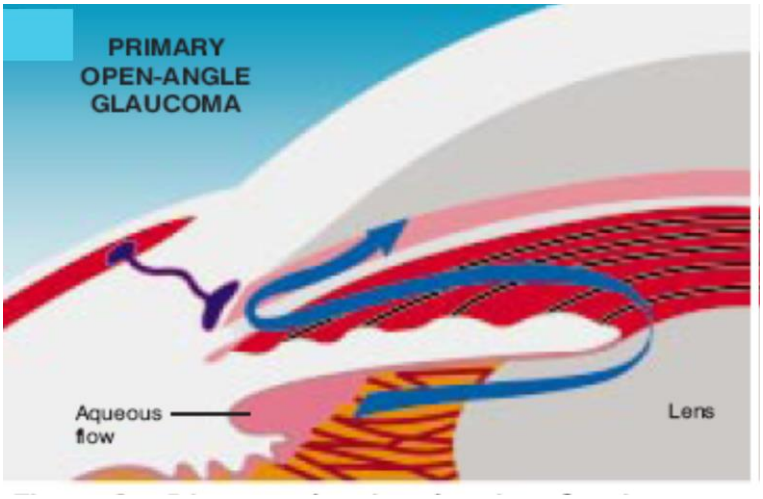
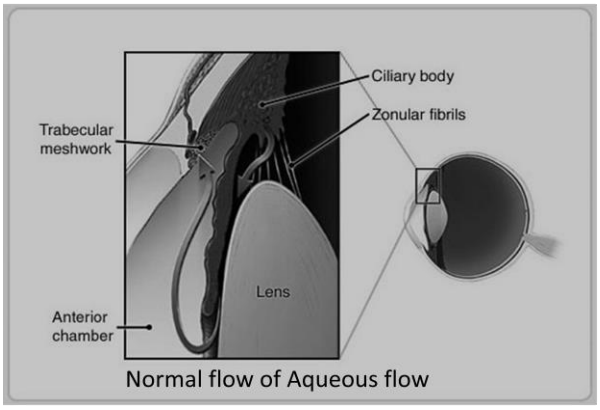
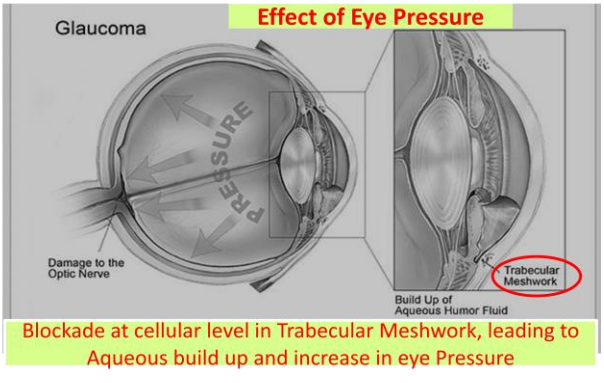
TYPES OF GLAUCOMA

There are two main types of glaucoma: Primary glaucoma and secondary glaucoma. Primary glaucoma has tow subtypes: open angle glaucoma and angle closure glaucoma.

Primary Open Angle Glaucoma: In "Open Angle" glaucoma increased resistance to outflow in the canal causes a rise in the pressure inside the eye. It is a long-standing slowly progressive disease process and can cause visual loss so quietly that the patient is unaware of the trouble till the advanced stage of the disease. There is a rise in the pressure of the eye, higher than what the eye can tolerate, for a long period of time. The optic nerve cannot withstand the prolonged periods of high pressure resulting in damage, i.e. loss of sight. The angles are open and the obstruction in fluid circulation is usually in the drainage channels. If not checked in time this form of glaucoma is reported to cause painless, progressive and total blindness. In open angle glaucoma, when disease process happens at normal eye pressure (IOP less than 22 mm Hg), it is called "normal tension glaucoma."

