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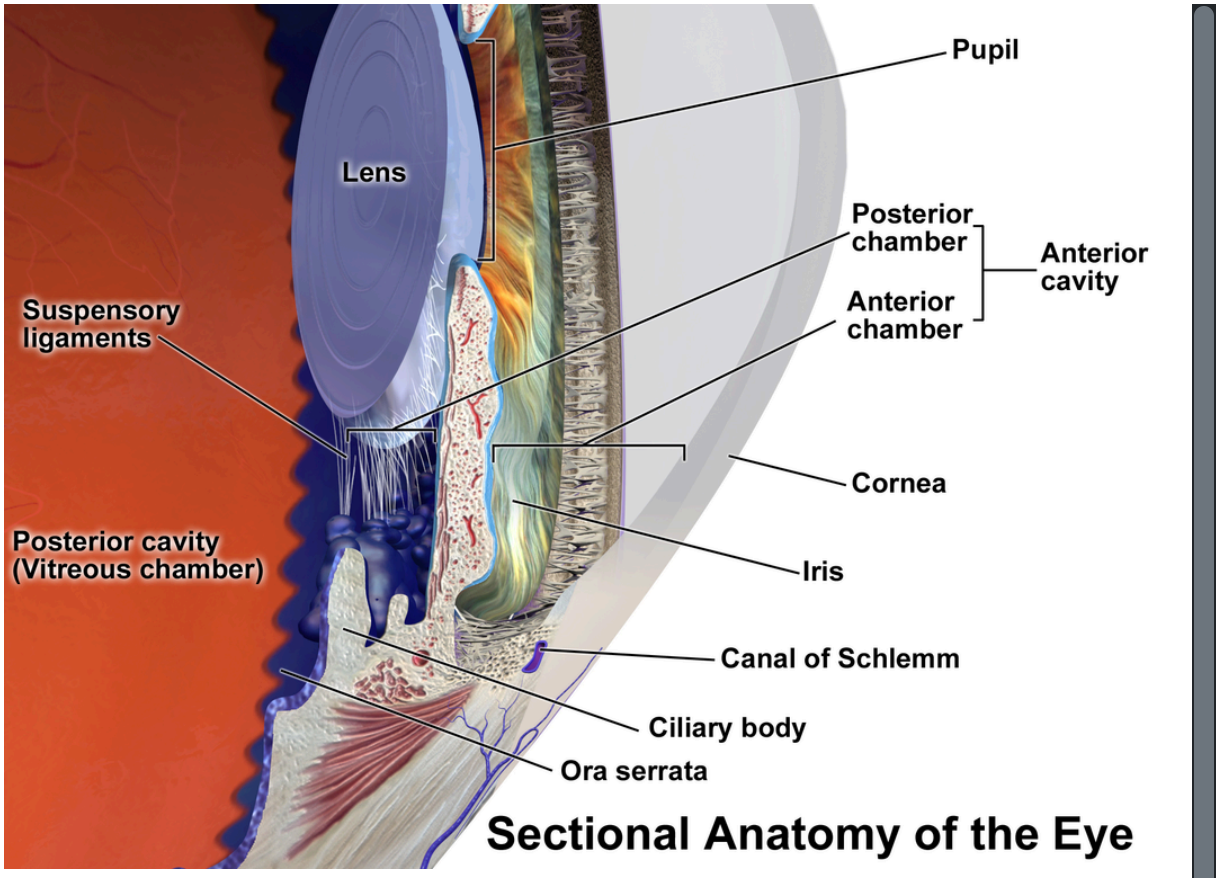
Glaucoma

