Brimonidine & Timolol), Cosopt® (Dorzolomide & Timolol), and Simbrinza® Suspension (Brinzolamide & Brimonidine).
- **Netarsudil (rhopressa)** – Newer medication, Rho-kinase inhibitor and norepinephrine transporter (NET) inhibitor. Rho-kinase inhibition increases outflow through the TM and lowers episcleral venous pressure. NET inhibition decreases production of aqueous humor.



Herpes/Varicella Zoster

- **Anti-virals** – Viroptic (Trifluridine) drops or acyclovir, valacyclovir, and famciclovir oral medications.

Inflammation

- **NSAIDS** – Bromsite (bromfenac), Acular LS (ketorolac tromethamine), Ilevro (nepafenac), Voltaren (diclofenac sodium)
- **Corticosteroids** – Most commonly Prednisolone Acetate 1% for uveitis and post-op; Kenalog can be injected sub-tenon or subconj; there are also depot steroids that can be placed in the retina to treat conditions like uveitis over a long period of time.

Common Eponyms

I have attempted to collect every eponym I have come across. If I'm missing any, let me know.

	Definition/Association	Appearance/Details
Adie Tonic Pupil	Unilaterally Dilated Pupil	with light-near dissociation
Aicardi Syndrome	Epilepsy, agenesis of CC, chorioretinal lacunae	Coloboma ONH, absence of corpus collosum, R ID, **Depigmented lacunae**, Infantile seizures
Alagile Syndrome	Arterio-hepatic dysplasia	posterior embryotoxin plus **butterfly** vertebrae and jaundice
Alexander's Law	Nystagmus increases in intensity (amplitude and frequency) as the eyes are moved in the direction of the fast phase	
Alport Syndrome	Familial Oculorenal, + hematuria	lenticonus, microspherophakia, like Lowe
Alström Syndrome	Obesity, RP fundus, DM 2, cardiomyopathy	cilliopathy; JABS
Amsler-Verrey Sign	Fuch's Heterochromic Iridocyclitis	small hyphema after pericentesis in FHI
Annulus of Zinn		ring-shaped structure formed by the tendinous insertions of the four rectus muscles
Anton Syndrome	Denial in cortical blindness	
Apert Syndrome	Cosntant XT; craniosyostosis facies + syndactyl	V pattern; mitten deformity; exorbatism
Arlt's Line	Trachoma	upper lid follicular scarring
Arlt's Triangle	Triangle with apex in center of cornea	KP location in uveitis
Argyll-Robertson	Light-Near Dissociation	pupils do not miose in response to light, but do so briskly in response to the near reflex
Arnold-Chiari Malformation	Periodic Alternating Nystagmus or Downbeat	cervical/medullary problem
Athen's Protocol	PRK combines with CXL	
Avellino Corneal Dystrophy	Combined Lattice-Granular Dystrophy	type 2
Axenfeld Conjunctivitis	Moraxella Conjunctivitis	follicular conjunctivitis
Axenfield-Reiger Syndrome	Neurocristopathy; ASD	posterior embryotoxin plus iris strands; **dental** malformation
Bardet-Beidl Syndrome	Hypogonadism, RP fundus, Obesity, Polydactyly	cilliopathy, JABS
Balint Syndrome	Optic ataxia, motor apraxia, simultagnosia	
Bangerter Foil	Amblyopia	foil to occlude lens
Berlin Nodules	Sarcoid iris nodules: angle	
Best Disease	Fried egg fovea, EOG Arden <1.7, BEST1/VMD1	
Bjerrum Area	central 25° of the visual field from the fixation point; glaucomatous field loss	
Blessig-Iwanoff Cysts	Cystic appearing peripheral retina	
Busacca Nodules	Sarcoid iris nodules: mid-iris	
Bassen-Kornzweig Disease	Abetalipoproteinemia	secondary RP; acanthocytosis; cilliopathy