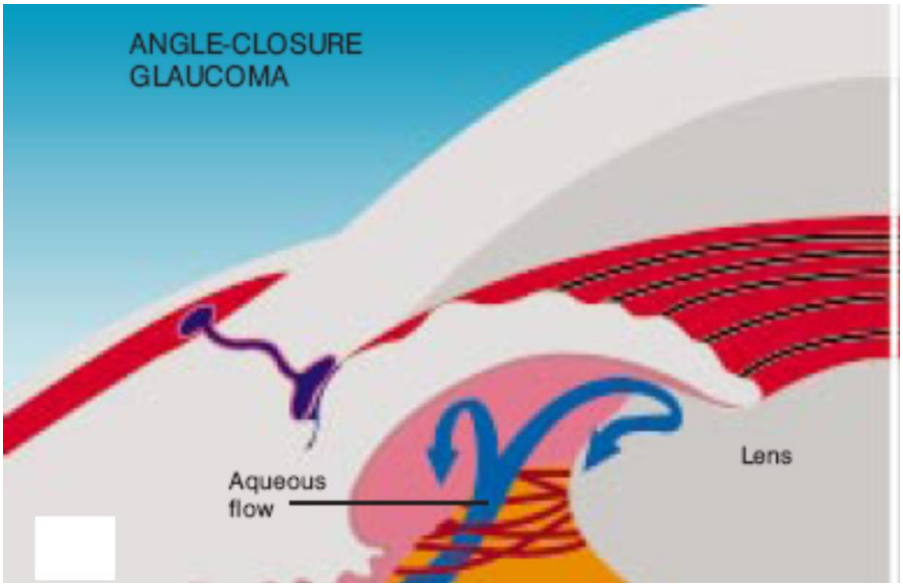






In India, nearly 60% of glaucoma patients have primary open angle glaucoma. This type of glaucoma is most common all over the world accounting for more than 90% of American & European population. In India, nearly 60% of glaucoma patients have primary open angle glaucoma. These patients manifest a chronic, idiopathic disease associated with progressive degeneration of the anterior optic nerve, known as glaucomatous optic neuropathy. Although elevated intraocular pressure is an important causative risk factor, only half of the 12 million Indians with glaucoma will manifest elevated intraocular pressure at a single measurement. Therefore, measurement of intraocular pressure alone is a poor screening technique for glaucoma. Like most biologic parameters, eye pressure fluctuates throughout the day and varies with other influences, including hydration, sleep, blood pressure and body position. With multiple measurements at different testing sessions, most, but not all of these glaucoma subjects, will eventually exhibit elevated intraocular pressure at least part of the time.
The rise in intraocular pressure associated with primary open angle glaucoma derives not from a visible obstruction of the trabecular meshwork, but rather from cellular dysfunction of the trabecular meshwork tissue, which leads to increased aqueous humor outflow resistance. Risk factors for primary open angle glaucoma include high intraocular pressure, family history, corticosteroid sensitivity, myopia, African-American race, systemic high blood pressure, diabetes, and age. In addition to these risk factors, early age of onset of disease and poor compliance with a medical regimen and physician visits are associated with a worse prognosis. As mentioned above, some patients with progressive optic nerve damage characteristic of glaucoma never manifest intraocular pressures above the statistically normal range. These patients are commonly diagnosed with "low pressure glaucoma," "low tension glaucoma," "or normal pressure glaucoma." While recognizing that non-pressure risk factors may play a stronger role in these than in than in their high-pressure counterparts, these patients are managed similarly to those with conventional primary open angle glaucoma.

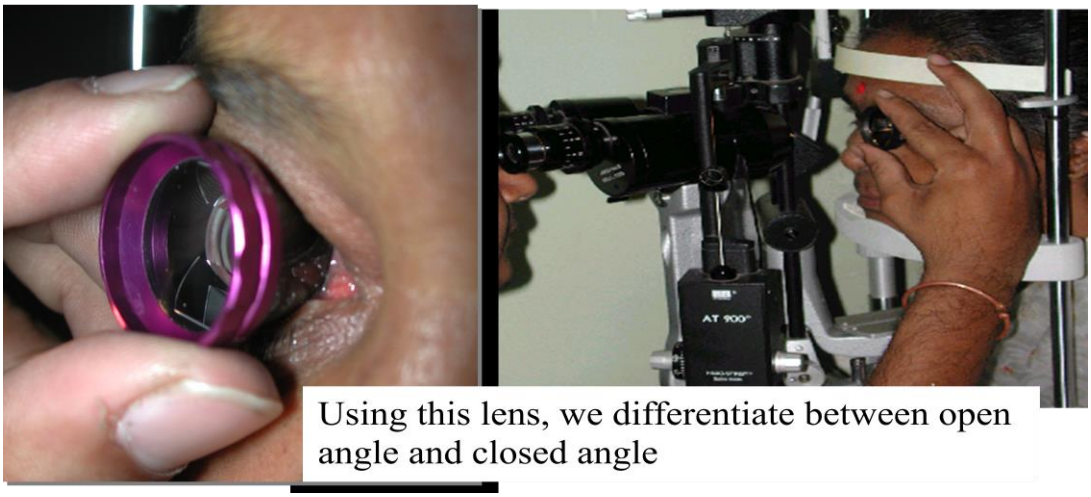
Primary angle closure glaucoma: There is a relative block to the flow of fluid through the pupil; this causes the iris to bow forwards, obstruct the canal and cause a raised pressure. Either mechanism can cause a rise in pressure capable of damaging a susceptible optic nerve. There are two subtypes: chronic angle closure and acute angle closure glaucoma.



