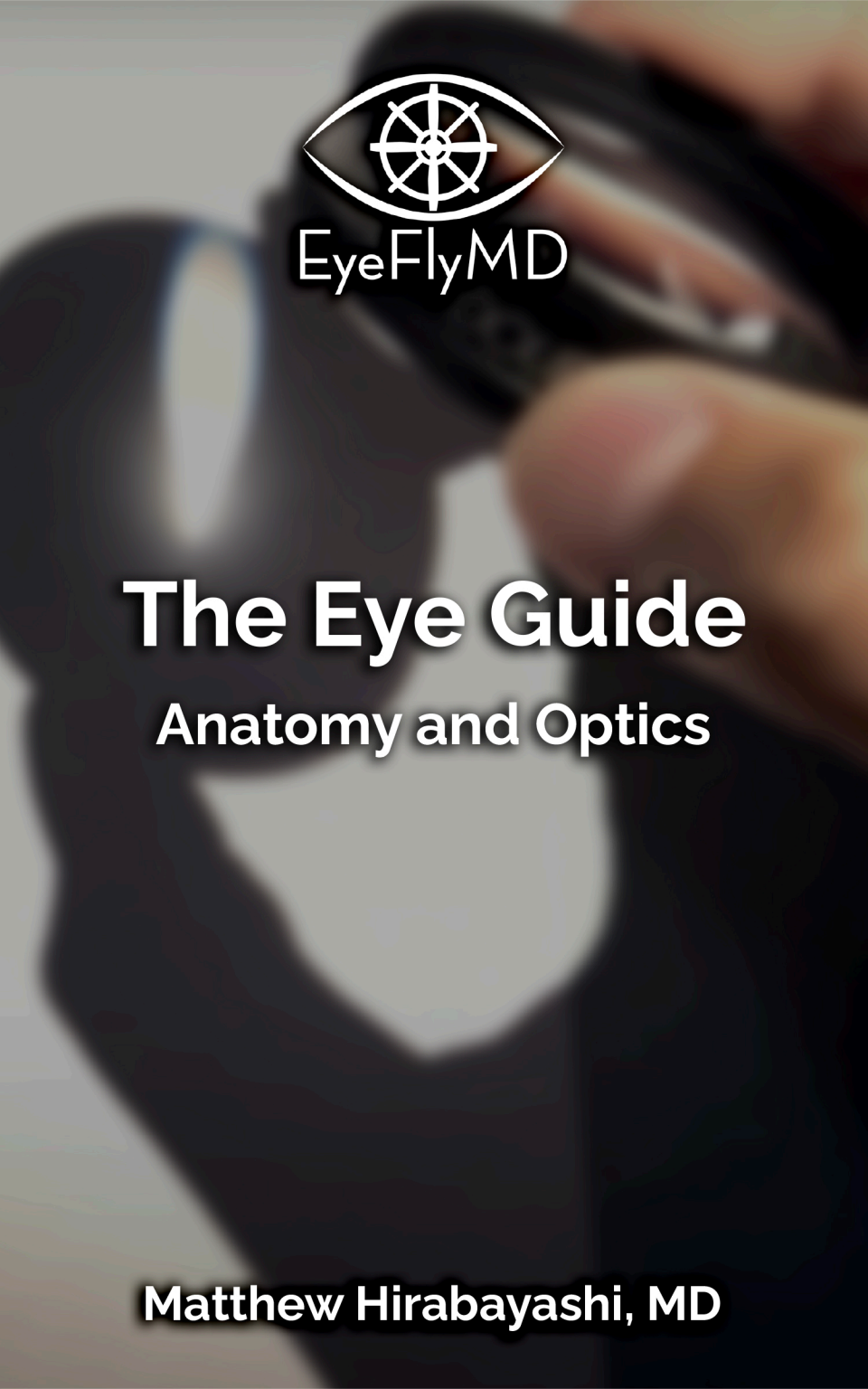
EyeFlyMD
The Eye Guide
Anatomy and Optics
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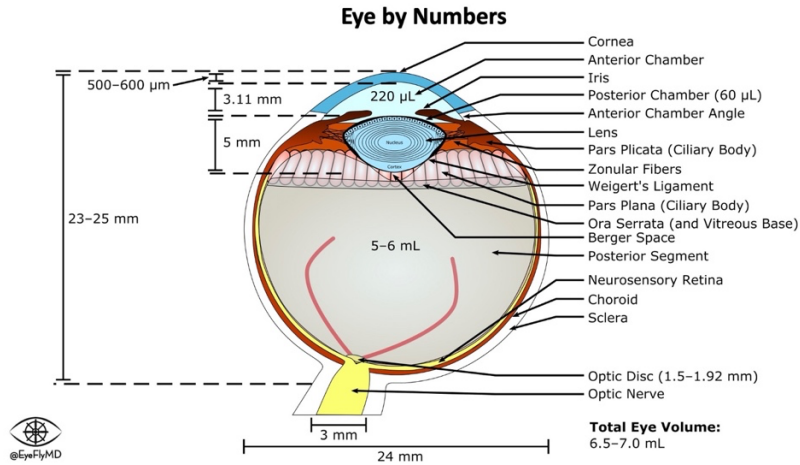
Proofread, fact-checked, and edited by Ariess Gharabagi and Travis Parker MD

Disclaimer: This writing and all the figures are my own and based on knowledge I've accumulated through various resources thus far in my ophthalmology residency training. I don't provide citations but encourage every reader to consult the relevant literature/texts for their program and/or training level. Don't take my word for it. The purpose of this work is to provide a concise summary of critical topics. This work contains no medical advice. Please submit errors to matt@eyeflymd.com

ALL PHOTOGRAPHS AND DIAGRAMS ARE ORIGINAL

Anatomy, Terms, and Basic Concepts

Anatomy Overview



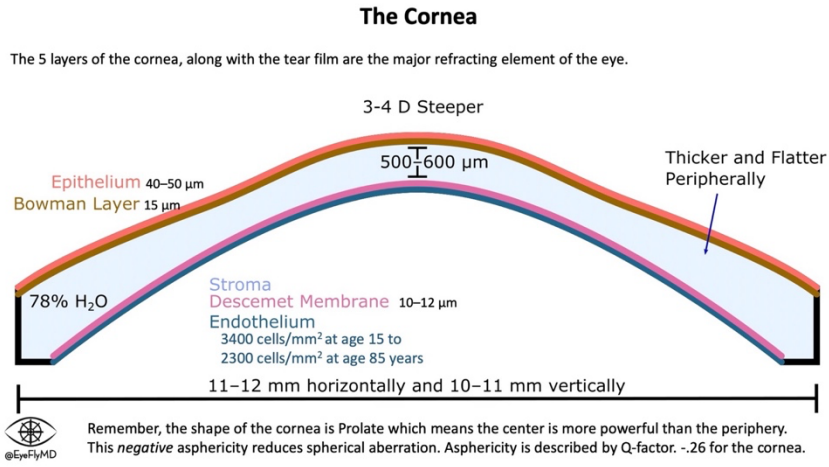
- **Conjunctiva** – Clear, thin epithelial membrane that covers the inner eyelids (palpebral conjunctiva) and surface of the eye (bulbar conjunctiva) continuously to the limbus (sclera-cornea interface). Because of the conjunctiva, it is impossible for contact lenses to "get lost" behind the globe.
- **Cornea** – The clear portion in the front of the eye. The limbus delineates the cornea from the surrounding sclera. Responsible for 2/3 to 3/4 (~43D) of the eye's refractive power.
- **Iris** – Pigmented structure that acts as an aperture to control the amount of light entering the eye.
- **Lens** – Transparent, biconvex structure. Responsible for 1/3 (~20D) of the eye's refractive power.
- **Zonular Fibers** – Connects the lens to ciliary body, supports the lens and helps 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. The muscle contracts to assist in accommodation and the processes (outpouchings) produce aqueous humor by active secretion at a rate of 2-3µL/min. There are around 80 ciliary processes.

The pars plana portion of the ciliary body is the posterior, flat (planar) part that meets the retina. Because there is no neurosensory retina in this area it is where trocars are placed for a Pars Plana Vitrectomy.

- **Anterior Chamber** – Aqueous-filled space between the corneal endothelium and iris. Volume: ~200 µL.
- **Posterior Chamber** – Aqueous-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 posterior lens capsule including the vitreous and retina.
- **Uvea** – The structure composed of the iris, choroid, and ciliary body. From the Greek for "grape" due to its deep violet color.
- **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 inner to the sclera and blood supply for the outer retina. The inner retina (from ~the inner nuclear layer) 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"

Cornea Anatomy



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's 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 the 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's 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.

