DAY 1

Focusing Light Onto The Retina

Basic And Clinical Science Course References
2.2, 2.7, 2.8, 2.9, 2.10, 3.3

Concepts

- [ ] Refractive states of the eye
- [ ] Tear film physiology
- [ ] Corneal anatomy
- [ ] Anterior chamber anatomy
- [ ] Aqueous physiology
- [ ] Lens anatomy
- [ ] Vitreous anatomy

Active Learning Ideas

- [ ] Identify ocular structures responsible for focusing light onto the retina
- [ ] Generate descriptions of symptoms suggestive of poor focus
- [ ] Plan efficient methods of assessing and managing problems related to focus of light

Suggested Self-Assessment Activities

- [ ] Draw out focal points for different refractive states
- [ ] Classify refractive errors
- [ ] Flashcard quiz about ocular media anatomy and physiology
- [ ] Question bank

Details To Know

- What types of ocular media states/conditions alter the refractive state?
- What structures produce tear film elements?
- What structures, components, and physiology affect the clarity of the cornea?
- What are the blood-ocular barriers? How do these barriers affect media clarity?
- What structures, components, and physiology affect lens clarity?
- What affects vitreous clarity? Why don't patients with asteroid hyalosis see them?

Introduction

Diagnosing and managing problems with focusing light onto the retina is a significant part of an ophthalmologist's daily work. Although refractive error is one of the more commonly implicated causes for impaired light focus, it helps to think through the entirety of ocular media.

Organizing by anatomy provides a systematic process for evaluation/examination. Since we're just starting our journey, reviewing the anatomy and the physiology of ocular media is a good place to start. Today's topics will be a broad overview (with a few details sprinkled in). Don't worry if some concepts are too hard to understand or feel too simplistic right now; this is intentional, as all of the topics covered today will show up again in later iterations.

Suggested Reviews

In Clinic

You may find that some of the patients you evaluate today have abnormalities affecting the ocular media. Take notes on how your patients described their symptoms, what the exam findings looked like, and what the final diagnosis was.

By adding this information to your anatomic framework, you're incrementally learning the context for how to evaluate different diseases.

Example

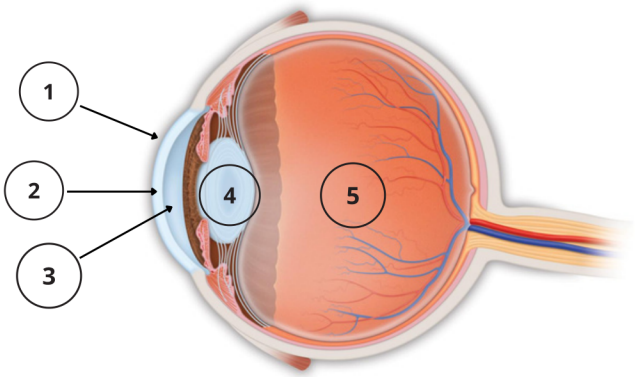
Symptom	Clinical Exam	Diagnosis
Blurred vision	Corneal punctate epithelial defects	Dry eye syndrome
Monocular diplopia	Astigmatism	Astigmatism
Blurred vision	Cataract	Cataract

Ocular Media and Refraction

Ocular Media

Five major structures comprise the ocular media:

1. Tear film
2. Cornea
3. Aqueous humor
4. Lens
5. Vitreous humor



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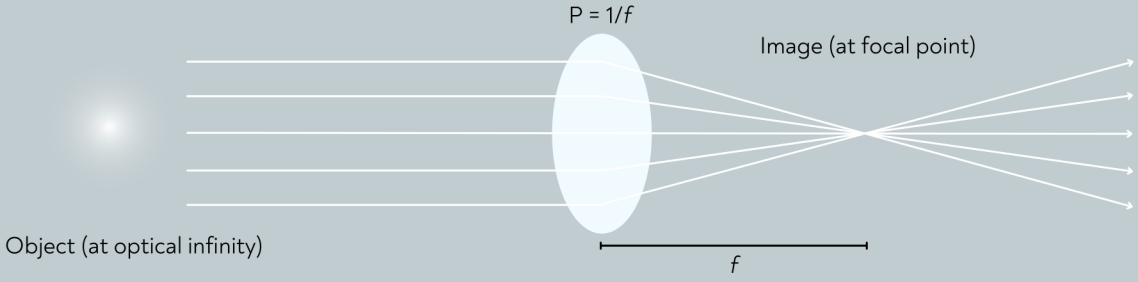
The air-tear film/corneal interface (~40 diopters, D) and lens (~20 D) both contribute refractive power to the eye, for a total refractive power of approximately 60 D.

What is a diopter?

A **diopter** (abbreviated D) is the unit of measurement for a lens's power (P). It represents the reciprocal of the focal length ($1/f$). A lens's **focal length** (measured in meters) is the distance between the lens and the image it produces (at the lens' **focal point**). By convention, *convex* lenses, which *converge* light rays, are represented with *positive* diopter powers (focal points on the opposite side of the lens than objects are positive). *Concave* lenses, which *diverge* light rays, have *negative* power.



Clinical Correlation: Refractive Power Of The Eye

The intraocular lenses used in cataract surgery generally are approximately 20 D. Intraocular lenses for correcting hyperopic eyes tend to require a higher intraocular lens power (more plus). In contrast, myopic eyes require a lower intraocular lens power (less plus).

Corneal topography measurements in average corneas have a power of approximately 40 D. Steeper corneas (such as in keratoconus) have a higher keratometric power (K). In contrast, flatter corneas (such as in sclerocornea or post-keratorefractive myopic surgery) have a lower K.

The aqueous and vitreous humor are predominantly comprised of water and are normally clear, allowing transmission of light through them with minimal refractive or diffusing effects. Conditions that opacify the aqueous (such as anterior uveitis or hyphema) or the vitreous (such as vitreous hemorrhage) can result in blurred or dim vision.

Refraction

Let's review some basic optics principles before we get into the details for the day.

Refractive Index

Light travels fastest in a vacuum. In denser but clear materials, light travels slower. This property allows light rays to bend or change direction as the light rays move from one material to another. This is called refraction.

Every material slows light down. Its composition affects how much slower light travels relative to a vacuum - for example, the speed of light in water is about 75% of the speed in a vacuum. Because each material affects the speed of light differently, expressing this relationship to the speed of light in a vacuum is helpful. A material's refractive index (n) is the ratio of the speed of light in a vacuum to the speed of light in that material.

In ophthalmology, we apply this principle to understand the optics of the eye, in spectacle correction, and in intraocular surgeries (intraocular lens materials, refractive effects of vitreoretinal surgery, etc.).