In chronic angle closure, there is no symptom in early phase and behaves very similar to open angle glaucoma. The treatment is different and it is very important to diagnose the disease in earlier stage only.

Acute angle closure: As the name suggests its onset is quite sudden. The patient develops sudden blurring of vision with severe pain around the eye. The same patient may have had repeated episodes of a similar nature of lesser intensity which may have passed of unnoticed or with some treatment. Every attack mild or severe leaves behind some residual damage. At times the attack may be so severe that if not relieved promptly blindness can result in a matter of days.

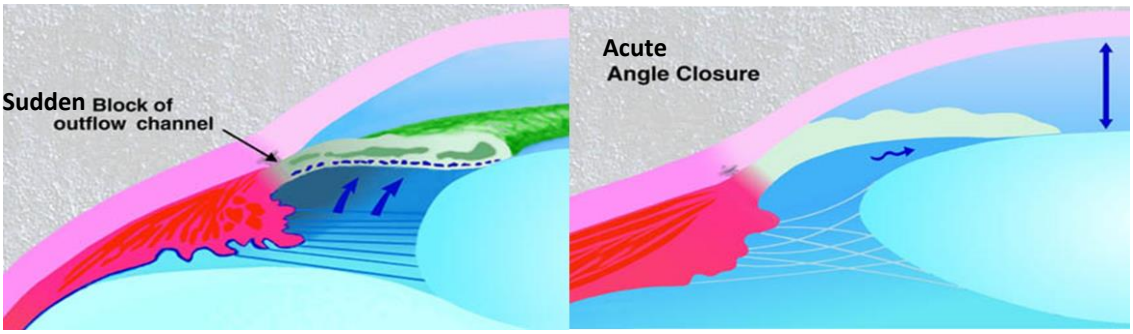
As the treatment of open angle and angle closure glaucoma is different, it is important to identify which mechanism is involved. (This can only be done with the contact lens 'gonioscope'.



Closed Angle Glaucoma

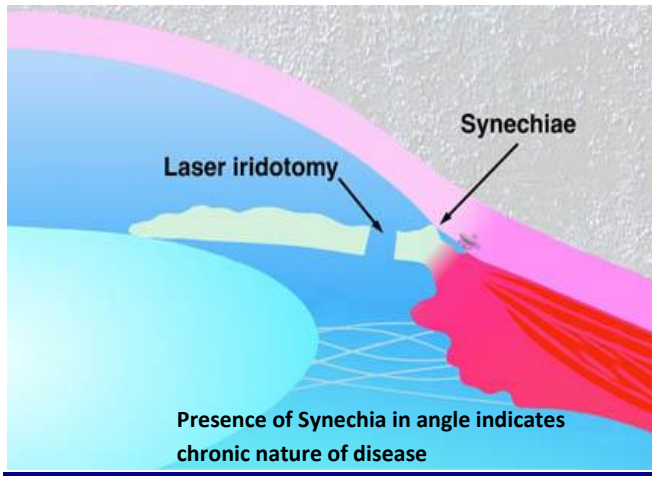
This may present acutely or may be silent and chronic. This disorder, quite unrelated to open angle glaucoma, derives entirely from blockade of the trabecular meshwork by the peripheral iris, either by simple and reversible anatomical apposition, or pressing together, of the two tissues or by generally irreversible scarring and adhesion. These irreversible fibrotic adhesions may occur after unrecognized long-standing appositional angle closure (chronic angle closure glaucoma) or from other ocular conditions, such as uveitis or neovascularization (secondary angle closure glaucomas).