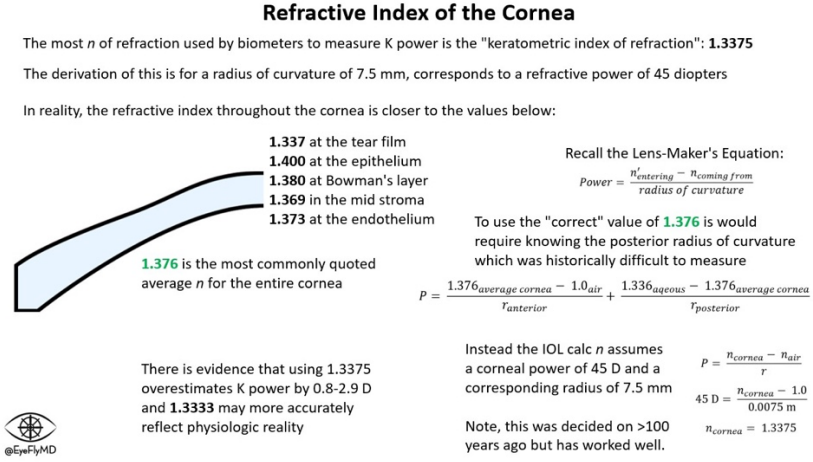
We are born with a central endothelial cell density of ~ 5,000 cells/mm^2. The cell density decreases ~ 0.6% per year with landmarks of 3,400 cells/mm^2 at age 15 and 2,300 cells/mm^2 at 85. At any stage of life 2,000 – 3,000 cells/mm^2 is a reasonable normal. < 500 cells/mm^2 risks corneal edema as 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) 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 its 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 1.3375 when using standardized corneal radius-of-curvature and dioptric power measurements. This is due to how difficult the posterior cornea was to measure.

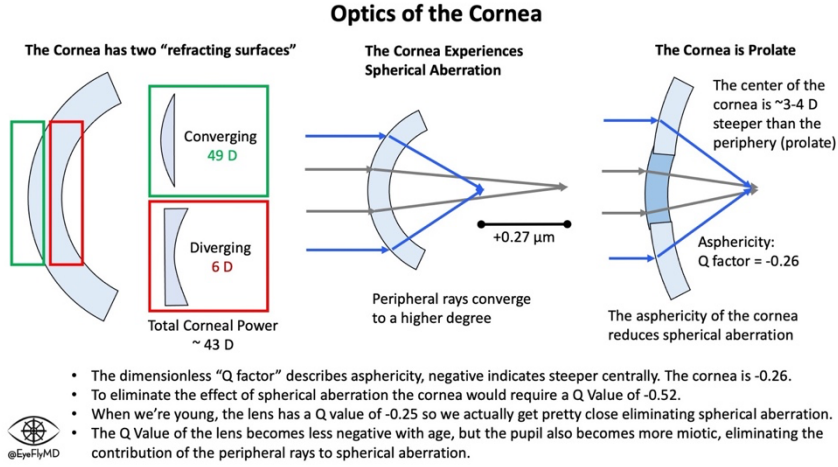


The greatest change in refractive indices occurs when light crosses from air (n ~1.00) to the tear film (n ~ 1.34) so that is where most of the refracting magic happens.

The cornea also has some other important optics. The total refractive power of the eye is ~60 D and the cornea contributes about 2/3 to this. The front of the cornea adds about 49 D of convergence to the system (since it is a convex surface) and the posterior cornea adds about 6 D of divergence (since it is a concave surface) for a total power of ~43 D. Near the periphery of a spherical system, light rays with no vergence strike the refracting surface at a sharper angle and thus are refracted more.

This results in *spherical aberration*. The average eye has often reported an average of +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.*

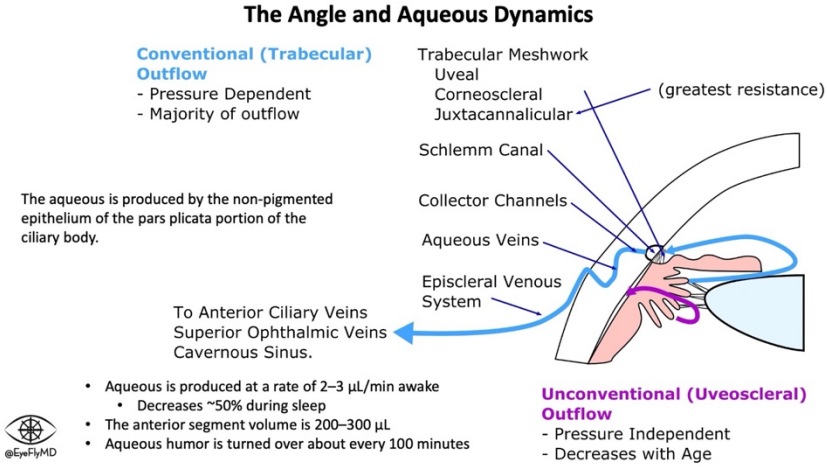
The term *prolate* describes when optical surfaces are steeper centrally than peripherally (or flatter peripherally than centrally). This is negative asphericity and is indicated by negative Q factors. An *oblate* cornea is flatter centrally than peripherally (e.g., after myopic refractive surgery) and 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.



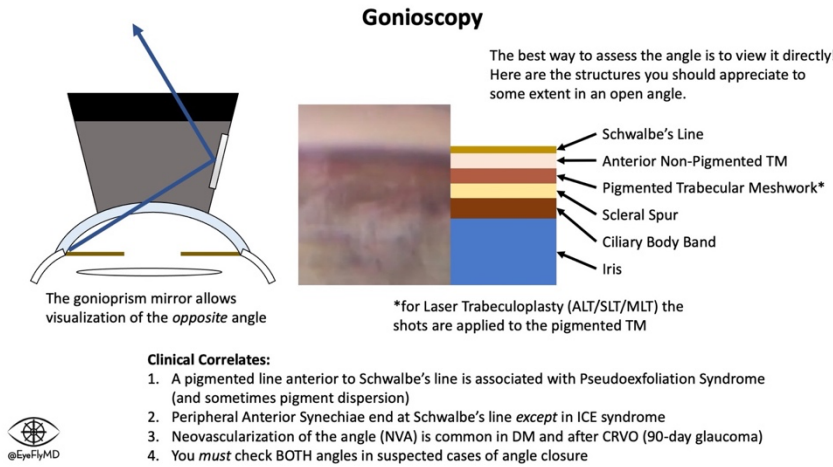
- The dimensionless "Q factor" describes asphericity, negative indicates steeper centrally. The cornea is -0.26.
- To eliminate the effect of spherical aberration the cornea would require a Q Value of -0.52.
- When we're young, the lens has a Q value of -0.25 so we actually get pretty close eliminating 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.

Angle Anatomy 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 trabecular meshwork, specifically the juxtacanalicular portion, is the site of greatest outflow resistance.



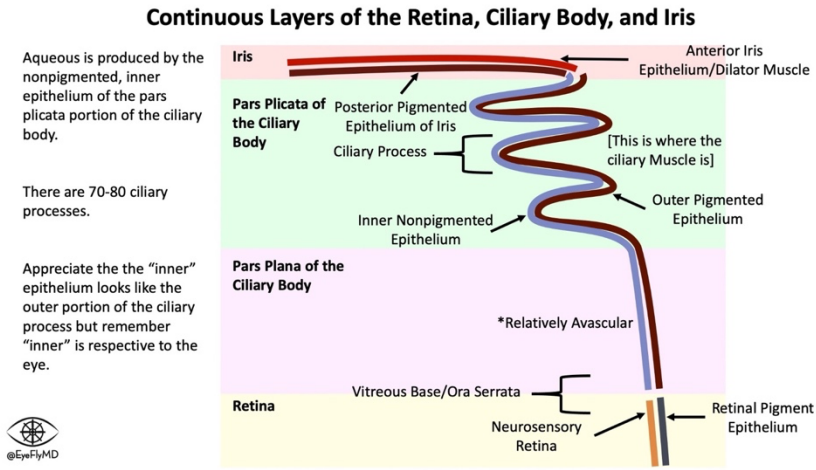
One of the most important tools in glaucoma is gonioscopy. This is visualization of the angle using a lens. The most common types of gonioprisms use mirrors. It can be hard to see all the structures even if the angle is wide open but with practice the major structures can be identified.



Gonioscopy is a good place to look for a Sampaolesi Line which is a pigmented line anterior to Schwalbe's line. This finding is associated with Pseudoexfoliation Syndrome.

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.

The "inner" language with respect to the eye may become confusing. Always think of a theoretical point at the exact center of the eye. The "inner" portion of any structure will always be whatever is closest to this theoretical point regardless of its orientation to the rest of the structure.