Memorize This: Refractive Indices

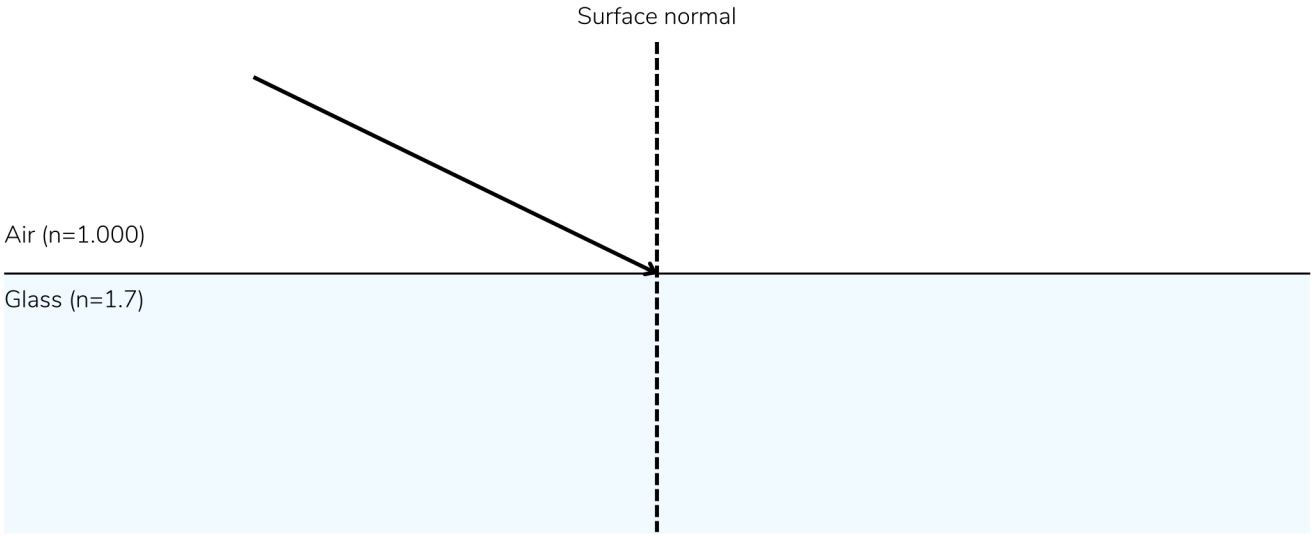
According to the Basic And Clinical Science Course (BCSC), "[t]he refractive index values of air, water, and cornea should be committed to memory."‡

Material	Approximate Refractive Index
Vacuum	1.000
Air at standard temperature and pressure	1.000
Water	1.33 (or 4/3)
Cornea	1.37

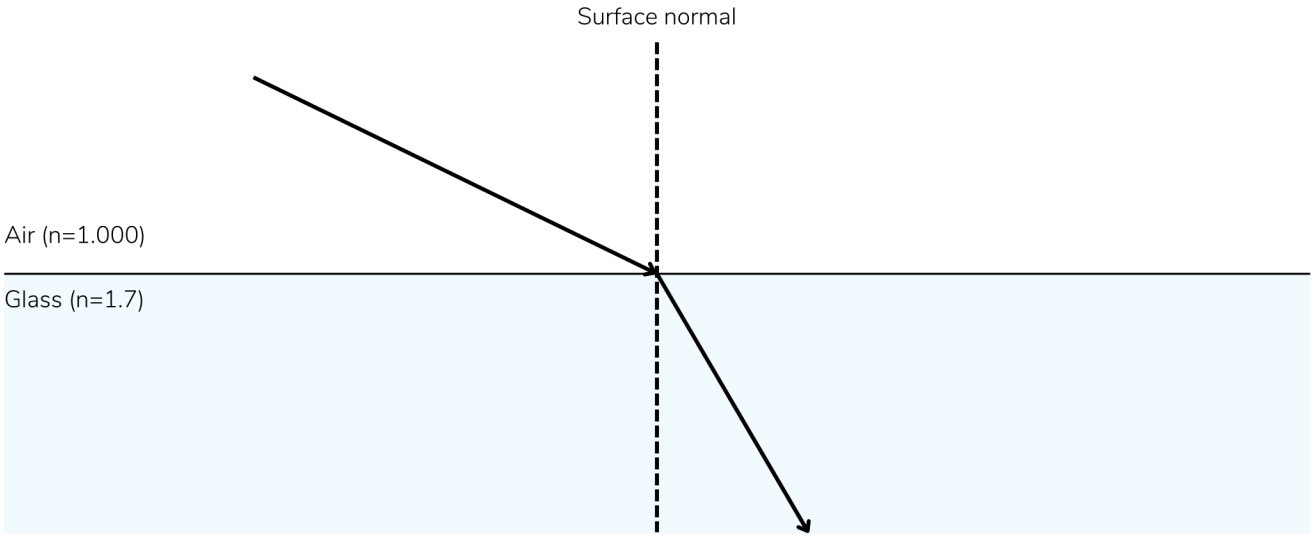
‡Basic and Clinical Science Course, Section 3: Clinical Optics. American Academy of Ophthalmology. San Francisco: 2022-2023, 57.

Optics Review: Snell's Law

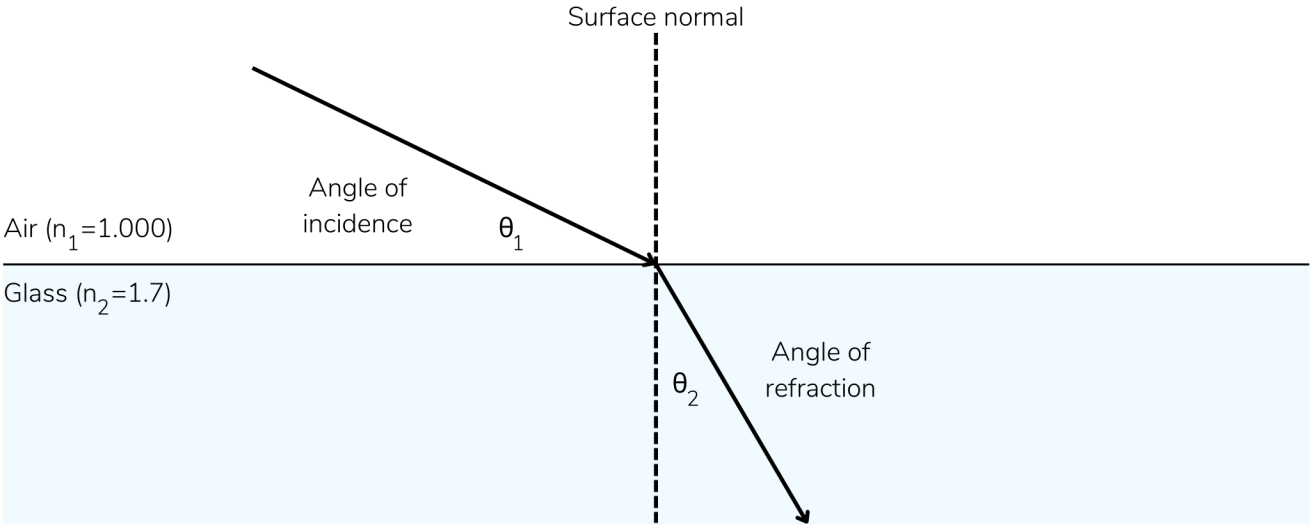
As light travels through different materials (media), the relative difference in speeds of light results in the light rays bending. Let's start by discussing what happens to light as it passes through air then hits a flat refracting surface, like a flat piece of glass.



Because light travels faster through the air than through glass, the light rays will slow down in the glass. This results in the light rays bending toward the surface normal, which is the imaginary line perpendicular to the surface at the point where the light rays hit the material.



Snell's law of refraction states that the amount that the light bends (angle of refraction) is related to the angle the initial light ray hits the surface (angle of incidence) by the proportion of the refractive index for each material.



$$n_1 \sin \theta_1 = n_2 \sin \theta_2$$

Clinical Correlation: Refraction

From a practical perspective, **don't** memorize the equation for Snell's Law! Chances are, you won't be pulling out your calculator to calculate angles and refractive indices in the middle of a busy ophthalmology clinic (hopefully not!).