Batten Disease	Neuronal ceroid lipofuscinoses	secondary RP; ceroid and lipofucein in cells, AR; cognitive decline, myoclonus
Behçet's Disease	Oral ulcer, acute nongranulomatous uveitis	also causes a retinal vasculitis; AKA Adamantiades-Behçet's Disease
Bell's Phenomenon		involuntary elevation of the eye when attempted lid closure
Benedikt Syndrome	Ipsilateral 3rd plus hand flap	fascicular syndrome; red nucleus
Bietti Crystalline Dystrophy	Crystalline Choroidal Dystrophy	other two choroidal dystrophies are choroideremia (RP) and Gyrate Atrophy
Binkhorst Formula	Measured axial length (AL) to estimate ELP	2nd generation theoretical formula
Bitot Spot	Xerosis	
Blair Witch Cataract	Chlorpromazine (Thorazine)	think corneal verticillata though
Blau Syndrome	Band keratopathy, polyarthritis, skin rash, anterior uveitis, chorioretinopathy	
Bloch-Sulzberger syndrome	Incontinentia Pigmenti	splashed paint skin, **dental** malformation; consider ARS and congenital Lues
Bournville Disease	Tuberous Sclerosis	epiloia = epillepsy, low intelligence, angiomas; ash leaf spots; retinal astrocytic hamartoma
Brown Syndrome	SO dysfunction	deficient elevation in adduction 2 o to restriction of the SO tendon at the trochlea; M > F, OD > OS
Brown-McLean Syndrome	Long-Term Aphakia	peripheral corneal edema with pigmented bullae
Bruckner Test	Strabismus screening	red reflex is lighter on deviating side
Brushfield Spots	Down Syndrome	pale areas on iris periphery
Burgmeister's Papillae	PFV	hyaloid remnant at ONH
Chandler Syndrome	ICE Variant	corneal edema and glaucoma
Charcot Joints	Tertiary Syphillis	malformed joints from tabes dorsalis of neurosyphilis
Charles-Bonnet Syndrome	Glaucoma	hallucinations
Chediak-Higashi Syndrome	Oculocutaneous albinism + immune dysfunction	silver-grey hair
Ciancia Syndrome	Congenital Comitant Esotropia cause	nystagmus worsens on abduction; LARGE deviation unlike nystagmus blockage syndrome
Claude Syndrome	Ipsilateral 3rd plus contralateral ataxia	fascicular syndrome; cerebellar peduncle
Cloquet's canal	Trans-vitreous passageway	of the old hyaloid artery
Coat's Disease	White-yellow subretinal exudates	male, 6-8, unilateral, non-inherited, telangiectasias, laser or cryo
Cogan Lid Twitch	MG	upper lid overshoots on upgaze
Cogan Syndrome	IK, tinnitis, vertigo	noninfectious
Cogan-Reese Syndrome	Iris-Nevus Syndrome	ICE variant
Cogan's Microcytic Dystrophy	EBMD	aka map-dot-fingerprint
Conoid of Sturm	The space from one focal line to the other	

Collier's Sign	Parinaud Syndrome	lid retraction on upgaze
Cowdry Bodies	HSV, VZV, and CMV	eosinophilic nuclear inclusions
Crigler Massage		
Crouzon Syndrome	Cosntant XT; craniosyostosis facies	V pattern; exorbatism; FGFR2
Dalen-Fuchs Nodules	Sarcoid/VKH/SO (granulomatous dz)	focal collections of inflammatory cells between Bruch's membrane and the RPE
de Morsiers	Septo-Optic Dysplasia	optic nerve hypoplasia, absence of septum pellucidum, absence of corpus colosum
Devic's Disease	Neuromyelitis Optica	bilateral optic neuritis that fails to recover + transverse myelitis; aquiporin-4
Dorello's Canal	CNVI Palsy	CN6 gets to the CS by traversing…Dorello's canal; cavernous sinuses drain into the internal jugular vein primarily via the inferior petrosal sinus that gets from the CS to the internal jugular by travelling within a structure called
Dresden Protocol		
Duane Syndrome	Congenital Strabismus	lateral rectus innervated by CNIII; globe retraction on adduction, F > M, OS > OD
Eales Disease	Obliterative retinal vasculitis	bilateral peripheral neovascularization; TB positive, India; Occlusive retinal vasculitis
Eardheim-Chester Disease	Substype of xanthogranulomatous disease; causes cardiac problems and death; INFLAMMATORY vs. normal Xanthelasma	
Elschnig Pearls	Opacity from grouped Wedl/Bladder Cells	in PCO
Elschnig Spot	Hypertensive Choroidopathy	black spots surrounded by halo
Fermat's Principle of Least Time	Light has a tendency to optimize its path	lensmakers must make a lens that has equal timeat each point
Ferry Line	Filtering bleb	iron line
Felderstruktur	Tonic slow muscle fibers of EOMs	
Fibrillenstruktur	Twtch muscle fibers of EOMs	
Fleck Corneal Dystrophy	Stromal, dandruff like, childhood	
Fleischer Ring	KCN	iron in stroma at base of cone
Flexner-Wintersteiner Rosette	RB	retinoblasts around an empty lumen
Förster-Fuchs Spots	Pathologic Myopia	area of RPE hyperplasia overlying regressed CNVM
Foster Kennedy Syndrome	Intracranial mass damaging optic nerve	unilateral papilledema; olfactory groove hemangioma
Fossonella Servat	Tarsoconjunctival mullerectomy	For Small amounts of ptosis (<2mm)
Foville Syndrome	Ipsilateral 6th + 7th + loss of taste	fascicular syndrome; tractus solitarius
Fraser Syndrome	Cryptophthalmos	