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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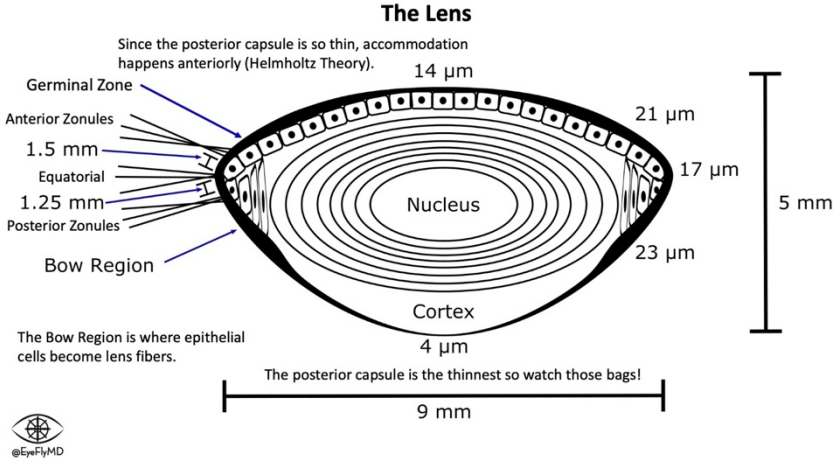
Lens Anatomy

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. The presence of excessive glucose though (e.g., as in diabetes) or else the enzyme *aldose reductase* will convert the excess glucose to sorbitol and precipitate a diabetic cataract.



Vitreous Anatomy

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 hyalocyte cells that help support the vitreous. They derive their oxygen from choroidal diffusion.

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

The vitreous has five major attachments to the rest of the eye.

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, some textbooks are at best vague and at worst incorrect. The phraseology and figure presentation of some texts 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 speaking to a few retina specialists 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 being aware of this discrepancy.

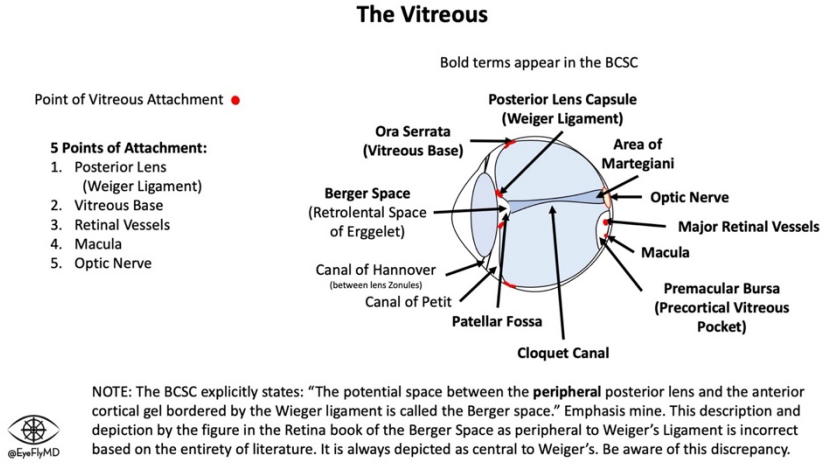
2. **Vitreous Base**

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 the strongest 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**

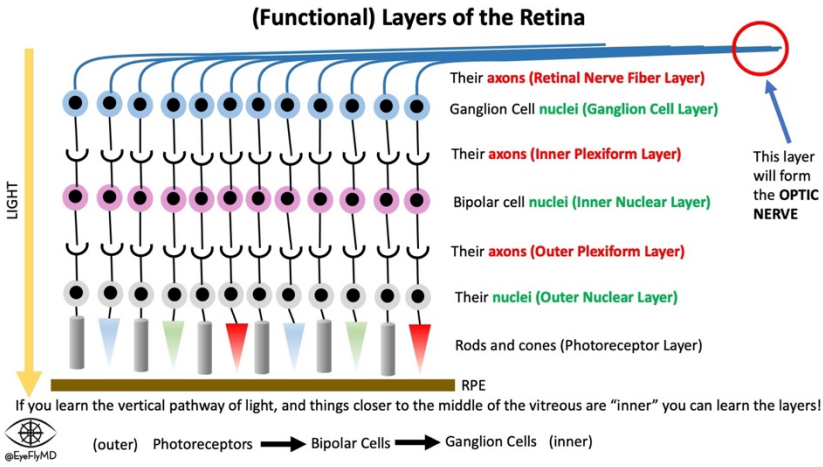
Posteriorly, the vitreous is attached to the internal limiting membrane of the neurosensory retina at the retinal vessels, optic nerve, and macula by fibronectin and laminin adhesion molecules. Anterior to the macula, there is an area of liquified vitreous called the *premacular bursa* or *precortical vitreous pocket*. The purpose of this area of liquified vitreous is to minimize tractional forces in the 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 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.

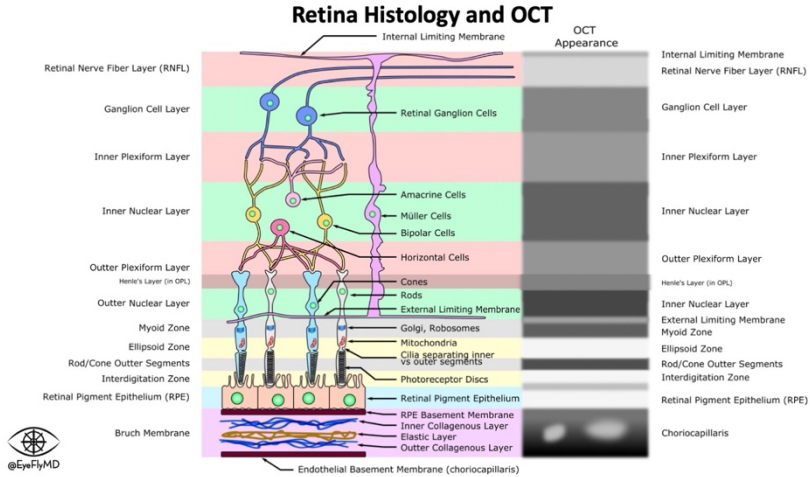


Retina Anatomy

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.

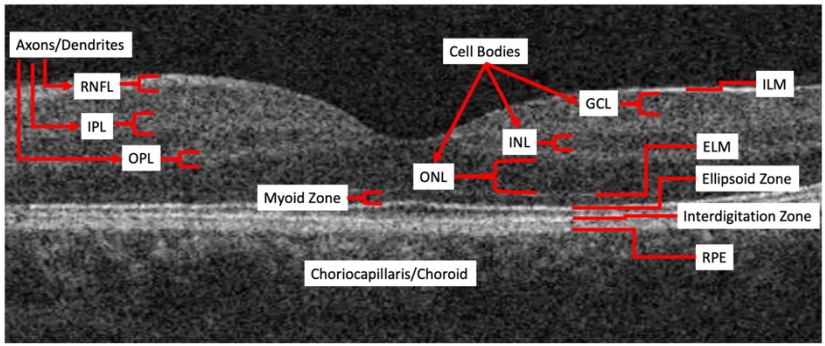


Here is a more detailed look at retina histology and how it compares to OCT. You'll notice the inner and outer photoreceptor segments are connected by cilia. You may remember the 9+2 configuration of cilia from medical school, but these cilia are 9+0 as they're nonmotile.



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. Again, appreciate that the *inner* layers are closest to that hypothetical geometric center of the entire eye.

Retina OCT



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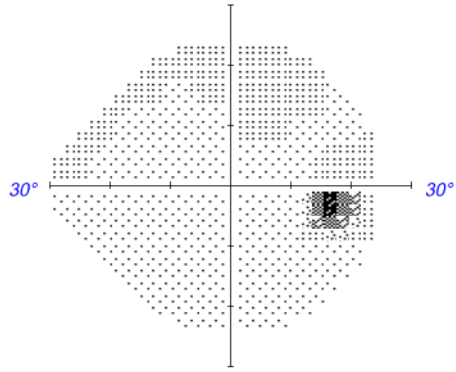
ILM – Internal Limiting Membrane (formed by Müller cell foot processes)
RNFL – Retinal Nerve Fiber Layer (axons of Ganglion cells)
GCL – Ganglion Cell Layer
IPL – Inner plexiform layer (axons of INL and dendrites of GCL)
INL – Inner Nuclear Layer (cell bodies of Bipolar cells, Müller cells, Horizontal 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 cell-Müller cell and Müller cell-photoreceptor junctions)
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 (outer blood-retina barrier)
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 personally obtained a visual field from 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 from the cellular level, 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 $\geq$ 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 providing the highest acuity vision.