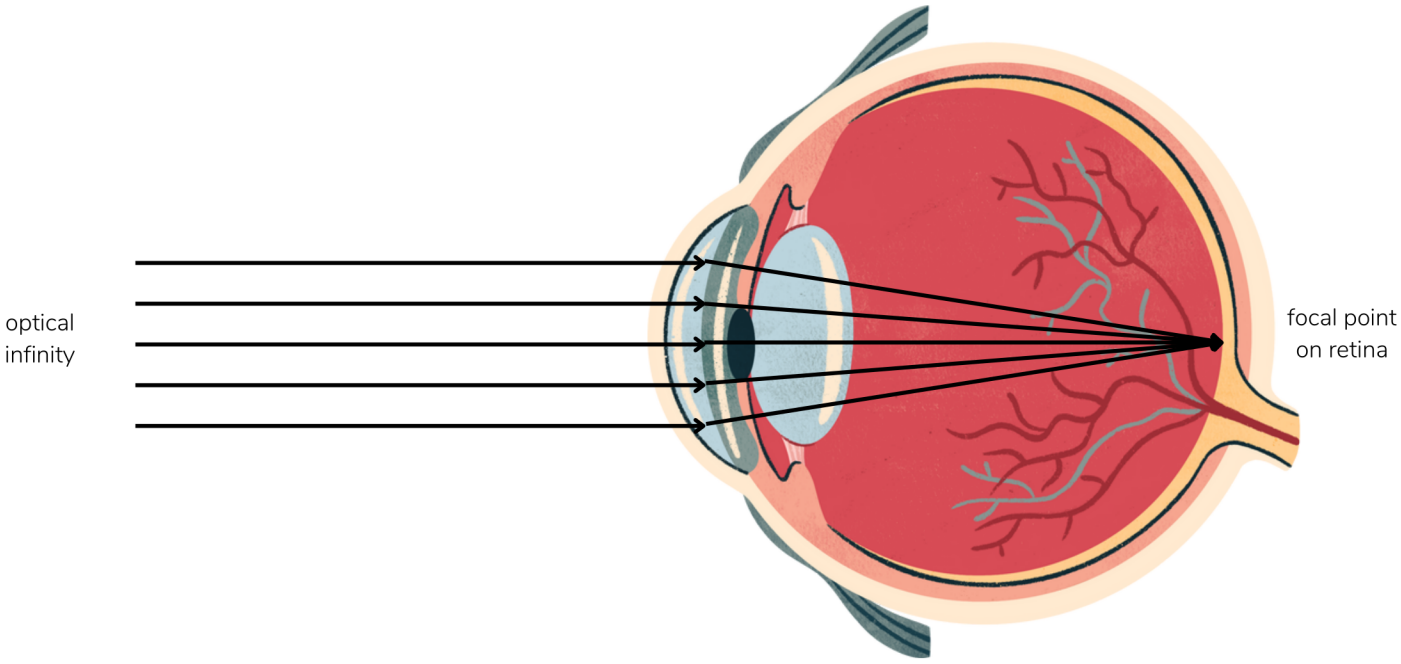
However, we apply the concepts every day. Here are some examples:

- A retina surgeon needs to know the refractive index of an intraocular lens implant to decide whether to use gas or silicone oil for some vitreoretinal surgery.
- A comprehensive ophthalmologist may suggest a high-index material for spectacle correction for high myopia.

Refractive States Of The Eye

Emmetropia vs. Ametropia

As we noted before, the average refractive power of the eye (cornea + lens) is about 60 D. Emmetropia describes the refractive state in which light outside the eye perfectly focuses onto the retina in the nonaccommodating eye.



Ametropia is the category of refractive states in which light outside the eye doesn't focus perfectly on the retina. There are two types of ametropia:

- Hyperopia is the refractive state in which light outside the eye is focused behind the retina.
- Myopia is the refractive state in which light outside the eye is focused in front of the retina.

Ametropia can be axial (caused by the length of the eye) or refractive (caused by the refractive power of the eye).

Astigmatism

Astigmatism describes refractive states where light rays focus on multiple points. Although we often consider astigmatism from the perspective of corneal shape, it is actually a refractive state, which can be affected by changes to the lens (lenticular astigmatism).

Astigmatism is categorized by the position of the focal lines (the main axes to which the light rays focus) and the regularity of astigmatism (if the amount of astigmatism is equal at every point across the pupil plane).

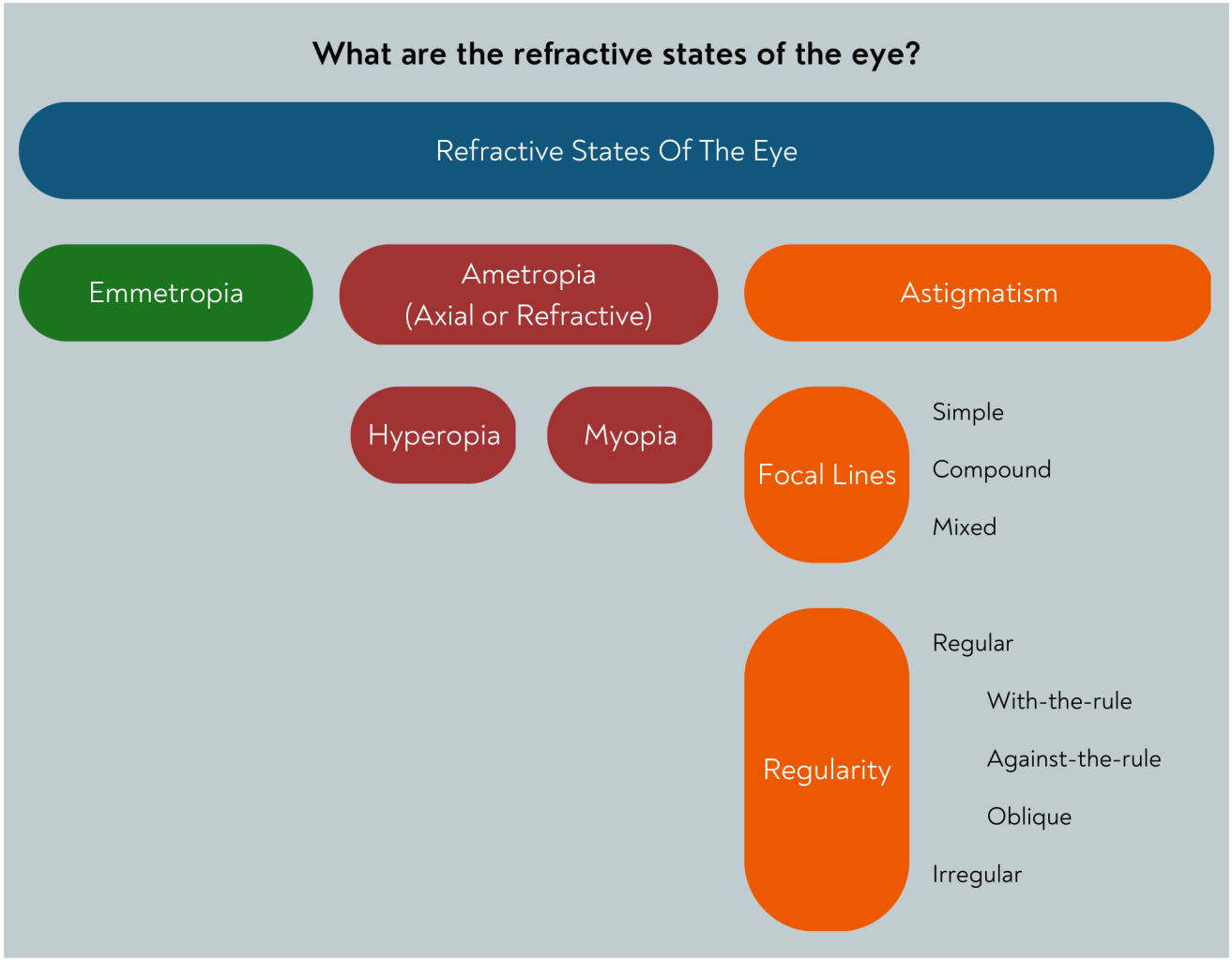
For each astigmatic point, there are two principal focal lines (axes) 90° apart. Simple astigmatism means that one of the axes is focused on the retina (simple myopic or simple hyperopic). If both axes are focused in front of the retina, the astigmatism is compound myopic; similarly, if both axes are focused behind the retina, the astigmatism is compound hyperopic. Mixed astigmatism means that one axis is focused in front of the retina, and the other is focused behind the retina.