Classically, acute angle closure glaucoma is the less common but well known variety of glaucoma that presents acutely with severe eye pain, blurring of vision, colored halos around lights, nausea and vomiting. Angle closure usually occurs in the hyperopic (farsighted) eye, which is smaller than the average eye and thus crowds the iris, cornea, lens and anterior chamber angle into a smaller than average space. Eventually, usually in the fifth to sixth decade of life as the lens gradually increases in size with aging, the lens becomes more firmly applied to the pupillary opening through which aqueous humor from the ciliary body must pass. This obstruction of aqueous humor flow at the pupil, known as relative pupillary block, eventually becomes clinically significant and traps the aqueous behind the pupil, raising the pressure in the posterior chamber above that in the anterior chamber and driving the iris anteriorly to lie against and block the trabecular meshwork. This trabecular meshwork blockade, or angle closure, leads to a sudden and dramatic rise of the intraocular pressure from its baseline normal level in the 10-20 mm Hg range to 60 mm Hg or more. This sudden change in pressure leads to swelling of the cornea with blurring, haloes, and severe ocular pain from iris ischemia and corneal edema. The pupillary margin of the iris becomes most tightly applied to the lens surface when the pupil is in the mid-dilated position; hence, it is often dilation of the pupil by exposure to stress, darkness, or drugs that precipitates an acute attack.



The immediate treatment of acute angle closure is directed toward reversal of the pupillary block, usually by moving the pupil with constriction. Ultimately, however, the pupillary block can be reversed and prevented by creating a new aqueous channel with peripheral iridectomy

In India, primary angle closure glaucoma is much more common than Caucasians and Africans; nearly 40% of overall glaucoma population has angle closure glaucoma. That means in patients with glaucoma, chances of having closed angle is 1.5:1 and the test we described, as Gonioscopy is very crucial for diagnosis of the disease and it's further management.



Congenital Glaucoma: Occurs in babies and children who are born with defective drainage channels. The defective outflow of fluid from within the eye gives rise to glaucoma. The child's eye appears cloudy and becomes abnormally large in size. If not diagnosed and treated, it leads to irreversible blindness at an early age. Some forms of congenital glaucoma may be hereditary and run in families. The treatment and visual rehabilitation of these children may at times be difficult.

What is Congenital Glaucoma?

Although glaucoma is commonly associated with adult and elderly, patients, glaucoma can rarely affects young child, known as childhood or infantile glaucoma. The most common, primary congenital open angle glaucoma occurs in children without other identifiable abnormalities. Most cases become evident in the first year of life and often are discovered in the newborn nursery or during the first few weeks of life. The exact cause of infantile glaucoma is unknown but appears related to a delay in development of the aqueous humor outflow channels.

Infants with congenital glaucoma present with photophobia (shyness to light), epiphora (tearing) and blepharospasm (blinking or squeezing the eyelids). The principal clinical sign is an enlarged cornea, often bilaterally. As the disorder advances, the cornea becomes edematous and appears cloudy. The appearance of an enlarged cloudy cornea in an infant essentially makes the diagnosis of congenital glaucoma and is obvious to casual penlight examination. Prompt referral to an ophthalmologist for intervention can make the difference between sight and permanent blindness.

Various forms of developmental glaucoma may also be associated with congenital malformations of the anterior chamber angle. Common is the Axenfeld-Rieger syndrome associated with dental, facial and other midline developmental abnormalities, adhesions between the cornea and iris, and glaucoma. Aniridia is a bilateral congenital absence of iris that may be inherited by autosomal dominant transmission or may occur spontaneously. These latter, spontaneous cases, may be associated with kidney tumors or other anomalies of the genito-urinary system. Glaucoma with aniridia usually occurs in early to mid childhood and is not typically associated with enlarged corneas.

Secondary Glaucoma: This type of glaucoma occurs secondary to some other disease process in the eye e.g. diabetes, trauma, bleeding, inflammation, tumour, hypermature cataract, etc. The treatment is primarily aimed at controlling the main disease process. Very often once the primary condition is treated the glaucoma also gets controlled. In some cases however there may be residual damage to the drainage channels causing glaucoma even after the primary process is controlled and may require long-term treatment.

How is Glaucoma Associated with Ocular Trauma?

Glaucoma may develop after ocular trauma. Penetrating injuries to the globe disrupt, or even destroy, intraocular contents and may lead to sustained elevation of intraocular pressure and glaucoma. (see Ocular Trauma) A more subtle, insidious glaucoma may arise from blunt