Anatomy of the Macula

Macula	Between the Arcades	5.5 mm	GCL $\geq$ 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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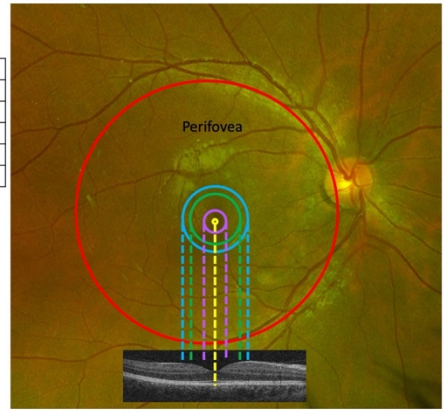
*Foveal avascular Zone, where the inner retina blood supply (from central retinal artery) disappears

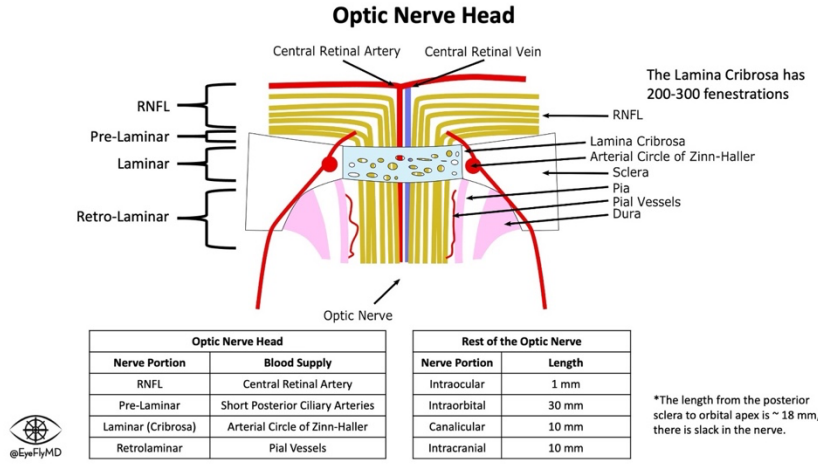


Optic Nerve Anatomy

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 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 after pruning. The lamina cribrosa is the fenestrated portion of the inner 1/3 sclera through which the fibers that form 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.

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). As the central fibers die, they will appear to “plunge” into the sclera further and further from the center, enlarging the C/D.



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.

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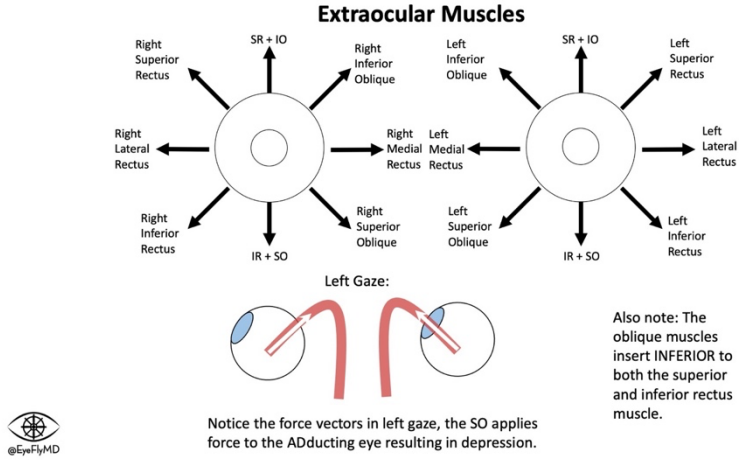
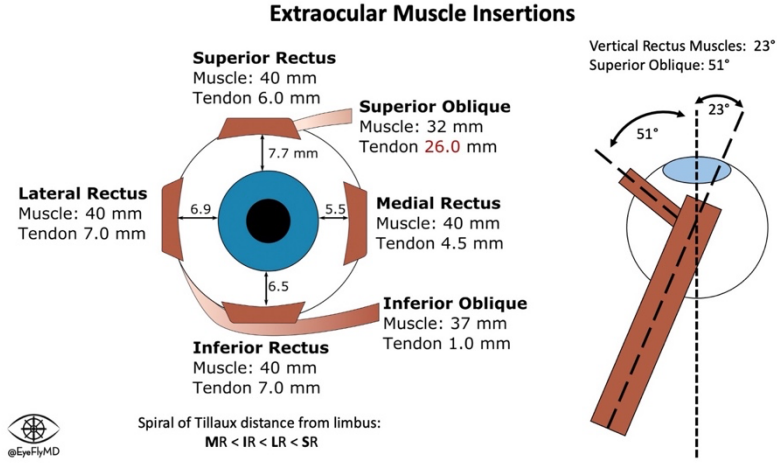
Extraocular Muscles

Let's exit the eye now 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 the lateral recti are instead innervated by CNIII. On attempted ADDuction, both the medial and lateral recti contract which can result in retraction of the globe into the orbit.

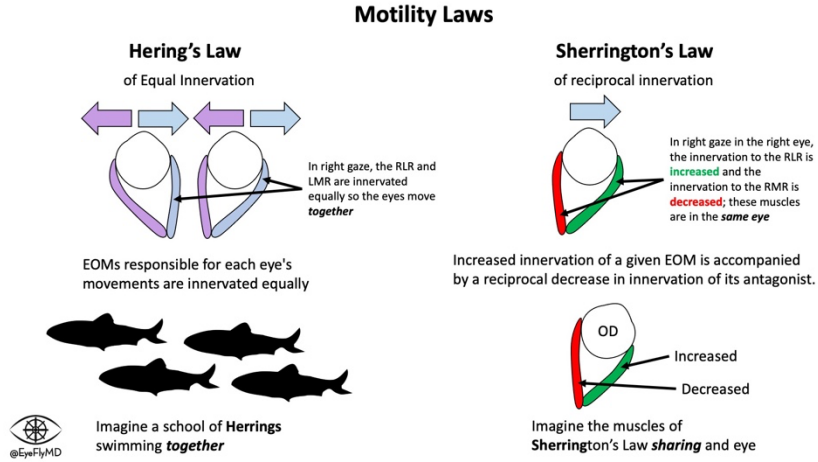
From the figure below, we can appreciate the simple "up" and "down" gaze require the input of two muscles. This is why having patients follow your finger in an "H" pattern is effective; it isolates each muscle's function. The oblique muscles are the primary cyclotorsional (eye rotating around the pupillary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



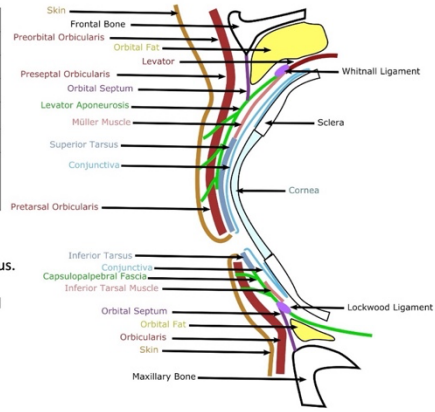
The Eyelids

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.

Glands

- Meibomium – Oil/Meibum – Tarsus
- Zeis – Oil/Sebum – Follicles
- Goblet – Mucus – Conjunctiva

→ **Holocrine** *mature cell dies*

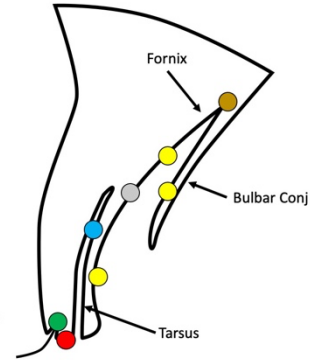
- Moll – Sweat – Eyelid Margin

→ **Apocrine** *part of cell pinches off*

Accessory Lacrimal

- Wolfring – Aqueous – Tarsal Border
- Kraus – Aqueous – Fornix

→ **Exocrine** *secretion*



Picture a **WOLF**(ring) stalking the border of the tarsus

Think of KRAUching in the fornix (Kraus)

Think of a sweaty **MOLE**

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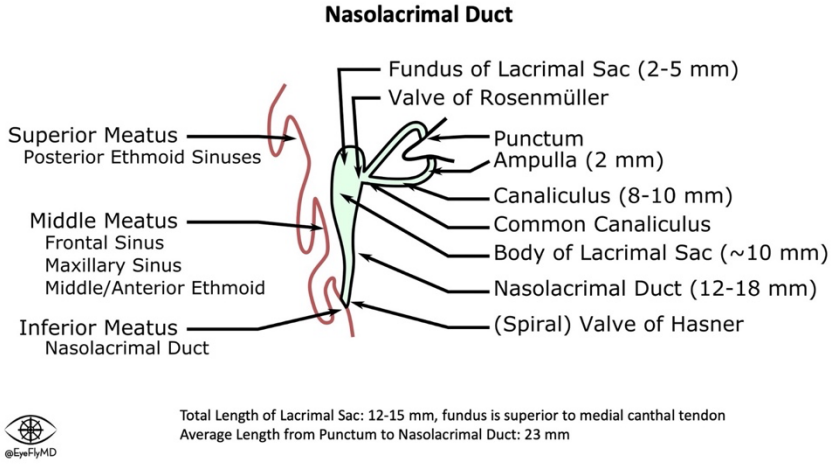
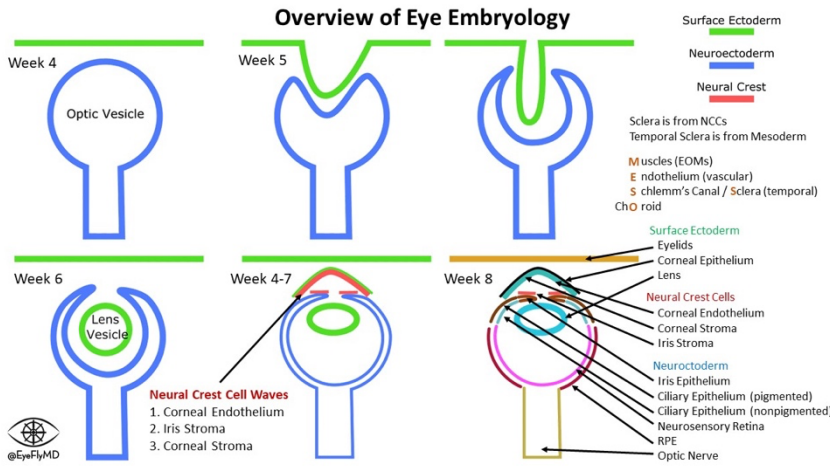
Part of the complexity of the eyelids is the nasolacrimal system. There is a superior and inferior punctum that can both be occluded with plugs as a treatment for Dry Eye Disease (DED). The puncta open to an ampulla and then a canaliculus which drains into the lacrimal sac via the *Spiral Valve of Hasner*. 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.