Fuch's Adenoma	Pseudoadenomatous hyperplasia of the CB	A small (<1mm) nodular hyperplasia of the nonpigmented epithelium of the ciliary body; coronal adenoma
Fuch's Coloboma	Tilted Disc Syndrome	bitemporal hemianopia, doesn't respect midline
Fuch's Heterochromic Iridocyclitis	Chronic unilateral uveitis/glaucoma	heterochromia iridis, cataract, and stellate KP
Fuchs Crypts	Craterlike openings in surface of anterior iris	
Fuchs Endothelial Dystrophy	Beaten Bronze	guttae and progressive corneal edema
Fuchs Marginal Keratitis	Peripheral nonulcerative keratitis	thinning to perphoration, looks like Terrien's
Gardner Syndrome	Familial adenomatous polyposis	Pisciform CHRPE-like lesions
Gass Plaques	Susac Syndrome	chronic accumulations of serous material
Goldenhar Syndrome	Oculo-auriculo-vertebral syndrome	limbal dermoids, **butterfly** vertebrae
Goldmann Equation	Aqueous Pruduction/Ouflow + EVP	
Goldmann-Favre Syndrome	Enhanced S-cone dz/syndrome	macula looks like XLJR, periphery looks like RP, nyctalopia; vitreoretinopathy
Gorlin-Gortz Syndrome	Basal Cell Nevus Syndrome	myelated RNFL (also high myopia, NF1, strab, ambylopia)
Gradenigo Syndrome	Ipsilateral 6th plus ipsilateral facial pain	subarachnoid lesion, petrous bone; "Petrous apex syndrome"; hemifacial palsy
Grave's Disease	TED	
Güllstrand Eye	Model eye, 60D power	
Gunderson Flap		
Haab Straie	Congenital Glaucoma	tears in Descemet's
Harada-Ito Procedure	SO palsy	both SO tendons are split and the anterior part is moved anteriorly and temporally
Hartmann-Shack wavefront sensor	Turns eye into a camera for wavefront analysis	
Hashimoto Thyroiditis	Hypothyroidism	
Hasall-Henle Bodies	Peripheral Corneal Gutae	normal in elderly
Haidinger Brush	View polarized light against a white or blue background	
Haigis Formula	4th generation	4th generation
Haller's Layer	Large Vessels	of the choroid
Heerfordt syndrome	Sarcoidosis	CN7 palsy (paralytic ectropion) 2/2 parotid swelling
Hermansky-Pudlak Syndrome	Oculocutaneous albinism + platelet dysfunction + pulmonary interstitial fibrosis	
Heimann-Bielschowsky Phenomenon	Monocular nystagmus of childhood in adult	vertical/eliptical nystagmus; involved eye never changes
Heinz bodies	Hemolytic glaucoma	hgb-laden macrophages
Hemholtz Theory of Accomidation	Accomidation occurs at the central anterior lens	

Henderson-Patterson Corpuscles	Molluscum	eosinophillic inclusions
Henle's layer	Outer Plexiform Layer	
Herbert's Pits	Trachoma	limbal follicle scarring
Hermansky-Pudlak Syndrome	Oculocutaneous Albinism, cause of	
Herring's Law	of Muscle Correspondence	Innervation to yoke muscles is equal
Hirschprung's disease	Congenital Megacolon	associated with Waardenberg Syndrome
Homer-Wright Rosette	RB	retinoblastss around a lumen of a neurofibrillary tangle
Horner Syndrome	Ptosis, Miosis, Anhidrosis	
Horner-Trantas Dots	VKC	papillae at limbus
Hudson-Stähli line	Old age iron line	junction of the lower- and middle-thirds of the cornea
Hurler Syndrome	Mucopolysaccharidosis	cornea cloudiness at takes 6 weeks to 24 months
Hutchinson Teeth	Congintal Syphillis	peg shaped, IK + Huthcinson teeth + deafness
Hutchinson's Sign	VZV erruption	a vescicular lesion located at the lateral aspect of the tip of the nose
Imbert-Fick Principle	P=F/A	diameter = 3.06mm
Infantile Refsum Disease	Infantile Phytanic Acid Storage Disease	RP fundus. deafness, hypotonia; AR
Irvine-Gass	Post-CE CME	inflammatory blood-retina barrier comprimise
Jackson Cross	Cylinder with S.E. = 0 (-1.00 + 2.00 @180)	
Jones Test		
Joubert Syndrome	RP fundus, seizures, hypotonia, apnea	MOLAR TOOTH SIGN; cilliopathy; JABS
Kaiser-Fleischer Ring	Wilson's/PBC/Active Hepatitis	iron in Descemet's
Kaposi Sarcoma	HIV	highly vascular tumor usually of the skin or mucous membranes
Kasabach-Merritt Syndrome	Capillary heamangioma	a thrombocytopenic coagulopathy secondary to sequestration of platelets in an associated large capillary hemangioma located in the GI tract
Kaye Dots	Necrotic epithelial spots after PKP near sutures	
Kearns-Sayre Syndrome	CPEO + pigmentary retinopathy + heart block	ragged red fibers
Kestenbaum-Anderson Procedure	Resettign gaze to dampen nystagmus	
Khododoust Line	Endothelial Rejection	blood cells on the endothelium
Kjer;s Syndrome	AD Optic Atrophy, most common heriditary optic neuropathy	
Klippel-Trénaunay syndrome	2nd cause of port wine stain	phakomatosis, stain on hypertrophied limbs too
Kleinfelter's Syndrome	AICARDI, IP	XXY, these conditions are lethal in males but appear in XXY
Knapp Procedure	Double Elevator Palsy	relocating the LR and MR insertions toward the SR