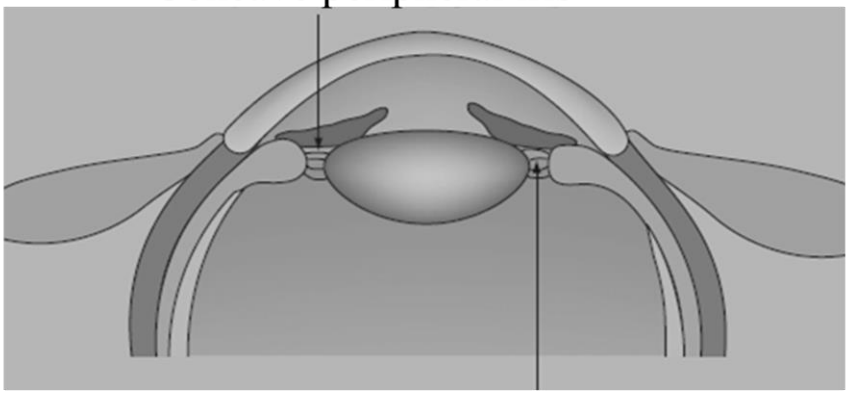
ocular injury or ocular contusion, as occurs when the globe is struck with a fist, ball or other object. Blunt injury temporarily deforms the globe, causing shearing between its internal tissue layers. These shearing forces may tear the insertion of the iris (iridodialysis) or ciliary body (cyclodialysis) from its attachment to the sclera. Most commonly, the fibers of the ciliary muscle that both controls accommodation and modulates aqueous humor outflow become detached, leading to collapse of the trabecular meshwork (known as angle recession) and subsequent secondary glaucoma.

Acutely, the contused eye typically presents with intraocular bleeding (hyphema), and the intraocular pressure may be low, normal, or elevated. Angle recession glaucoma may not manifest for months or even years after the original injury. Treatment of glaucoma from blunt ocular trauma follows a similar protocol to more common open angle glaucomas, except that these eyes do not respond well to pupil-constricting drops such as pilocarpine because of the damage of the ciliary muscle. Because of the damage to the trabecular meshwork, laser trabeculoplasty is similarly ineffective. Thus, when regular glaucoma drops are ineffective, filtration surgery usually becomes necessary.

What is Pigmentary Glaucoma?

Pigmentary glaucoma is relatively common secondary glaucoma in the young adult and appears to be exclusively an ocular disorder. This disease is relevant because of the potentially severe consequences in young people. Pigmentary glaucoma occurs primarily in young myopic (nearsighted) adults and is more common in males, usually manifesting between the ages of 20 and 40 years. Melanin pigment granules from the iris circulate freely in the aqueous humor, become deposited or entrapped in the surrounding tissues of the cornea, iris, lens, and particularly within the trabecular meshwork. This leads to obstruction of the meshwork, elevation of intraocular pressure, and glaucoma. This condition may manifest with intermittent visual blurring or dull ocular pain, but like other glaucomas may go unnoticed until severe visual loss occurs. Vigorous physical activity or pupillary dilation may induce a shower of pigment granules to be released from the iris in these patients, resulting in a transient, acute rise in eye pressure, corneal edema, blurred vision and ocular pain. Treatment of pigmentary glaucoma is similar to that for primary open angle glaucoma.

Concave peripheral iris



Mechanical rubbing of peripheral iris & anterior lens zonules

What is Neovascular Glaucoma?

Glaucoma is typically a disease of the middle aged and the elderly. Thus, its occurrence in children or young adults always raises the question of some associated condition, such as an intraocular tumor. Diabetes mellitus and other vascular disorders are frequently associated with neovascular glaucoma. The devastating consequences of diabetes upon the retina are well known. In addition to retina problems, the diabetic patient may also develop glaucoma as a result of retinal ischemia, known as neovascular glaucoma. Neovascular glaucoma is one of the most devastating varieties of glaucoma. Just as damage to the small blood vessels of the retina results in the formation of fragile new vessels and bleeding, retinal ischemia may also cause new vessels and scarring in the front part of the eye. It is believed that an angiogenic factor is produced by the ischemic retina, leading to new vessel formation inside the eye. Unfortunately, these vessels are abnormal and may cause a variety of vision-threatening problems. Neovascular glaucoma derives from proliferation of fragile new vessels on the iris (rubeosis irides) and into the anterior chamber angle. The trabecular meshwork provides a fertile template for neovascular growth, which ultimately leads to complete blockade of aqueous humor outflow, marked elevation of intraocular pressure and severe, often painful, blinding glaucoma. This process can also follow other vascular conditions associated with retinal ischemia, including occlusion of the central retinal vein, occlusion of the central retinal artery, and even carotid artery disease without known damage to the eye. Treatment of neovascular glaucoma is multifaceted and involves diligent systemic management of related vascular disease, treatment of retinal ischemia, and lowering of the intraocular pressure with medical and often surgical therapy.

Membrane formed by new abnormal vessels