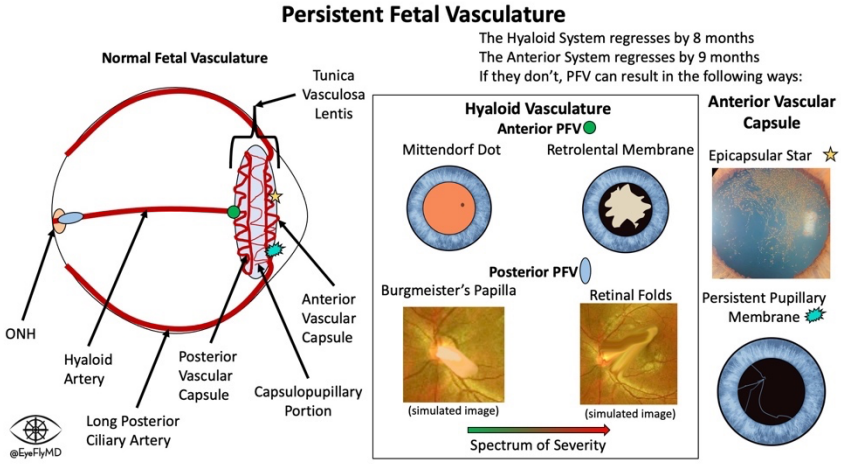
Embryology

Where did all this come from? The figure below is an overview from the 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.



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, these all form 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 are rarely clinically significant.



At birth, newborns have about +3.00 D of hyperopia which increases for a few months before decreasing to ~ +1.00 at the first year of life. The average axial length at birth is 14.5 – 15.5 mm. It increases ~ 4 mm during the first 6 months and ~ 2 mm more the rest of the year. Most of the axial length change is early. Final axial length is achieved by age 13 – 16. Remember, final axial length is usually 23 – 24 mm. The cornea loses ~ 0.1 D – 0.2 D and the lens loses ~1.8 D during age 3 – 14.

Anophthalmia describes complete absence of eye tissue. Microphthalmia describes a small, disorganized eye. Nanophthalmos describes small eye (axial length $\leq$ 18 mm) with high hyperopia.

Basic Optics and Refractive Principles

Important Refractive Concepts

Visual Acuity (VA)

Visual Acuity (VA) is essentially a measure of visual clarity. It is tested one eye at a time. There are different ways to measure visual acuity and the most common way in clinic is with a chart such as the Snellen (pictured left). There are different types of visual acuity tests, and *minimum legible threshold* is the most common. Minimum legible threshold describes the point at which a patient cannot distinguish progressively smaller optotypes (letters or numbers) from one another. Reading a chart like the Snellen is a form of minimum legible threshold. There are other types of visual acuity tests, but these are rarer compared to minimum legible threshold testing.

Minimum separable threshold measures the smallest distinguishable separation between two objects.

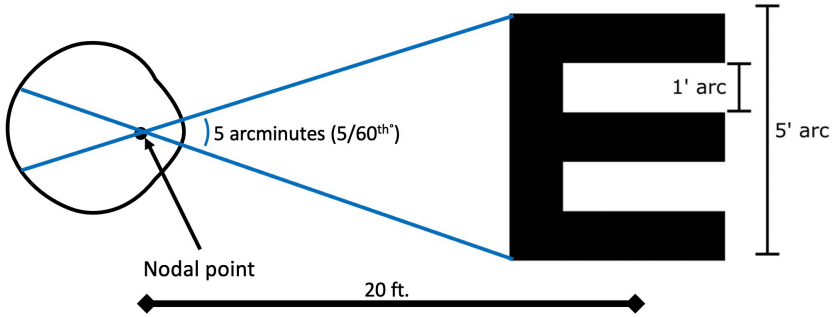
Vernier acuity measures the smallest perceivable break between in a line (the space between line segments).

There are also more modern analogs to the Snellen chart including the Sloan or Tumbling “E” chart. Chart preference varies substantially by clinic.

Minimum legible threshold is reported as the distance a patient could read a line of optotypes divided by the distance a person with normal vision could read the same line. Patients are asked (one eye at a time) to read smaller and smaller lines until they’re unable to read the optotypes.

So, 20/60 means the patient can see at 20 feet what a “normal” eye can see at 60. 20/15 means the patient can see at 20 feet what a normal eye can see at 15. Vision can be more specifically reported as well. For example, if there are 5 optotypes on the 20/20 line and a patient reads 3 of them, it can be reported as 20/20-2. If they read 2, it’s 20/25+2.

The technical definition of 20/20 vision is the ability to resolve a separation of one minute of arc on the retina which is the spatial resolution of the human eye. An arcminute is 1/60th of a degree. The angle refers to the angle formed as it relates to the "nodal point" of the (model) eye.



The optotypes are designed carefully with this in mind. On the 20/20 line, the letter "E" is constructed such that the spaces between the arms are 1' of arc. To distinguish the letter as "E" vs. "P", the eye must be able to resolve to 1' – the spatial resolution of the retina. The entire letter is 5' of arc tall.

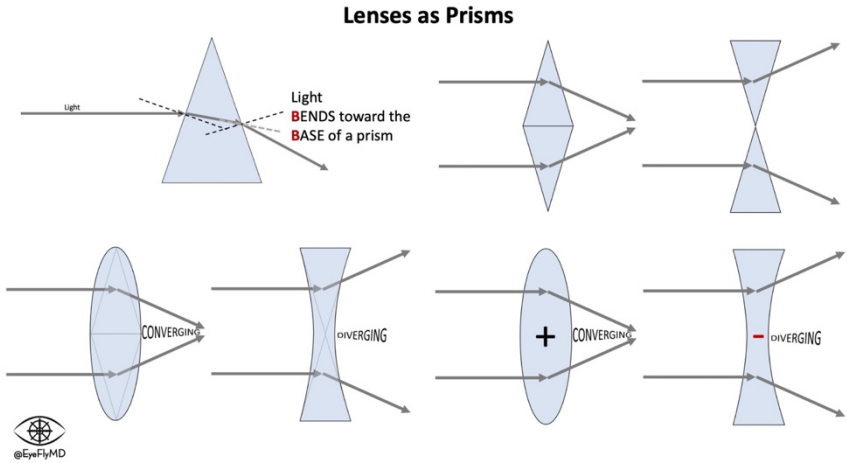
20/20 is "normal" vision, around 20/60 driving becomes questionable, and 20/200 is legal blindness. 20/800 (aka 5/200) is where walking around becomes challenging.

Lenses (As Prisms) and Vergence

If you can remember what a prism does to light, you will never forget **PLUS, CONVEX, CONVERGING** or **MINUS, CONCAVE, DIVERGING** lenses ever again. Remember from the strabismus discussion, due to Snell's law light **b**ends towards the **b**ase of a prism. The dotted lines on the following illustrated prism represent the *normal*, the imaginary line perpendicular to the optical surface. When light enters a substance with a greater refractive index (*n*) 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 *n*s include the cornea (1.376), 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, CONVEX, CONVERGING** lenses (two prisms base to base) are converging lenses and **MINUS, CONCAVE, DIVERGING** lenses (two prisms apex to apex) are diverging lenses.

PLUS, CONVEX, CONVERGING lenses are denoted by **BLACK** numbers. **MINUS, CONCAVE, DIVERGING** lenses are denoted by **NEGATIVE** numbers.