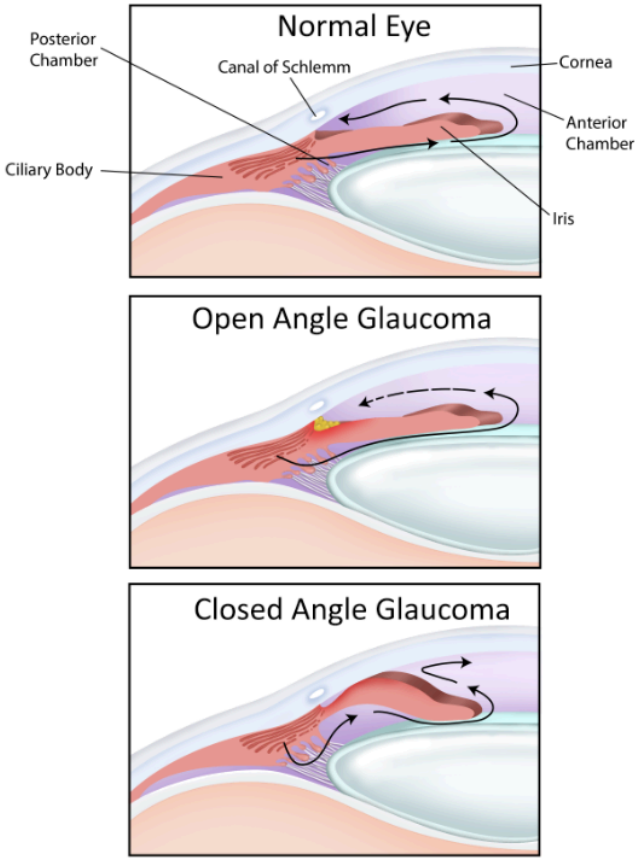


Title: File:Blausen File:Blausen 0388 EyeAnatomy 01.png and 0390 EyeAnatomy Sectional.png; Author: Blausen.com staff. "Blausen gallery 2014". Wikiversity Journal of Medicine. DOI:10.1534/wjm/2014.010. ISSN 20018762; Site: https://commons.wikimedia.org/wiki/File:Blausen_0388_EyeAnatomy_01.png and https://commons.wikimedia.org/wiki/File:Blausen_0390_EyeAnatomy_Sectional.png; License: This file is licensed und the Creative CommonsAttribution 3.0 Unportedlicense.

Glaucoma is higher than normal pressure in the anterior cavity of the eye due to some complication in draining the **aqueous humor (AH)** found within this compartment. Normal **intraocular pressure (IOP)** ranges from 12-22 mmHg. Values higher than this are considered glaucoma. Due to the high risk of damage to the optic nerve, most eye doctors will recommend prompt treatment if intraocular pressure is above 28 mm Hg.

AH flows through the anterior cavity as follows:

1. Ciliary body (where aqueous humor is made)
2. Posterior chamber
3. Iris
4. Anterior chamber
5. Trabecular network
6. Canal of Schlemm
7. Venous blood