Knobloch Syndrome	Occipital encephaloceole, high myopia, RD	hypoplastic RPE, cryptless iris; vitreoretinopathy
Koeppe Nodules	Sarcoid iris nodules: Pupillary margin	
Koplik Spots	Rubeola (Measles)	spots in mouth
Krause Glands	Accessory Lacrimal Glands	forniceal conjunctiva
Krukenburg Spindle	PDG/PXG	pigment deposition on endothelium
Labrador Keratopathy	Spheroidal degeneration	
Langerhans Cell	Antigen Presenting Cell of Ocular Surface	dendritic cells in surface epithelium
Langhans Cell	Giant Cells in Granulomas	horseshoe shaped
Leber's Congenital Amarosis	Marbelized fundus, AR	poor VA, nystagmus, photophobia
Leber's Hereditary Optic Neuropathy	Mitochonial Disease	ONH telangiectasias and edema; WPW; 11778
Leber's Idiopathic Neuroretinitis	Disc Edema + Macular Star	caused by *Bartonella henselae*
Leser-Trelat Sign	Benign pappilomas heralding GI cancer	
Letterer–Siwe disease	Langerhans cell histiocytosis	
Lisch Nodules	NF1	
Lockwood's Ligament	Changes direction of inferior tarsus	
Löfgren Syndrome	Sarcoid + fever, athralgias, EN, hilar adenopathy	and iritis
Louis-Bar Syndrome	Ataxia-telangiectasia	tortuous conj vessels, poor gait, COMA like; phakomatosis
Lowe Syndrome	Familial Oculorenal, + hematuria	lenticonus, microspherophakia
Maddox Rods		for testing cyclotorsion
Marcus-Gunn Jaw Wink	Synkinesis; congenital dysinnervation disorder	ptotic lid elevates in response to voluntary masticatory movements of the jaw; abnormal connection between the levator palpebrae muscle of the ptotic eye and the contralateral lateral pterygoid muscle
Marfan Syndrome	ST subloxation, microspherophakia	thin, slender, tall
Martegiani Area	funnel shaped void of vitreous at the ONH	opening to cloqet canal
Medicomentosa	Pseudopemphigoid	timolol and pilocarpine
Meige's Syndrome	Bilateral blepharospasm + facial spasm	
Melkersson-Rosenthos		
Meretoja Syndrome	Lattice, Gelsolin type	Amyloid deposition in the skin and perineural space leads to dermatochalasis, saggy skin, pendulous ears, and bilateral CN7 palsies. Bloodhound face.

Mikulicz syndrome	Lacrimal and salivary gland enlargement + keratoconjunctivitis sicca	
Millard-Gubler Syndrome	Ipsilateral 6th plus ipsilateral 7th	fascicular syndrome; ventral pons
MISME syndrome	NF2	multiple inherited schwanomas, meningiomas, and ependyomas
Mittendorf's Dot	PFV	hyaloid remnant at posterior lens
Mizuo-Nakamura Phenomenon	Oguchi Disease	after light exposure, posterior pole takes on a yellow iridescent sheen
Möbius Syndrome	Absent CNVI and lacrimal gland	
Moll Glands	Sweat glands (sweaty mole)	eyelash pilosebaceous unit
Moore's Lightening Streaks	Phosphenes	from syneresed vitreous
Mooren's Ulcer	Idiopathic PUK	overhanging edge, sclera not involved,
Morgagnian cataract	End stage cortical cataract	
Morquio Syndrome	Mucopolysaccharidosis	cornea cloudiness at takes 6 weeks to 24 months
Muir-Torre	Colon cancer + eyes	
Müller Cells	Span retina, form limiting membranes	
Müller's Muscle	Smooth muscle for lid retraction	
Munnerlyn Formula	Degree of Myopia in Diopters x (the optical-zone diameter in mm)^2 / 3;	
Munson's Sign	KCN/KGB/PMD	distortion of eyelid on downgaze
Muscle of Riolin	Grey Line	orbicularis at lid margin
Nettleship-Falls Albinism	Ocular Albinism	nystagmus, photophobia, poor central vision
Nevus of Ota	Ocular melanocytosis + hyperpigmentation of the periorbital skin; iris mammilations	
Norrie Disease	X-linked blindness, deafness	yellow RD, pthisis by 10; vitreoretinopathy
Nothnagel Syndrome	Ipsilateral 3rd plus ipsilateral ataxia	fascicular syndrome; cerebeller peduncle
Oguchi Disease	Rod CSNB	
Ohno's Sign	VKH (convalescent)	TM depigmentation
Pancoast Tumor	Mass near the superior sulcus (=apex) of the lung	pre-ganglionic Horner's
Panum's Area	Area around empirical horopter, stereopsis	
Parinaud Syndrome	Dorsal Midbrain/Pretectal Nuclei Syndrome	impaired upgaze, retraction convergance nystagmus, light-near, lid retraction
Parinaud's oculoglandular syndrome	Conjunctivitis + lymphadenopathy	caused by *Bartonella hensalae;* follicular conjunctivitis; also syphillis, TB, chlamydia, tuleremia
Parks-Bielschowsky 3-step test	Determine hyper eye (cover/uncover), identify gaze that worsens, identify head tilt that worsens	
Parks-Weber Syndrome	Nevus Flammeus	