Regular astigmatism is categorized as with-the-rule, against-the-rule, and oblique.

- With-the-rule astigmatism means light rays at or near the vertical (~90°) meridian require hyperopic (plus lens) correction. If caused by the cornea, another way this is described is that the vertical meridian is steeper than the horizontal meridian (like an American football or rugby ball lying on its side).
- In against-the-rule astigmatism, light rays at or near the horizontal (0° or 180°) meridian require hyperopic correction.
- In oblique astigmatism, the principal meridians are near the 45° or 135° angles.

Irregular astigmatism describes conditions for which the orientation of the principal axes varies from point to point across the pupil plane. Although this often refers to corneal irregularities, tear film or lens abnormalities can cause irregular astigmatism.

Corneal irregular astigmatism is measurable with corneal topography and wavefront aberrometry. Zernike polynomials are mathematical shapes (e.g., spherical aberration, coma, trefoil) that can approximate some forms of irregular astigmatism and are important for refractive surgery.



Visual Acuity

Although we often think of visual acuity as the measure of how well we see, we typically mean that we are testing the minimum legible threshold when we test for visual acuity. The BCSC defines minimum legible threshold as:

> *The point at which a patient's visual ability cannot further distinguish progressively smaller letters or forms from one another.*

Basic and Clinical Science Course, Section 3: Clinical Optics. American Academy of Ophthalmology. San Francisco: 2022-2023, 151.

What this means is that there are many different ways to describe how well someone can see, but we typically use letters, symbols, numbers, or other standardized forms to help us determine just how finely we can see.

You'll learn the details of Snellen visual acuity and how to measure visual acuity predominantly through clinical experience. However, in a section about focusing light onto the retina, we should review a few details and general concepts.

Memorize This: The Meaning Of The Snellen Fraction

The Snellen eye chart is comprised of standardized forms, called **optotypes**, that are designed so that the entire optotype is 5x the size of the individual strokes that make up the shape. For example, each horizontal line of the E is a fifth of the entire letter E.

When we use a Snellen visual acuity chart for testing, we express the visual acuity in a fraction (e.g., 20/20 in feet or 6/6 in meters). So how do we determine that fraction?

The numerator specifies the testing distance. The patient should be tested 20 feet (or 6 meters) from the test chart. In shorter rooms, mirrors and projectors can effectively create the 20 feet of testing distance. Sometimes shorter testing distances may be used (10 feet, 5 feet, 2 feet, 1 foot, etc.).

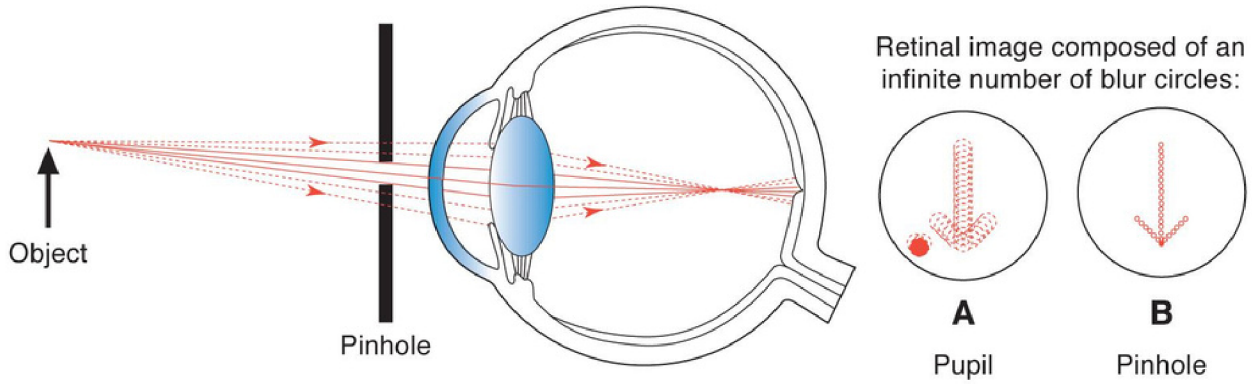
The denominator represents the distance at which the letter subtends an angle of 5 arcmin. So a larger letter will subtend 5 arcmin further away than a smaller letter.

So when we say that a patient is reading on the 20/30 line, we really mean that when the patient is 20 feet from the eye chart, they can read the 30 foot optotype.

Pupils And Pinholes

The amount of light our eyes receive is limited by the size of the pupil in each eye. Because the cornea's shape is not perfectly globular (which we'll discuss later in this section), larger pupil sizes can result in blurred images.

A pinhole can help reduce these effects of larger pupils or irregularities in the cornea or lens. By reducing the size of the aperture, the depth of focus is improved and the "blur circle" on the retina is much smaller.



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When light is imperfectly refracted through the cornea and lens, the light rays create an infinite number of **blur circles** on the retina (A). A pinhole reduces the blur circles by only allowing light to pass through the center of the optical system. This shrinks the blur circles to improve the image resolution.

Pinholes also improve the **depth of focus**, that is, the level of focus for objects at different distances from the observer. The smaller the pinhole (aperture), the better the depth of focus (objects at any distance will be in focus).

Squinting creates a pinhole effect to improve vision.

Pinholes are used to "eliminate" refractive errors to approximate best-corrected visual acuity. There are some limitations to pinhole - the smallest pinhole size is 1.2 mm. The refractive error correctable by pinhole is between -5.00 D and +5.00 D; larger refractive errors require corrective lenses in addition to the pinhole. Monocular diplopia can also be eliminated with a pinhole.

There are some downsides to pinholes. Diffraction is the property of light that describes how light behaves when traveling through a small opening (like a pupil). Instead of traveling straight through the hole, light tends to spread out. This can result in blurred or poor vision when the pinhole is too small. Contrast is reduced as the pinhole size is reduced. Patients with macular disease often have poor contrast sensitivity and will have worsened vision with pinhole.

Tear Film

Tear Film Composition

The tear film maintains a clear and smooth ocular surface (essential for focusing light onto the retina), provides nutrition to the cornea, and contributes to the ocular surface's immune response and antimicrobial actions.

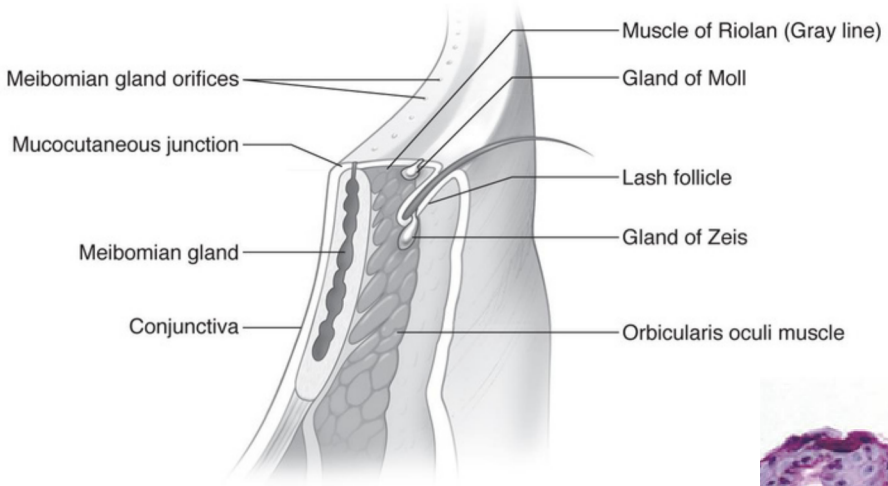
It is predominantly comprised of three elements arranged in a bilayer:

- The lipid layer slows tear evaporation. Disruption to the lipid layer may result in evaporative dry eye syndrome.
- The mucoaqueous layer contains electrolytes, proteins, immunoglobulins, enzymes, and other solutes. Abnormalities of the mucoaqueous layer may result in aqueous deficiency dry eye syndrome or filamentous keratopathy.