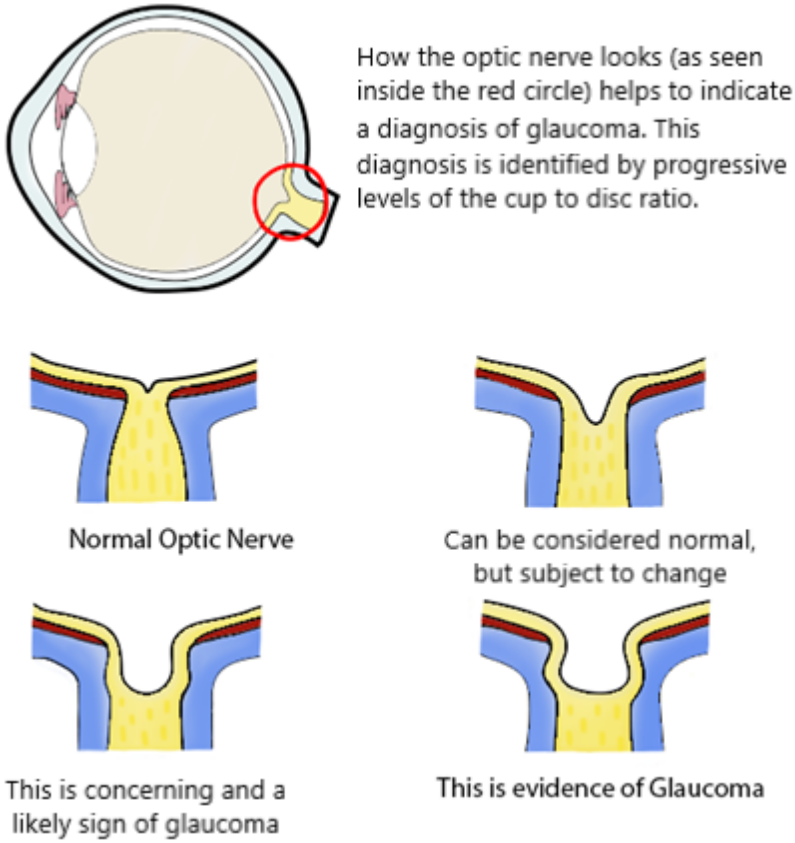


Types of Glaucoma *Image by Becky T. BYU-I W20*

There are two causes for higher than normal pressure in the anterior cavity. The first is **open angle glaucoma**. This typ is more common and is caused by abnormalities that block the trabecular meshwork that drains AH. The second type **closed angle glaucoma** (also called angle closure glaucoma) and is caused by an obstruction of the outflow of AH fro the anterior chamber due to a narrow angle. This narrowed angle occurs when the iris bulges forward and narrows or even blocks the drainage angle formed between the iris and the cornea. Closed angle glaucoma has a more sudden onset and is characterized by a very high IOP. An acute attack of angle-closure glaucoma is more likely to occur with conditions that lead to pupillary dilation (dark conditions) because AH flow is even more blocked when the dilator pupillae contracts. Some clinical manifestations of an acute attack due to angle-closure glaucoma include episodes o unilateral eye pain and headache, conjunctival redness, and blurred vision with halos around lights.

High IOP can damage the optic disc (i.e. the ganglion cell axons leaving the eye). To evaluate the health of the optic nerve, the **cup to disc ratio** is measured. The disc is the circular yellowish area on the retina that can clearly be seen o a fundoscopic evaluation. The center of the disc contains a smaller and brighter area referred to as the “cup.” The cup disc ratio compares the diameter of the cup portion with the diameter of the entire disc. A normal cup to disc ratio is 0 (meaning that the cup is about a 1/3 of the size of the disc). A high IOP causes damage to the axons and capillaries that run together on their way out of the eyeball through the optic disc. The axons closest to the vitreous humor are in greatest danger as they are in direct contact with the higher pressures and thus not likely to receive as much blood flo

from the choroid capillaries. As these axons die and regress, the cup gets bigger. When the ratio reveals that the cup to disc ratio approaches 0.6 (or nearly 2/3) then glaucoma is likely.



Signs of Glaucoma *Adapted from: https://www.wikidoc.org/index.php/Cup-to-disc_ratio; Creative Commons Attribution/Share-Alike License*

There are several drug classes that can be helpful in the treatment of glaucoma:

- **Beta-1 blockers:** activation of the beta-1 receptors on the ciliary epithelium promotes the production of AH, so blocking these receptors helps decrease AH production and lower IOP.
- **Cholinergic agonists:** these drugs stimulate muscarinic receptors that help constrict the iris which can benefit those with closed angle glaucoma because it opens up the anterior chamber angle.
- **Prostaglandin analogs:** This is a newer class of drugs. Cells of the trabecular meshwork are known to have PGF2 alpha receptors. As the prostaglandin analogs stimulate these receptors of the trabecular meshwork, they cause remodeling to occur that widens the spaces for aqueous humor to flow through to the venous drainage.
- **Alpha agonists:** The ciliary body also expresses alpha 2 receptors that when stimulated to inhibit aqueous humor production. Stimulation of alpha 1 receptors expressed on choroidal blood vessels can induce vasoconstriction which also decreases aqueous humor production.
- **Carbonic anhydrase inhibitors:** The cells of the ciliary body that make aqueous humor require Cl^- transported from the blood into the anterior chamber of the eye. This Cl^- contributes to the osmotic gradient that draws water into the cavity. The Cl^- gradient is developed as CO_2 in the blood diffuses into cells of the ciliary body from the blood. Carbonic anhydrase then catalyzes the conversion of CO_2 and water into carbonic acid which dissociates into H^+ and HCO_3^-. The HCO_3^- is then exchanged for Cl^- in the blood via an antiport protein. Blocking carbonic anhydrase decreases HCO_3^-, and thus the Cl^- gradient required for AH production.

Another treatment for elevated intraocular pressure associated with open-angle glaucoma is selective laser trabeculoplasty. This consists of using a laser to blast away some of the trabecular network in order to improve drainage and relieve pressure.