Paton Lines	Peripapillary straie from disc edema	
Peter's Anomaly	Neurocristopahy; ASD	PAX6; corneal opacity, vascularized lens, cataract, strands from lens to cornea; no descemet's
Perkinje Effect	Peak luminance sensitivity of the eye to shift toward the blue end of spectrum during dark adaptation	
Pfeifer Syndrome	Constant XT; craniosyostosis facies + big thumbs	V pattern; exorbatism
Posner-Schlossman	Glaucomatocyclitic crisis	periodic, painless spikes in IOP
Prentice's Rule	PD = hD	
Proteus Syndrome	Nevus Flammeus	
Pulfrich Phenomenon	Objects moving towards view seem to move perpendicular	
Purtscher Flecken	Purtscher's Retinopathy	Like CWSs but deeper, doesn't obscure vessels
Quickert-Rathbun Sutures	A suturing technique that everts an entropic lid	
Randleman Criteria	Predict ectasia risk after LASIK	
Rayleigh Effect		
Raymond-Castan Syndrome		
Reis-Bückler	Epithelial-stromal TGFBI dystrophy	
Riddoch Phenomenon	Can see moving targets in cortical blindness	
Riley-Day Syndrome	Familial Dysautonomia	keratitis ulcers
Riolin Muscle	Gray Line	special slip of pretarsal orbiculari on surface of lid margin
Rizzuti's Sign	KCN	A cone-shaped reflection that appears on the nasal side of the cornea when a light is shone from the temporal side
Roth Spots	Childhood ALL, endocarditis	white-centered RNFL lesions
Rush Disease	ROP	Zone 1 + Plus disease
Saethre-Chotzen	Craniosyostosis	TWIST gene
Salzmann Nodular Degeneration	Post-inflammation/corneal scar	blue white nodule midperiphery of cornea
Sampolosi Line	PXG/PDG	pigment anterior to Schwalbe's
Sands of Sahara	Deep Lamellar Keratitis	noninfectious inflammation of the flap-bed interface, LASIK complication
Sattler's Layer		
Sattler's Veil	Central corneal edema associated with hard contact lens use (build up of CO2)	
Scheie Stripe/Zentmeyer Line	PDG	pigment antiorior to zonules
Scheie Syndrome	Mucopolysaccharidosis	cornea cloudiness at takes 6 weeks to 24 months
Schnabel Cavernous Optic Atrophy	Mucopolysaccharidosis	Arteroscxlerosis
Schynder Corneal Dystrophy	Stromal dystrophy, lipid metabolism	opaque disc or crystals