Memorize This: Tear Film Production

The histological and anatomical origins of the tear film elements are clinically relevant.

Layer	Gland(s)	Anatomical Location(s)
Lipid	meibomian sebaceous glands (of Zeis)	tarsus (upper > lower lid) eyelash roots
Aqueous	main lacrimal gland Krause Wolfring	superotemporal orbit fornices proximal margin of tarsus
Mucin	goblet cells	conjunctiva



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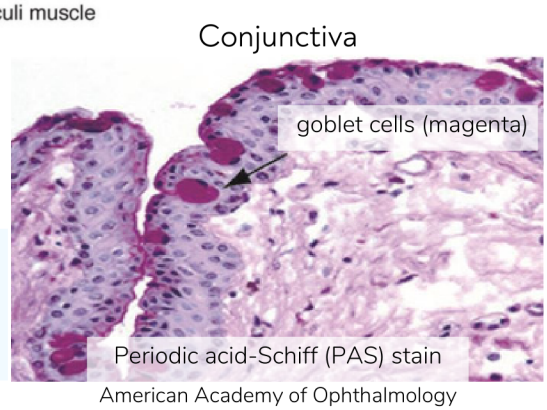
The structures responsible for tear film production are located in the eyelid and orbit.



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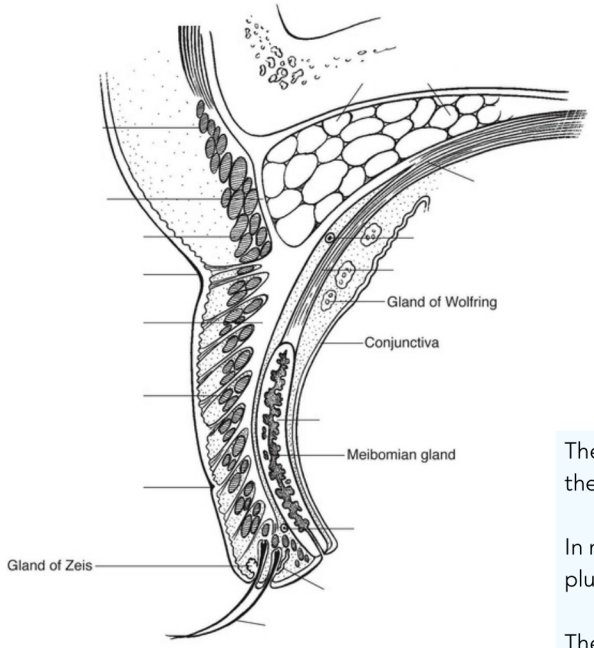
Histology of the conjunctival epithelium (nonkeratinized stratified squamous epithelium with goblet cells) and stroma. Goblet cells stain positive with PAS.



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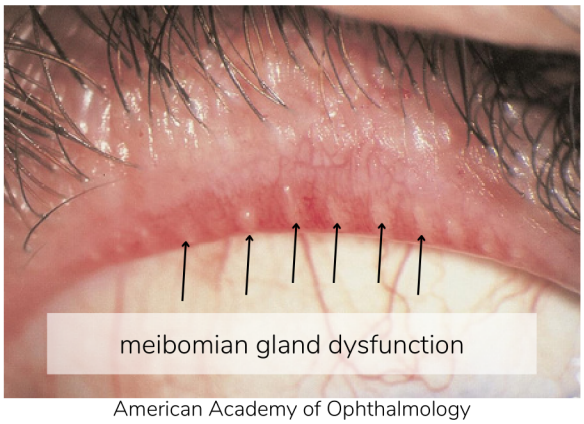

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The meibomian glands lie within the tarsus and have their openings in the eyelid margin.

In meibomian gland dysfunction, the meibomian gland orifices may be plugged or inflamed.

There are more meibomian glands in the upper lid than the lower lid; this is the rationale for the increased frequency of sebaceous cell carcinoma of the eyelid in upper eyelids.

Innervation

The parasympathetic nervous system innervates the meibomian glands and the lacrimal glands. This pathway differs from the parasympathetic pathway which mediates pupil constriction and accommodation.

Segment	Cell Bodies	Notable Structures	Synapse
First-order (preganglionic)	• Superior salivatory (salivary) nucleus • Caudal pons near motor nucleus of CN VII	• CN VII (facial nerve) • Geniculate nucleus • Greater superficial petrosal nerve • Petrous bone • Nerve of the petrosal canal (vidian nerve) • Sphenoid bone • Foramen lacerum	Sphenopalatine ganglion
Second-order (postganglionic)	Sphenopalatine ganglion	• Zygomatic nerve (division of maxillary branch, CN V2) • Superior orbital fissure • Lacrimal nerve (division of ophthalmic branch, CN V1)	Lacrimal gland

Clinical Correlation: Parasympathetic Innervation And Lacrimation

Patients with CN VII (facial nerve) palsy due to lesions between the pons and the geniculate nucleus may have poor aqueous tear production and lid closure.

Conversely, parasympathomimetics such as pilocarpine, echothiophate, neostigmine, physostigmine, pyridostigmine, or edrophonium may increase aqueous tear secretion.

Clinical Correlation: Notable Tear Film Solutes

Many substances are dissolved in the tear film, some of which we'll learn about in future sessions. Here are a few notable substances that have clinical significance:

Immunoglobulin E (IgE)

- IgE levels in the tear film affect allergic states, such as allergic conjunctivitis and vernal keratoconjunctivitis.

Matrix metalloproteinase 9 (MMP-9)

- MMP-9 damages the corneal epithelium by cleaving the epithelial basement membrane and tight junctions. Higher levels of MMP-9 may be present in many ocular surface diseases (e.g., Sjögren, graft-vs-host, post-LASIK, late dry eye syndrome).
- Tetracyclines such as doxycycline may be used in treating dry eyes and rosacea because they inhibit metalloproteinases and phospholipase A2, reducing damage to the ocular surface and increasing the lipid layer.

Intercellular adhesion molecule 1 (ICAM-1)

- ICAM-1 is upregulated on lymphocytes to increase lymphocytic migration to the lacrimal gland and conjunctiva in dry eye syndrome, contributing to the inflammatory component of dry eye syndrome.
- Lifitegrast blocks the adhesion of lymphocytes (via the LFA-1 receptor) to ICAM-1.

More On Mucin

Mucin coats the corneal and conjunctival epithelial cells as a glycocalyx, a filamentous material that stabilizes the tear film and improves the wettability of the corneal surface.

Conditions that cause mucin deficiency (or reduce goblet cell density) include:

- Vitamin A deficiency
- Inflammatory conjunctival disease (such as mucous membrane pemphigoid, drug-associated pemphigoid, superior limbic keratoconjunctivitis, trachoma)
- Chemical conjunctival injury

Conditions that stimulate mucin production include:

- Foreign bodies
- Hyperthyroidism
- Allergic/vernal/giant papillary conjunctivitis

Blinking

The tear film circulates during eyelid blinking. The preseptal orbicularis oculi muscles are critical to tear film turnover.