Far Points and Focal Points

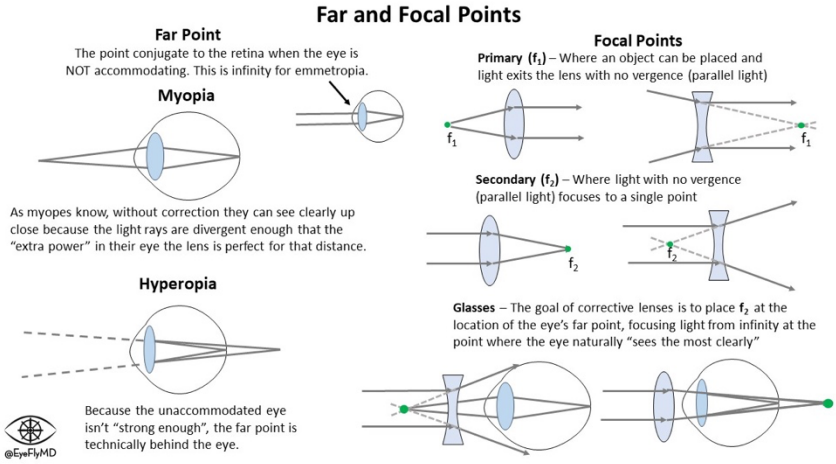
The eye must take light with no vergence (remember, parallel light from infinity) and focus it to a single point on the retina over a distance of ~24 mm (the average length of an eye). This requires quite a bit of refracting power and the average eye is ~ +60 D. The eye must be **PLUS, CONVEX, CONVERGING** naturally because natural light must be focused onto the retina. The cornea (especially the air-tear interface where the biggest refractive index change occurs) contributes ~74% 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 exit the lens with zero vergence. A secondary focal point (f_2) is where light with zero vergence would focus on a single point after passing through the lens. 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 eye 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 describes 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. Unaccommodated is an important modifier because the lens can accommodate and become more **PLUS**.

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. Uncorrected myopes can see at near but not at a distance.

- Correction: **Concave** lenses that diverge the light (denoted by a **-**, **MINUS** prescription)
    - One way to think of it is that a concave lens will "spread" the light rays or "push" the image back, so it comes to a point 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 (close in front of the eye in myopes).

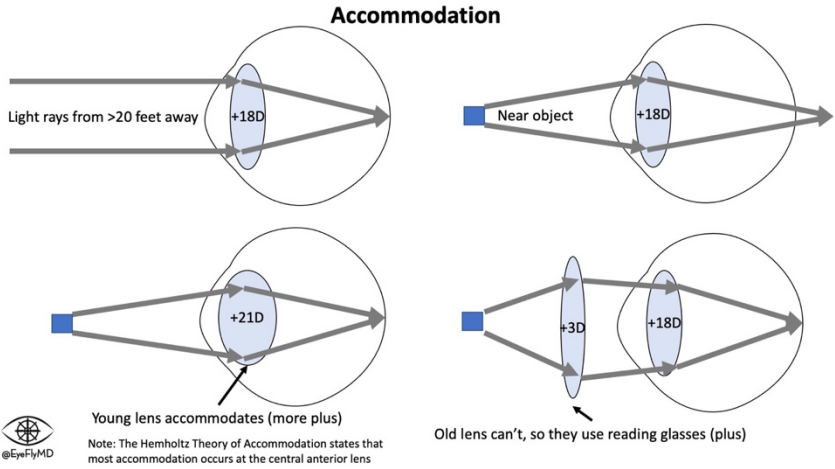
Hyperopia, or "farsightedness", is a refractive error of the eye characterized by light focusing BEHIND the retina after passing through the optical system of the unaccommodated eye. Technically for the *unaccommodated* hyperopic eye no distance (near or far) will result in clear vision but (especially 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 **+**, **PLUS** 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 comes to a point 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).

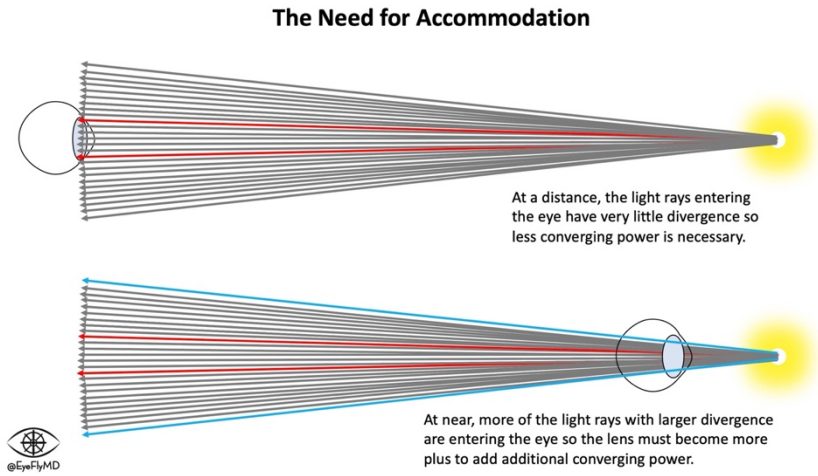
Accommodation

Accommodation is necessary because as objects become nearer to the eye, more divergent rays are allowed to enter the eye. The eye must **accommodate** to allow the light to keep focus on the retina. The 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 (according to the *Hemholtz* theory) is:

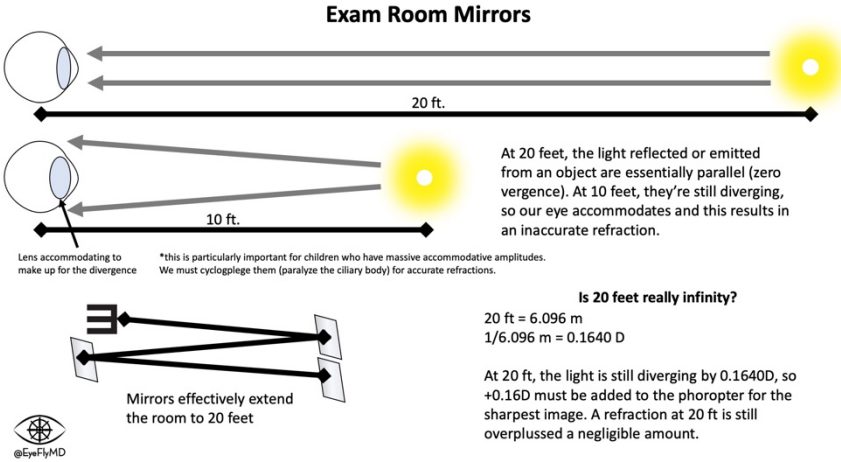
- The ciliary muscles contract
- This loosens the zonular fibers supporting the lens
- The lens bulges becoming more **CONVEX (+)** and increases converging power



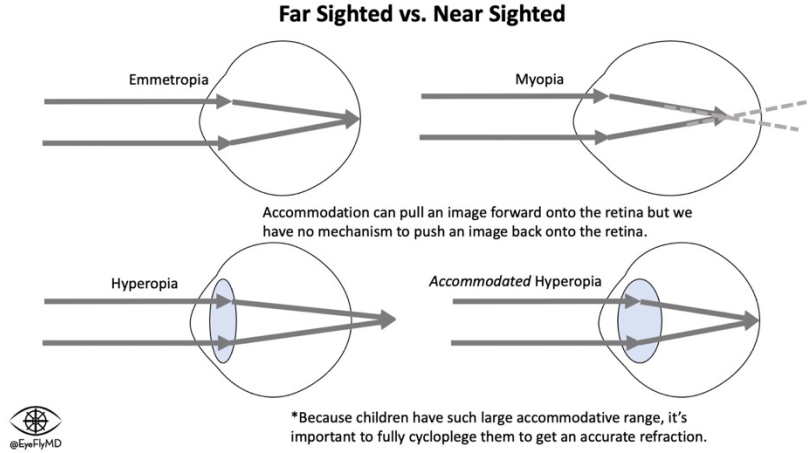
The physical basis for this is illustrated in the following image. Consider that the closer the eye is to a light source the more divergent the rays of light are. If an eye is infinitely (well, about 20 feet) far from a light source the only rays of light making it in the pupil are essentially parallel 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…



20 feet (as in 20/20) is close to "optical infinity" where light rays entering the eye are parallel and the eye doesn't need to accommodate. This is also expressed as 6 meters. The eye in an unaccommodated state allows for an accurate refraction prescription. 20ft rooms are expensive so most eye clinics use mirrors to simulate this distance.



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.