Schwartz Syndrome	RRD	photoreceptors clogging TM
Seidel Testing	Wound leak	
Senior-Løken Syndrome	RP fundus, progressive renal failure	cilliopathy; JABS
Shafer's Sign	Retinal Break	
Sherington's Law	Of Recipricol Innervation	Increased innervation to a muscle is accompanied by decreased innervation to its antagonist
Siegrest Streaks	Hypertensive Choroidopathy	hyperpigmented streaks
Soemmering ring	Wedl/Bladder cells in ring around bag	peripherlaly migrated after phaco
Sorby's Macular Dystrophy	AD; drusen-like deposits	bilateral subfoveal CNVMs at age 40; consider Stargardt, Best, Adult-Onset Vitelliform, Pattern, Central Areolar, North Carolina
Spiral of Tillaux	Muscle Insertion	5.5-6.5-6.9-7.7 for M-I-L-S
Stargardt Disease	Pisciform lesions, AR, ABCA4, A2E buildup	dark choroid FA, bull's eye FAF
Stevens-Johnson syndrome (SJS)	Erythema Multiforme Major	severe cases are toxic epidermal necrolysis (TEN); Nsaids, Antiepileptics, Sulfonamides, Allopurinol
Stickler Syndrome	Hereditary Hyaloideoretinopathy	optically empty vitreous, Pierre Robin sequence; AD
Still Disease	Nongranulomatous Chronic Anterior Uveitis	form of JIA
Still's Disease	JIA variant	
Stocker's Line	Pterygium	iron line
Sturge-Weber Syndrome	Encephalotrigeminal angiomatosis	port wine stain; elevated EVP; diffuse choroidal hemangioma (ketchup fundus)
Sugiura Signs	VKH (convalescent)	perilimbal depigmentation
Susac Syndrome	SICRET; Autoimmune Encephalopathy in women	bilateral BRAO, hearing loss, encephalopathy; Small Infarctions of Cochlear, Retinal, and Encephalic Tissue; Occlusive retinal vasculitis; boxcarring
Taches de Bougie	Sarcoidosis	candle wax lesions on retina
Tangier disease	Cloudy cornea, α-lipoprotein deficiency	like LCAT or Fish Eye
Teller Card	Card with stripes, preferential looking	
Terrien's Marginal Degeneration	Noninflammatory peripheral cornea thinning	corneal neovascularization, opacification and lipid deposition
Terson Syndrome	Sub-ILM, sub-hyaloid, or intra-vitreal	2ndry to abrupt intracranial hemorrhage
The (spiral) Valve of Hasner	NLDO	distal valve that usually causes NLDO
Thiel-Behnke	Epithelial-stromal TGFBI dystrophy	5q31

Thygeson Superficial Punctate Keratitis	Multiple Discrete Punctate Lesions	epithelial; pain out of proportion
Tolosa-Hunt Syndrome	Painful ophthalmoplegia secondary to noninfectious inflammation of the cavernous sinus	
Touton Giant Cells	JXG	
Touton giant cells	Juvenile Xanthogranuloma	presents with skin papules and heterochromia
Treacher Collins Syndrome	Non-Craniosyostosis; downsloping orbits	mandibular arch like Goldenhar
Tyndall Effect	Scattering of light in colloidal substance	
Uhtoff's Phenomenon	MS	transient vision obscurations with heat
Urrets-Zavalia Syndrome	Fixed and dilated pupil following	penetrating keratoplasty (PKP) 2/2 ischemia
Vernier Acuity	Misalignment between lign segments	
Vieth-Müller Circle	Set of points that stimulate corresponding areas	of retina
Virchow's Law	Craniosyestosis	bone growth parallell to the fused plates instead of perpendicular
Vogt Striae	KCN	vertical lines in deep stroma
Vogt-Koyanagi-Harada	Granulomatous panuveitis	CNS (tinnitis), then serous RDs/disc edema, then depigmentation; consider SO
Vogt's Pallasades		
Vogt's Triad	Epiloia, Tuberous Scleoris	epillepsy, low intelligence, angiomas
von Graefe’s sign	TED	lid lag
von Hippel Lindau	Retinal Angiomatosis, a Phakomatosis	capillary hemangioma; large feeder vessels
von Recklinghousen	NF1, phakomatosis	lisch nodules, cafe au lait, plexiform neurofibromas
Vossius ring	Blunt trauma cataract	ring of pupillaryruff pigment on the anterior capsule
Waardenburg Syndrone	Heterochromia, Dystopia Canthorum	white forelock
Wagner Disease	Hereditary Hyaloideoretinopathies with Optically Empty Vitreous	
Wait-Beetham Lines	Deep Stromal Wrinkles	corneal edema
Wallenberg Syndrome	Lateral Medullary Syndrome	central horner's; loss of pain and temperature sensation to the ipsilateral face and contralateral body
Watze-Allen Test	Macular Hole	slit beam over macula, if patient reports a break in line = full thickness hole
Weber Syndrome	Ipsilateral 3rd plus contralateral hemiplegia	fascicular syndrome; cerebral peduncle
Wedl Cells	Bladder Cells	swollen lens epithelial cells that have migrated posteriorally to the posterior capsule in a subcapsular cataract
Weiger’s Ligament	Attachment between the anterior vitreous and the posterior lens capsule	

Weill Marchesani Syndrome	Microspherophakia	think opposite of Marfan
Weiss ring	Ring shaped Floater after PVD	
Whitnall's Ligament	Changes direction of levator	
Whipple's Disease	Adult xanthalasma, nystagmus, GI	
Wilm's Tumor	Nephroblastoma	WAGR complex
Wilson's Disease	Hepato-lenticular Degeneation	KF rings and Sunflower cataracts
Wofflin Nodules	Brushfield Spots in non-Down	
Wolfram Syndrome	DIDMOAD Syndrome	**DI**, **DM**, **O**ptic **A**trophy, **D**eafness
Wolfring Glands	Accessiry lacrimal glands	palpebral conjunctiva
Wyburn-Mason syndrome	Racemose Angioma	
Zeiss Glands	Sebaceous (zebaceous)	eyelash pilosebaceous unit
Zellweger Syndrome	Cerebrohepatorenal syndrome	hypotony, deafness, RP fundus, seizures, abnormal face; AR
Zernike Polynomials	A mathematical system for describing and systematizing optical aberrations	