Clinical Correlation: Cyclosporine A

Cyclosporine A is a commonly used medication for the treatment of dry eyes. It has many functions that improve dry eyes, including:

- Increasing aqueous tear secretion
- Improving goblet cell density
- Decreasing conjunctival epithelial cell turnover
- Reducing lymphocytes in the conjunctiva

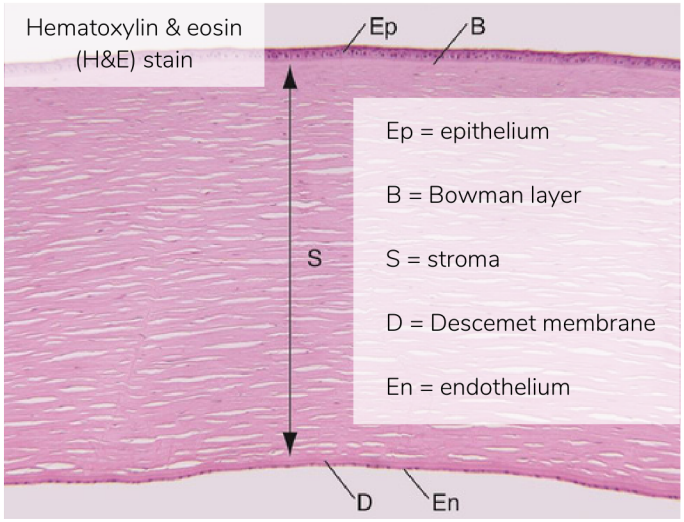
Cornea

Anatomy And Histology

The cornea has 5 layers:

- Epithelium
- Bowman layer
- Stroma
- Descemet membrane
- Endothelium

As ophthalmologists, we will become experts in all of these layers. For today, we will review how the cornea facilitates light focus.



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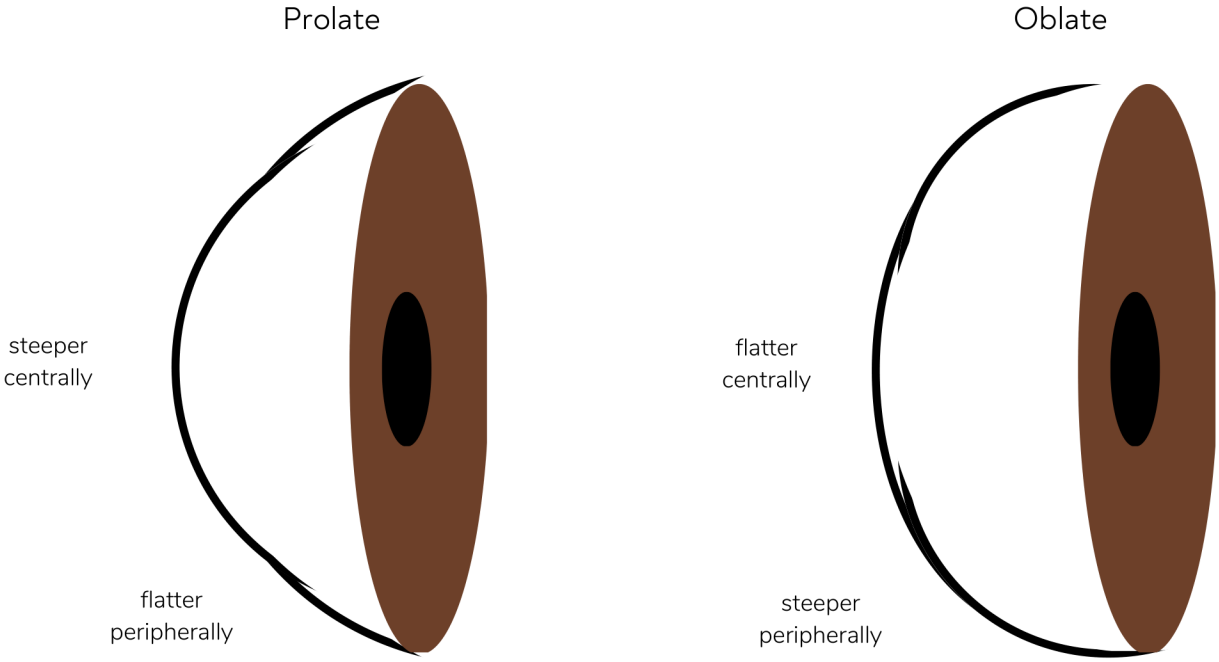
Corneal Shape

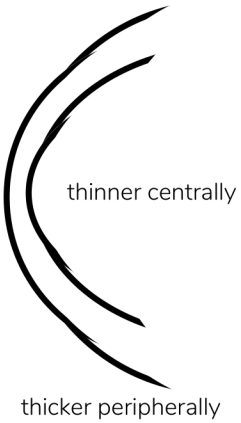
The corneal shape contributes to how light is refracted in the eye. Normally the cornea is curved like a globe centrally, flattening peripherally. This orientation is called prolate. The orientation is called oblate if the cornea is flat centrally and steeper peripherally.

These concepts help us understand corneal refractive power, refractive surgery principles, and contact lens fitting.



The refractive power of the cornea is also affected by the difference in curvature between the anterior and posterior surfaces of the cornea.

The anterior surface of the cornea is slightly flatter than posterior surface. This results in the central cornea being thinner than the peripheral cornea, and the total corneal power being slightly less than the anterior corneal surface would otherwise indicate.



Corneal Epithelium

The corneal epithelium and its basal lamina protect the eye from many problems, such as infections and desiccation. Damage to the epithelium can increase the risk of eye infections and affect the cornea's clarity.

The corneal epithelium is continually turned over, with the epithelial stem cells originating at the limbus and migrating centrally. Damage to the limbal stem cells results in poor epithelial health and visual clarity. We'll go into more detail later.

Memorize This: Basement Membrane Histology

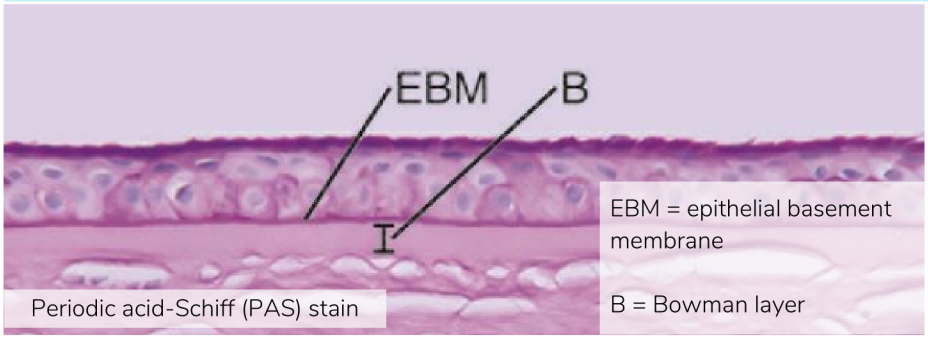
There are many basement membranes that we encounter in the eye. They may be created by epithelium or endothelium, and share some common characteristics. Let's review some of these features, which we will encounter frequently.

Type IV Collagen

- Basement membranes are composed of type IV collagen.
- Alport syndrome, a type IV collagen disease, affects basement membranes throughout the body.

Periodic acid-Schiff (PAS) Staining

- Basement membranes stain positive (magenta) with PAS.



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The Bowman layer is the dense condensation of the anterior stroma.

The corneal epithelium is described histologically as nonkeratinized stratified squamous epithelium.