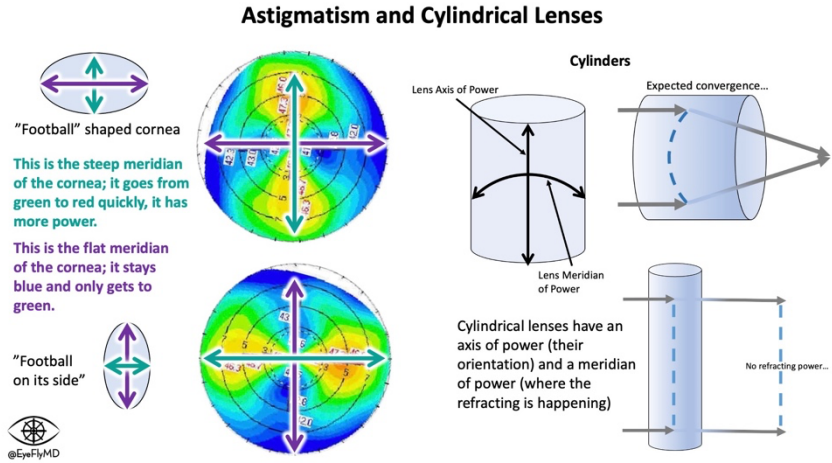


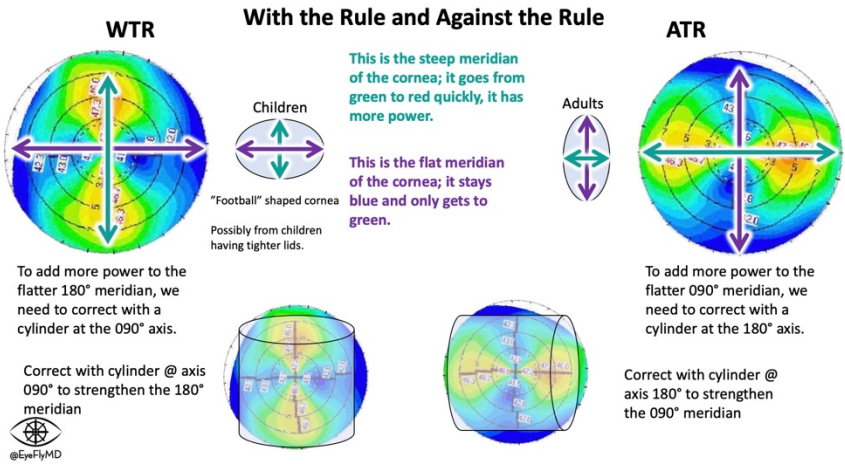
Do you think a patient would prefer if their prescription were too minus or too plus? Think about if their prescription was too plus first. Too much convex, converging power will cause the image to fall closer to the lens, thus in the vitreous cavity and in front of the retina. Does the eye have a mechanism 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 eye does have a way to make itself more powerful (more plus) and that's the entire principle of accommodation. If a prescription is too minus the eye has essentially become hyperopic and 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 allows individual power control of each axis for 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 lens makers) use negative cylinder because this is how glasses are actually constructed. This is also where the whole "with the rule" "against the rule" nomenclature comes from. Ophthalmology and optometry are working in opposite directions so the axes are going to be reversed. Using the simple "rule" language helps simplify things and facilitate communication.

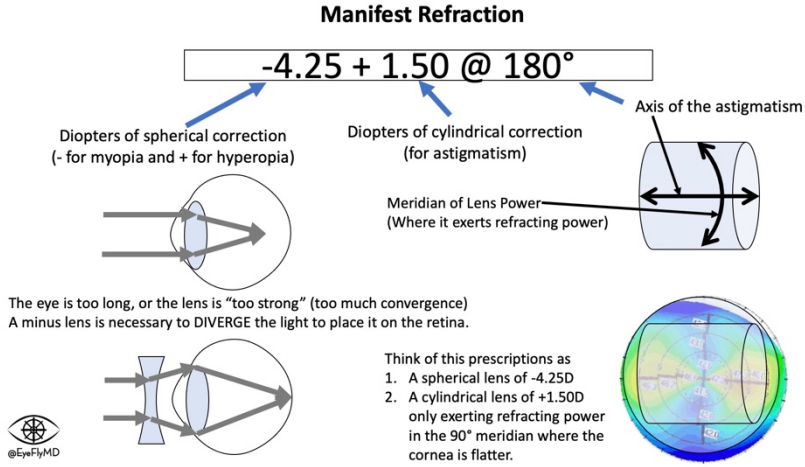
Here is how to convert between plus and minus cylinder:

1. ADD sphere and cylinder
2. REVERSE the sign of the cylinder
3. FLIP the axis by 90°

For example, a prescription of -4.00 + 2.00 x 090° is equivalent to -2.00 - 2.00 x 180°. We only use 0° - 180° for simplicity. 270° is the same as 90°.

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 because this is how ophthalmology imagines cylinder.

Let's 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.



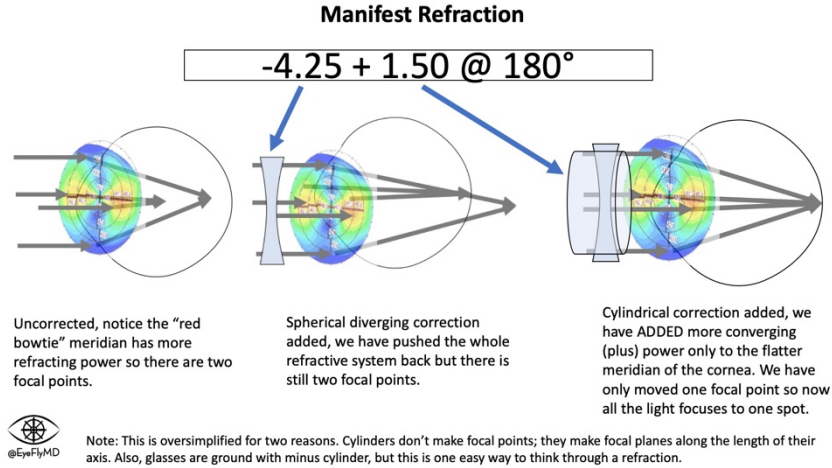


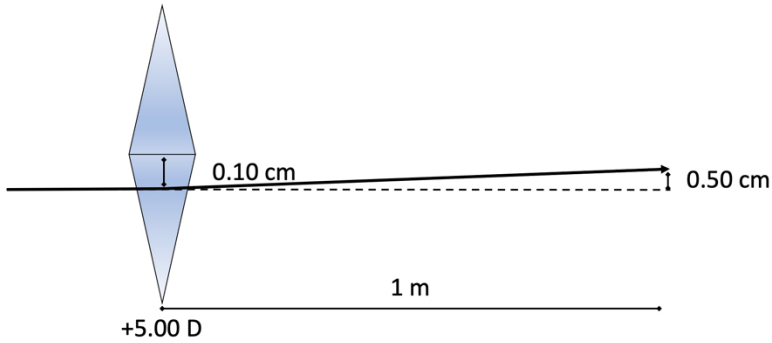
Image Jump vs. Displacement

It's important to consider the effects of adding lenses together as is the case for bifocals. Bifocals typically have a distance section and a reading section (usually around +2.50 D) on the bottom portion of the lens. Now that we understand how lenses can be viewed as prisms, we can explore how bifocals can impact the final image due to induced prismatic effects – The Prentice's Rule.

$$\Delta = h \cdot D$$

Δ is displacement in Prism Diopters (PD, every prism diopter would cause a 1 centimeter image shift at a distance of 1 meter).
h is height in centimeters from the base of the prism.
D is diopter strength of the lens.

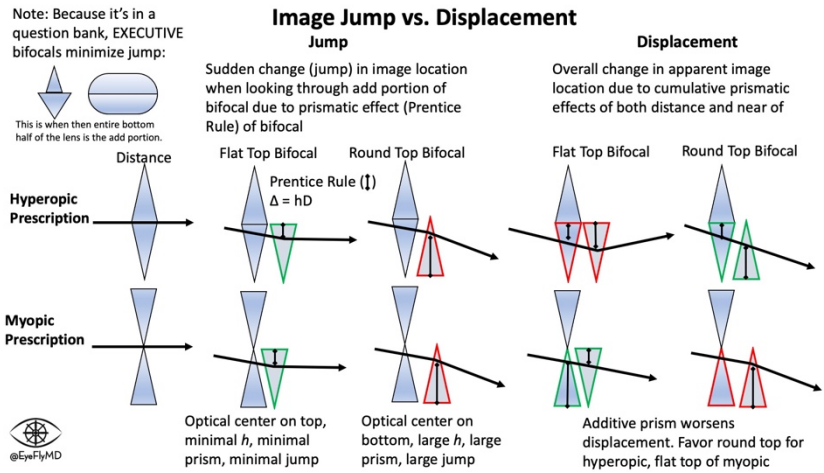
For example, viewing a 5.00 D lens 10 mm (0.1 cm) below the optical center would result in the following induced prism. Δ = 0.1 cm · 5.00 = 0.50 PD. Remember, due to Snell's law light **b**ends towards the **b**ase of a prism



The two critical concepts to understand with regard to bifocals and induced prism are **image jump** and **image displacement**.

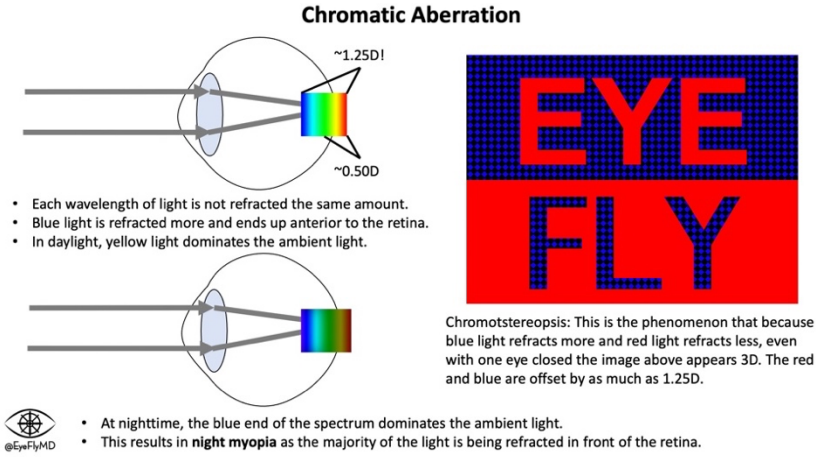
Image jump is the consequence of looking through a new optical center. When the eye looks down to read and looks through the bifocal portion, the further the line of sight from the optical center is, the larger the induced prism will be (Prentice's Rule). Suddenly looking through a new optical element, far from its optical center will cause more induced prism and image will appear to jump. Flat top ADD bifocals have their optical center at the top and close to the center of the rest of the lens and thus have less image jump. Imagine sewing with round top bifocals though. Looking at the sewing machine may require use of the distance portion but threading a needle in your hands would require use of the add portion. Shifting from the machine to the needle in your hands would cause the needle to jump locations. This may be frustrating. Executive type bifocals reduce image jump to the greatest degree.