Common Abbreviations

Abbreviation	Meaning	Definition
AC, A/C	anterior chamber	The space from iris to posterior cornea
ACIOL	anterior chamber intraocular lens	An IOL placed in front of the iris
AK	actinic keratosis	A precancerous skin lesion
AL	axial length	Length of eye, normally ~23mm
AMD/ARMD	age-related macular degeneration	Loss of the highest acuity portion of the retina
APD	afferent pupillary defect	Pupils do not constrict equally to light
ASA	aspirin (acetylsalicylic acid)	Weak anti-coagulant
AT	applanation tonometry/artificial tears	i.e. Goldmann applanation
BC	base curve	Measure of curvature for contact lens
BCC	basal cell carcinoma	A type of skin cancer
BID/b.i.d	twice a day (*bis in die*)	e.g. drops 2x/day
bil/BIL	bilateral	Both sides
BCL	bandage contact lens	A large contact to protect the cornea
BSS	balanced salt solution	Well tolerated solution for the eyes
BTX	botulinum toxin (Botox)	Muscle paralyzing compound
cat	cataract	Opacification of the lens
cc	with correction (*cum correctio*)	Visual acuity with glasses/contact lenses
CC	chief complaint/cortical cataract	
c/d	cup-to-disc ratio	Normally 0.3, increases with glaucoma
CE	cataract extraction	Removal of opacified lens
CF	confrontational field/counts fingers	Holding fingers in peripheral vision/Vision so poor they can only count fingers
CHRPE	congenital hypertrophy of the retinal pigment epithelium	A usually benign condition before age 30
CL	contact lens	Lens on corneal surface to correct vision
cm	centimeter	A little under ½ inch
CME	cystoid macular edema	Swelling of the macula
CN	cranial nerve	One of the major brainstem nerves
II	2nd cranial nerve (optic)	Caries all information
III	3rd cranial nerve (oculomotor)	Responsible for most eye movements
IV	4th cranial nerve (trochlear)	Responsible for looking at the nose
V	5th cranial nerve (trigeminal)	Responsible for sensation of the face
VI	6th cranial nerve (abducens)	Responsible for looking to the side
VII	7th cranial nerve (facial)	Responsible for motion of the face
c/o	complains of	Patient's primary concern
CT	computed tomography	Like a 3D x-ray
CVA	cerebrovascular accident, stroke	Region of brain is starved of oxygen
CWS	cotton-wool spots	Usually due to diabetes or hypertension
DC, d/c	discontinue	Stop taking
DES	dry eye syndrome	Chronic sensation of eye dryness
DFE	dilated fundus exam	Eye is dilated for visualization of retina
DKA	diabetic ketoacidosis	A serious acute manifestation of diabetes

DME	diabetic macular edema	Swelling of the macula due to diabetes
DMEK	descemet membrane endothelial keratoplasty	Transplant of only part of the cornea
DNR	do not resuscitate	An end of life wish
DOB	date of birth	Birthday
DSAEK	descemet stripping automated endothelial keratoplasty	Transplant of only part of the cornea
DSEK	descemet stripping endothelial keratoplasty	Transplant of only part of the cornea
Dx	diagnosis	Established diagnosis
Ddx	differential diagnoses	Diagnoses to still consider
Dz	disease	
EOM	extraocular muscles	The muscles that control eye/eyelid movement
EOMI	extraocular muscles intact	Patient can move eyes normally
FB	foreign body	Something that shouldn't be in the eye
f+f	fix and follow vision	Babies can't read charts but being able to fixate on a target and follow it can indicate vision
FH	family history	Diseases that run in the family
f/u	follow up	Either for chronic condition or after surgery
GCA	giant cell arteritis	Rare inflammatory condition of blood vessels
GCL	ganglion cell layer	A layer of the retina
GVF	Goldmann visual field	Visual field testing for peripheral vision function
HA	headache	
HCL	hard contact lens	Non-flexible contact lens
HM	hand motion	Very poor vision only able to perceive motion
h/o	history of	
HPI	history of present illness	History pertaining to their primary concern
HSK	herpes simplex keratitis	Herpes infection of the cornea
HSV	herpes simplex virus	A sexually transmitted infection
HTN	hypertension	High blood pressure
HVF	Humphrey visual field	Visual field testing for peripheral vision function
hx	history	
HZ	herpes zoster	A sexually transmitted infection
HZO	herpes zoster ophthalmicus	Shingles around the eye
IOL	intraocular lens	Artificial lens placed after cataract surgery
IOP	intraocular pressure	Pressure inside the eye
Ish.	Ishihara Color Plates	Test for color blindness
IV	intravenous	Injection into the vein
JRA	Juvenile Rheumatoid Arthritis	An inflammatory joint disorder in children (can be associated with inflammation of the eye)
KC	Keratoconus	A cone shaped cornea
LLL	Left lower eyelid	
LOC	loss of consciousness	
LP	light perception	Very poor vision only able to perceive light
LPI	laser peripheral iridotomy	Emergency glaucoma procedure