All basement membranes stain positive (magenta) with PAS and are comprised of type IV collagen.

Bowman Layer

The Bowman layer represents the anterior-most part of the corneal stroma and is not a true basement membrane.

The Bowman layer does not regenerate if damaged and is instead replaced by scar tissue. Corneal scars cause irregular astigmatism, and may result in degradation of the focus of light onto the retina.

Corneal Stroma

The corneal stroma maintains clarity due to the regular spacing of the collagen fibrils (predominantly type I collagen). Disruption of the spacing (such as corneal edema) results in opacification.

Diseases that affect collagen, such as osteogenesis imperfecta type I (a type I collagen disease), can affect the corneal stroma, such as predisposing the eye to ectasia (keratoconus).

Type III collagen is associated with corneal stromal wound healing.

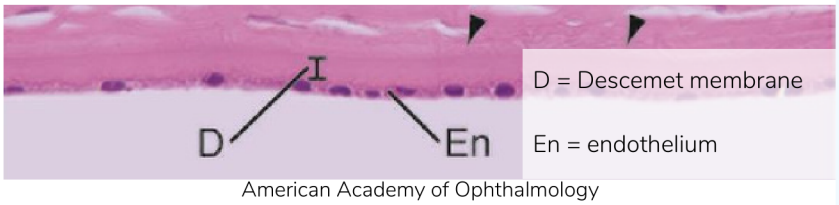
Descemet Membrane And Corneal Endothelium

The Descemet membrane is the basement membrane of the endothelium.

The corneal endothelium regulates the water content of the cornea. Endothelial cell density decreases with age and does not regenerate, even with injury.

Diseases that affect the endothelium, such as Fuchs endothelial corneal dystrophy, will affect water regulation in the cornea, which can cause increased corneal thickness and corneal edema.

Hematoxylin & eosin (H&E) stain



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The endothelial cells are regularly spaced and spread out if damaged.

Pigment and inflammatory cells can build up along the endothelium. Some endothelial diseases (such as Fuchs endothelial corneal dystrophy) can form excrescences of the Descemet membrane, seen as corneal guttae (singular guttata).

Corneal Physiology

The cornea maintains its clarity by remaining avascular. This is mediated by soluble vascular endothelial growth factor receptor 1 (sflt-1), which binds to vascular endothelial growth factor A (VEGF-A) and blocks its effects.

Because there is normally no direct vascular supply to the cornea, stromal glucose metabolism is mediated by glycolysis and the tricarboxylic acid (TCA) cycle when oxygen is present.

If the cornea is hypoxic (such as with tight-fitting contact lenses), the pyruvic acid products from glycolysis are metabolized to lactic acid instead of carbon dioxide and water. The accumulation of lactic acid can then lead to corneal edema and endothelial damage.

Anterior Chamber And Aqueous

Aqueous Secretion And Composition

The aqueous humor is secreted by the nonpigmented epithelium of the ciliary body. Aqueous is relatively protein-free; this keeps the aqueous relatively transparent.

Memorize This: Blood-Aqueous Barriers

There are three blood-aqueous barriers, which are formed by tight junctions:

- Nonpigmented ciliary epithelium
- Iris vasculature
- Inner wall endothelium of the Schlemm canal

Disruptions to the blood-aqueous barrier (such as trauma, chemical injury, inflammation, ischemia, etc.) results in an increase in aqueous proteins, which lead to fibrinous exudates, synechiae formation, and damage to surrounding intraocular structures.

Increases in VEGF-A levels (such as in ocular or retinal ischemia) may disrupt the blood-aqueous barrier.

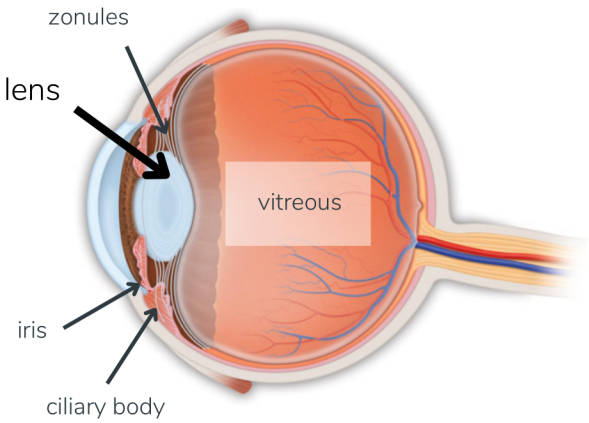
Aqueous Outflow

Aqueous outflow is mediated through the trabecular meshwork and Schlemm canal, as well as the uveoscleral pathway through the iris root.

Lens

Anatomy And Histology

The crystalline lens is located posterior to the iris and pupil but anterior to the vitreous cavity in the posterior chamber.



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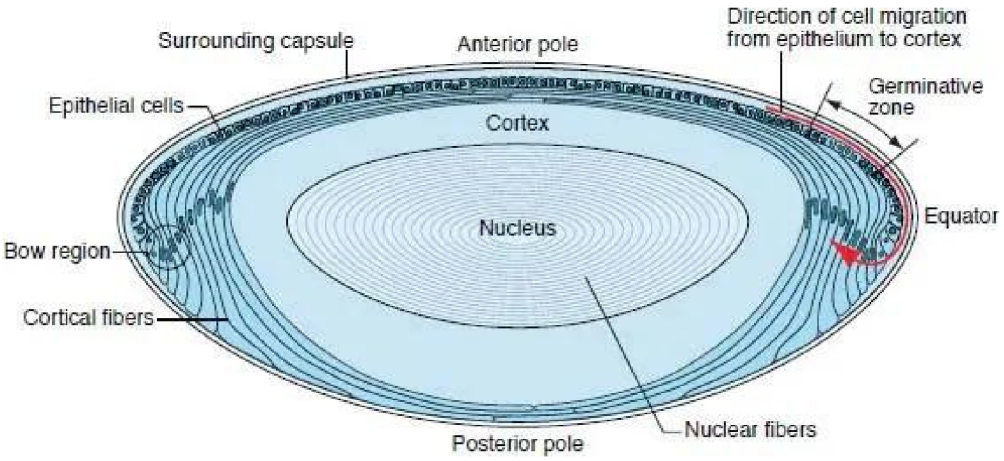
It is avascular and is encapsulated by a basement membrane (capsule). As with all basement membranes, the capsule is composed of type IV collagen and is PAS-positive.

Lens Epithelium

The lens epithelium proliferates throughout life and divides just anterior to the equator in a ring called the germinative zone. After lens extraction, residual epithelial cells can continue dividing and proliferating, resulting in lens capsular opacification and fibrosis.



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The lens is layered like an onion.

After cataract surgery, epithelial cells from the germinative zone can continue to proliferate, leading to anterior or posterior capsular fibrosis or opacification. Elschnig pearls or a Soemmering ring are other findings associated with residual lens epithelial cells.

Crystallins

Crystallins are proteins specific to the lens that stabilize the regular orientation of the lens proteins (minimizing aggregation which can cause opacification) and reduce oxidative damage from ultraviolet light.

Lens Physiology

The lens typically relies on glycolysis for energy.

In hyperglycemic states such as diabetes mellitus, it is theorized that the excess glucose shifts metabolism to the sorbitol pathway, resulting in an accumulation of sorbitol and an influx of water into the lens secondary to osmotic pressure. This increased water in the lens leads to cataract formation.

Galactosemia and galactokinase deficiency may result in cataract formation due to abnormalities in galactose metabolism.

Vitreous

Anatomy And Composition

The vitreous comprises 80% of the globe's volume, and, while predominantly water, it is highly viscous primarily due to the presence of hyaluronic acid, a mucopolysaccharide.