Image displacement is the consequence of cumulative prismatic effect from both the distance and add portion. Displacement is affected by the type of distance correction. For concave lenses, flat top ADD "cancels" the prismatic effect from the lens while round top add contributes to it. The opposite case is true to for convex lenses. Consider building a model with a hyperopic prescription and a flat top bifocal. In this situation, displacement is maximized and the true location of every image is slightly different from what you're seeing.



Aberrations

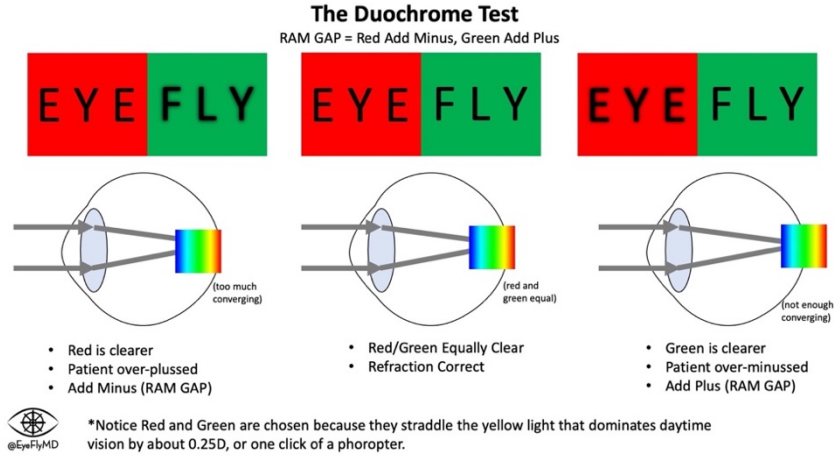
Eye charts often have a Red and Green split background for the acuity charts. What would this be used for? Well, first we must understand the concept of **chromatic aberration**. Not all light experiences refraction the same way. Shorter wavelengths are slowed more by optically dense substances. The 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 so the red looks closer than blue.



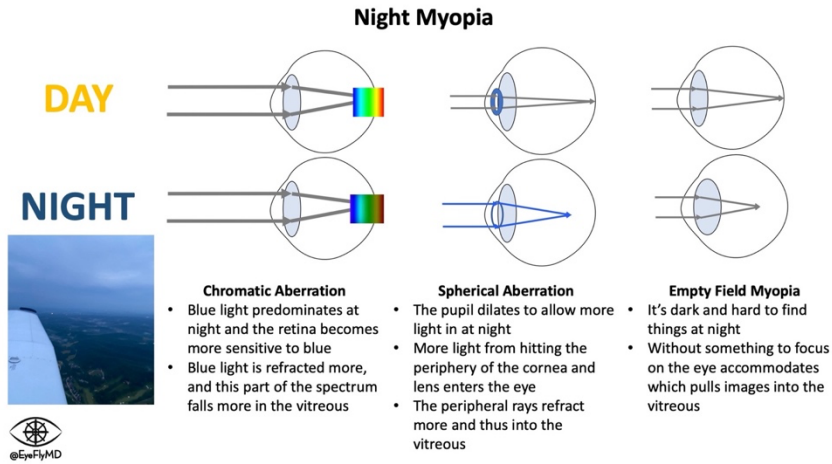
If you had to pick one color to precisely fall on your retina, which would you choose? During the day, yellow light dominates the ambient spectrum, so this is the color for which we try to optimize vision. How positive are we, though, that the yellow spectrum is the one falling on the retina? Remember earlier we said that people will generally prefer 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 minus” we can perform the **Duochrome Test**. This involves overlaying the Snellen (or other) chart on a red and green background. Why red and green though?

Well, we just said yellow light dominates the daytime atmosphere and red and green tend to straddle the yellow spectrum by about 0.25 D on either side.

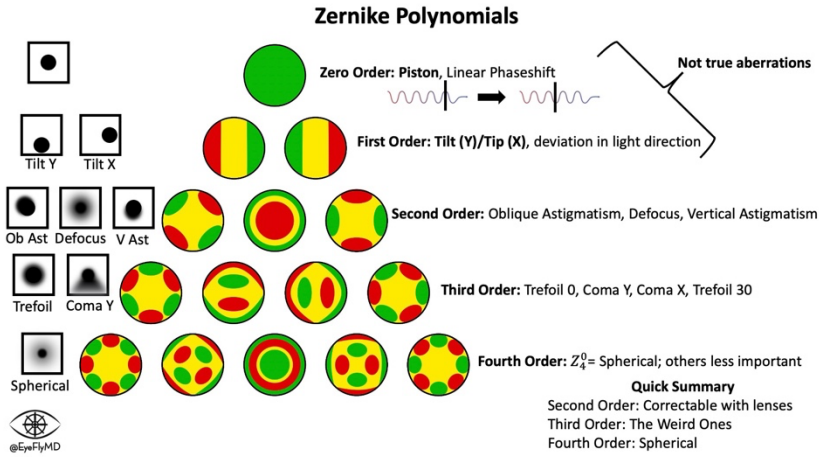
The mnemonic **RAM GAP** can help us remember what to do depending on what the patient reports seeing. If the **red** side is clear but the **green** side is blurry then this means the **red** part of the spectrum is falling on the retina and there is too much converging power (**PLUS**) so we need to "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**. Visual acuity needs to be at least 20/30 in each eye for this test to be effective.



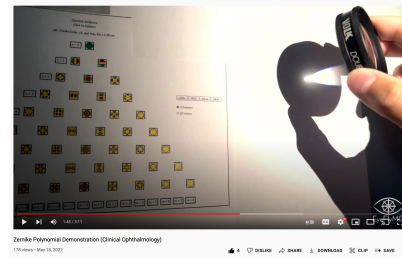
As mentioned before, this chromatic aberration is a cause of night myopia but it's not the only contributing factor. Another cause is spherical aberration referenced before in the discussion of the cornea. Lastly, the eye experiences something called "empty field myopia" where at night when the eye can't 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.



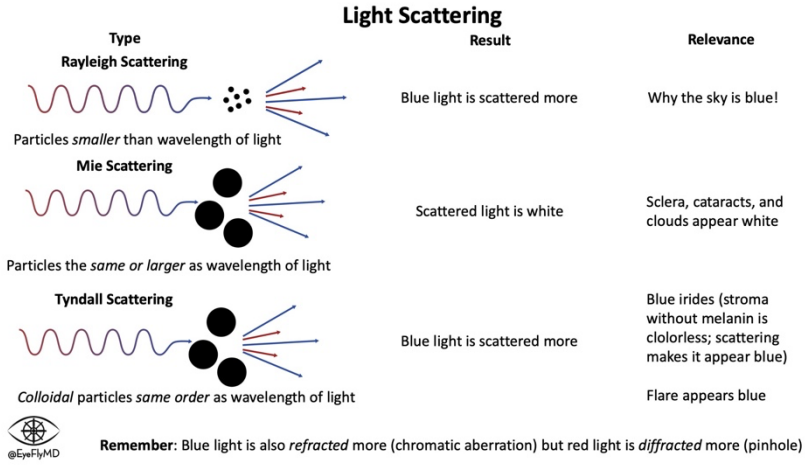
This is a very complex and advanced topic, so it is easier to watch this video where I physically demonstrate the aberrations.



https://youtu.be/ESr3Uiqt4xs

Scattering

A final optics topic that comes up with reasonable frequency on standardized questions and has clinical significance is scattering. Particles (in the air or in a medium) cause light to scatter in different ways. The most clinically significant of these are probably Mie Scattering (why the sclera and cataracts are white) and Tyndall Scattering (why a blue iris appears blue). See the summary below.



Notes:

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This book series is dedicated to my first teachers:
Raymond & Helen Holderle

Thank you for reading! Please submit any errors, feedback, questions, or inquiries to matt@eyeflymd.com.

The Eye Guide: Anatomy and Optics

This short book is a summary of high-yield anatomy and optics relevant to ophthalmology. It is a good introduction for new ophthalmology residents and eager students to the basics of the specialty. The format is a logical and coherent "guided tour" through the educational slides from EyeFlyMD.com. There is a broad selection of basic principles highlighted by significant detail that an ophthalmology resident would need to know. Hopefully this will be a helpful reference guide.

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