LRI	limbal relaxing incisions	Procedure to correct astigmatism
LUL	left upper eyelid	
LVA	low vision aids	
MRx	Manifest Refraction	Refraction using the phoropter
MVA/MVC	motor vehicle accident, motor vehicle crash	
NFL	nerve fiber layer	A layer of the retina
NKA/NKDA	no known allergies/ no known drug allergies	
nl	normal	
NLD	nasolacrimal duct	Tear duct
NLDO	nasolacrimal duct obstruction	Obstruction of the tear duct
NLP	no light perception	Complete blindness
n.p.o/NPO	nothing by mouth (*nil per os*)	It's important to stop eating before surgery
NS/NSC	nuclear sclerosis (cataract)	A type of cataract
NSAID	nonsteroidal anti-inflammatory drug	e.g. ibuprofen, naproxen, acetaminophen
OCT	Optical Coherence Tomography	Noninvasive imaging of the back of the eye
OD	right eye (*oculus dexter*)	
ophth.	ophthalmology, ophthalmic, etc.	
OR	operating room, over-refraction	
ORA	ocular response analyzer	Test for glaucoma progression
OS	left eye (*oculus sinister*)	
OU	both eyes (*oculi uterque*)	
PC	posterior capsule	The back portion of the bag the lens sits in
PCH	posterior capsule haze	Hazing of the back portion of the lens bag
PCIOL	posterior chamber intraocular lens	Lens placed in the position of the natural lens
PCN	penicillin	Antibiotic
PCO	posterior capsular opacification	Opacification of the back portion of the lens bag
PD	pupillary distance	Distance between the two pupils
PERRLA	pupils equal, round, reactive to light and accommodation	Indicates healthy nerve function
PF	palpebral fissure/PredForte	Open space between eyelids/Steroid
PFAT	preservative free artificial tears	Artificial tears without preservatives
pH	acidity/alkalinity of a solution	Acid has a low pH, bleach has a high pH
PH	past history/pinhole (visual acuity)	Medical history/Vision through a small hole
Phaco	phacoemulsification	Removal of natural lens using ultrasound energy
PHNI	pinhole no improvement	Vision does not improve when looking through a small hole, indicates problem is not refractive
prn	as needed (*pro re nata*)	e.g. take artificial tears when eyes feel dry
PMH	past medical history	
p.o	by mouth (*per os*)	
POP	post-operative	After surgery
POAG	primary open angle glaucoma	A type of glaucoma
pt	patient	
QD/q.d	every day/once a day (*quaque die*)	

QH/q.h	every hour (*quaque hora*)	
QHS/q.h.s	every bedtime (*quaque hora somni*)	
QID/ q.i.d	four times a day (*quater in die*)	
q3h/ Q3H	every 3 hours (*quaque 3 hora*)	
RA	rheumatoid arthritis	An inflammatory joint condition
Ref	refraction	Determining prescription to correct vision
RLL	right lower eyelid	
RNFL	retinal nerve fiber layer	A layer of the retina
r/o	rule out	A disease that must be considered
ROS	review of systems	Asking about health of other organ systems
RP	retinitis pigmentosa	Genetic disorder that can cause blindness
RPE	retinal pigment epithelium	A layer of the retina
RTC	return to clinic	
RUL	right upper eyelid	
sc	without correction (*sine correctio*)	Visual acuity without glasses/contact lenses
SCC	squamous cell carcinoma	A type of skin cancer
Sph	sphere	Correction for the non-astigmatism portion of the refractive error
SPK	superficial punctate keratitis	Corneal problem that can cause reduced vision
s/s	signs and symptoms	
Sx	surgery; symptoms	
TB	tuberculosis	A rare but serious infection (usually of lungs)
TBT/ TBUT	tear break-up time	Assesses composition of tears, <10s in dry eye
TED	thyroid eye disease (Graves Disease)	Causes eyes to protrude from effect of hormone
t.i.d/TID	three times a day (*ter in die*)	
U/A	urinalysis	
UL	upper lid	
ULC	upper lid crease	
US	ultrasound	
UV	ultraviolet	Light above the visual spectrum
VA	visual acuity; vision	Ability to see, smallest readable line on chart
VAcc	visual acuity with refractive correction worn	Visual acuity with glasses/contact lenses
VAsc	visual acuity without refractive correction	Visual acuity without glasses/contact lenses
VF	visual field	The normal view of vision
WBC	white blood cell	Responsible for fighting infections
WC	warm compress	
YAG	yittrium-aluminum-garnet laser	For treating PCOs
y/o	years old	