Collagen fibrils confer gel-like properties to the vitreous; decreased amounts of collagen result in the vitreous behaving more like a liquid. The predominant collagen type is type II (also present in ligaments).

The predominant cell in the vitreous is called a hyalocyte. They have some immune functions (phagocytosis) and some fibrotic functions, contributing to epiretinal membrane formation.

Chondroitin sulfate is a sulfated glycosaminoglycan that interacts with hyaluronic acid in the vitreous to maintain its gel structure. Versican is the predominant form of chondroitin sulfate in the vitreous.

Clinical Correlation: Stickler And Wagner Syndromes

Stickler syndrome is a genetic disease (COL2A1 gene) affecting type II procollagen. It has vitreous structural abnormalities which manifest as a clear liquid vitreous cavity (optically empty vitreous) and vitreoretinal interface problems (increased risk for retinal detachment, lattice degeneration).

Stickler syndrome patients also have ligamentous abnormalities (type II collagen is also present in ligaments), manifesting as joint problems (hyperextensibility, joint enlargement, arthritis, spondyloepiphyseal dysplasia), and the Pierre Robin sequence (cleft palate, micrognathia, glossoptosis).

Wagner syndrome is a genetic disease (VCAN gene) affecting versican, the predominant form of chondroitin sulfate in the vitreous. Patients with Wagner syndrome present with an optically empty vitreous, peripheral condensation, and retinal degeneration.

Memorize This: Vitreous Attachments

The vitreous is strongly adherent to the eye in five locations:

- Vitreous base (strongest attachment)
- Optic nerve head margin (vitreous separation here often results in a complete or partial ring [Weiss] which can manifest as a large "floater")
- Perimacular region surrounding the fovea
- Along retinal vessels (vitreous separation here can result in vitreous hemorrhage ("hemorrhagic PVD")
- Peripheral posterior lens capsule

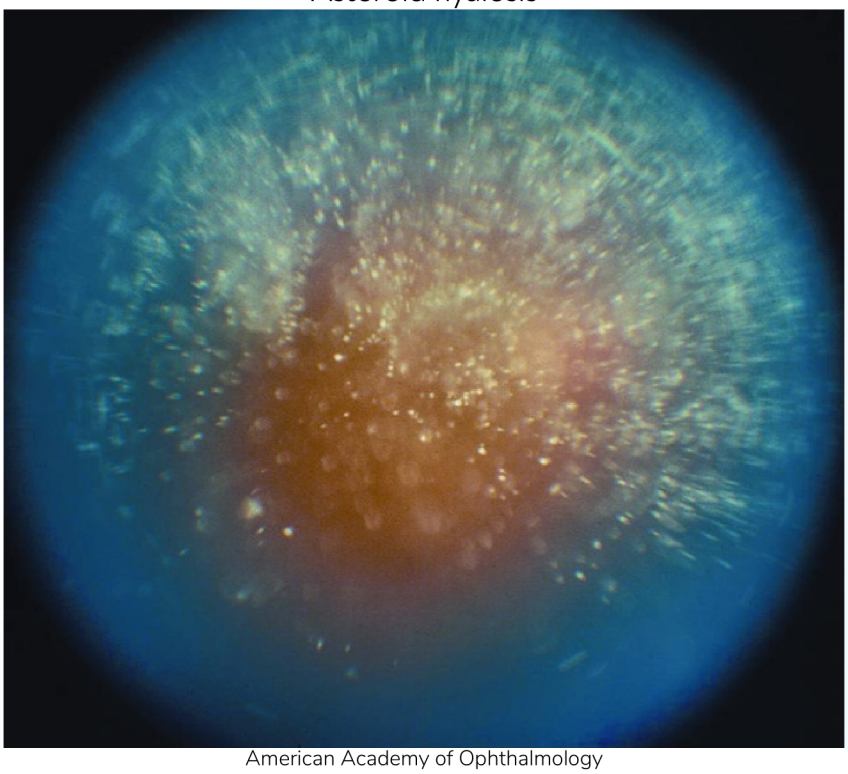
Vitreous Opacities

Some vitreous opacities, such as asteroid hyalosis, may be numerous but often do not cause visual symptoms when they remain small and discrete. This is because the opacities are small and smooth enough that minimal light scattering exists. However, because the opacities are refractile, the light reflections back to the observer can interfere with visualization of the fundus.

Direct and indirect ophthalmoscopic techniques have fewer reflections and result in better visualization of the fundus than slit-lamp biomicroscopy.

Larger and more posterior vitreous opacities, such as posterior vitreous detachment, may be more visible to patients.

Asteroid hyalosis



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Asteroid hyalosis is a degeneration consisting of countless very small (10-100 nm) lipid and calcium-containing particles (asteroid bodies).

Though they have a very striking clinical appearance, patients are rarely symptomatic. This is thought to be due to the minimal deflection of light being focused into the eye by these particles.

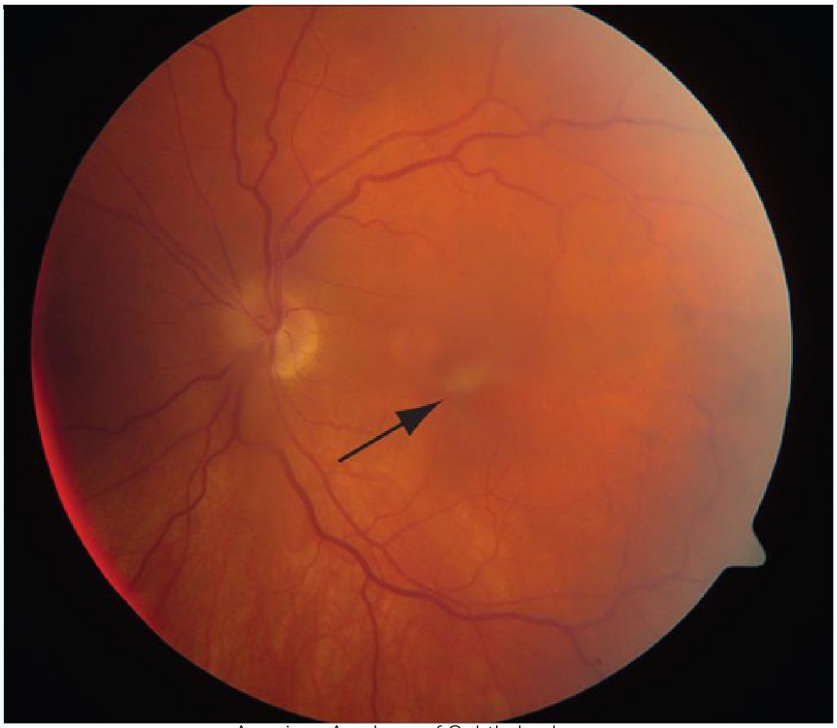
Because the asteroid bodies are often highly reflective (refractile), observers attempting to view the fundus (or performing fundus imaging) often have limited views.

Fluorescein angiography can help visualize retinal lesions in patients with asteroid hyalosis.

Posterior vitreous detachment

Posterior vitreous detachment (PVD) is a common degeneration in which the vitreous loses some of its viscosity, becoming more watery (**synchysis**). This leads to the collapse of the vitreous sac (**syneresis**).

As the vitreous gel contracts, it separates from the posterior retinal surface, including the strong adhesions to the optic nerve head, macula, and blood vessels. This can lead to the painless appearance of "floaters," which can be fibroglial tissue from the optic nerve head ("Weiss" or "Vogt" ring), or proteinaceous condensations. Vitreous hemorrhage ("hemorrhagic PVD") or retinal tears/detachments are also possible.



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