

## Glaucoma - the Essentials

### Definitions/Introduction

*Glaucoma* - a group of diseases which result in characteristic optic nerve damage and visual field loss. It is often but not always associated with elevated intraocular pressure.

*Intraocular Pressure (IOP)* - The eye is a closed space. *Aqueous humor*, produced by the *ciliary body*, flows through the pupil into the anterior chamber and leaves the eye through the *trabecular meshwork* and *Schlemm's canal*. The pressure inside the eye is a result of the balance between inflow and outflow. Normal intraocular pressure ranges from 6mm Hg to 21mm Hg with an average of approximately 14mm Hg. Intraocular pressure varies diurnally and from day to day.

*Ocular Hypertension* - high intraocular pressure (>21 mmHg)

*Tonometry* - a method to measure pressure. The most common technique uses a Goldmann applanator attached to slitlamp. Other techniques include pneumo-tonometry ("puff of air" test) and a variety of handheld devices including the Tono-Pen and the older, now rarely used Schiotz tonometer.

### Primary Open-Angle Glaucoma (POAG) -

*Prevalence* - approximately one percent of all Americans. Second leading cause of blindness in US. Most important cause of blindness in African-Americans.

*Risk factors* - family history, African-American heritage, diabetes, age over 45.

*Symptoms* - Usually asymptomatic until late in disease

*Signs* - elevated intraocular pressure, normal appearing anterior chamber angle,

characteristic optic nerve damage (cupping) and characteristic visual field defects.

*Etiology/Pathophysiology* - the cause of the high intraocular pressure is unknown. Visual loss is due to damage to retinal nerve fibers which make up the optic nerve. The exact mechanism by which high intraocular pressure damages optic nerve fibers is unknown.

*Treatment* - treatment in all forms of glaucoma is directed toward lowering intraocular pressure to arrest further damage to the optic nerve. In POAG initial therapy consists of eye drops which act to decrease aqueous secretion, increase trabecular meshwork outflow or increase alternative outflow paths for aqueous humor.

When topical therapy fails the eye may be treated with *argon laser trabeculoplasty (ALT)*. In ALT, the trabecular meshwork is treated with laser and aqueous outflow is increased. The mechanism for improved outflow is not well understood.

Patients who fail topical therapy may also undergo *trabeculectomy* in which a fistula is created between the anterior chamber and the subconjunctival space allowing aqueous humor to bypass the trabecular meshwork on its way out of the eye.

### Most common types of glaucoma

Primary Open-Angle Glaucoma
Normal Tension Glaucoma
Pigmentary Glaucoma
Pseudo-exfoliation syndrome Glaucoma
Acute Angle-Closure Glaucoma
Trauma-Related Glaucoma
Uveitis-Related Glaucoma
Congenital Glaucoma
Neovascular Glaucoma

## **Normal Tension Glaucoma (NTG)**

*Prevalence* - unknown

*Symptoms* - identical to POAG

*Signs* - identical to POAG except for lack of elevated IOP

*Etiology/Pathophysiology* - unknown but theories abound --

Diurnal fluctuation in IOP

Artifactual low pressures due to thin corneas

True higher susceptibility to optic nerve damage from “normal” IOP

## **Pigmentary Glaucoma**

*Risk factors* - typically develops in 20's and 30's. Men more than women. More often in near-sighted patients.

*Symptoms* - usually same as POAG. Occasionally pt may notice blurred vision with exercise.

*Signs* - optic nerve and visual field changes identical to POAG, elevated intraocular pressure, iris transillumination defects, heavily pigmented trabecular meshwork

*Etiology/Pathophysiology* - rubbing of pigmented layer of iris against lens causes shedding of pigment which may clog the trabecular meshwork

*Treatment* - same as POAG. ALT appears to work better for these patients. Miotic therapy may also be appropriate.

## **Pseudo-exfoliation Syndrome Glaucoma**

*Risk factors* - over age 50, European or Russian descent.

*Symptoms* - identical to POAG but often unilateral

*Signs* - optic nerve and visual field changes identical to POAG, elevated intraocular pressure, “dandruff like” material deposited on lens iris and trabecular meshwork

*Etiology/Pathophysiology* - clogging of trabecular meshwork with the pseudoexfoliation material. The origin of the material is unknown.

*Treatment* - same as POAG. As in pigmentary glaucoma, ALT appears to work better for these patients.

## **Angle Closure Glaucoma**

*Prevalence* - less common than POAG, affects approximately half a million people in US

*Risk Factors* - hyperopia, Asian descent, age, family history, acute attacks may be precipitated by anything causing prolonged dilation ie prolonged time in dark, drugs with anticholinergic effects, dilation for eye exam, emotional stress

*Symptoms* - asymptomatic between acute attacks, during acute attacks patients experience intense pain, blurry vision and perhaps halos around lights

*Signs*

Between attacks - normal or elevated intraocular pressure (depending on chronicity), narrow anterior chamber angles, typical optic nerve damage and visual field defects

During acute attack - very high intraocular pressure, cloudy swollen cornea, conjunctival injection, intraocular inflammation, closed anterior chamber angles

*Etiology/Pathophysiology* - as mentioned above the trabecular meshwork is located in the angle where the cornea and iris meet. In most people the angle of approach is approximately 45 degrees. If this angle is decreased, the peripheral iris may block access to the trabecular meshwork. This is more likely to happen when the eye is dilated and the iris is crowded

peripherally. Age may play a part as the lens becomes thicker with age and may push the iris forward.

*Treatment* - a peripheral iridectomy (PI), a hole in the peripheral iris, usually created with a laser, is the definitive treatment and reestablishes flow from the posterior to anterior chamber. Patients with narrow (occludable) angles should have PI's placed prophylactically to prevent acute narrow angle glaucoma attacks. Patients with prolonged angle closure may form adhesions from the iris to the cornea permanently occluding the trabecular meshwork and causing a particularly intractable form of glaucoma.

### **Trauma-Related Glaucoma**

### **Uveitis-Related Glaucoma**

### **Congenital Glaucoma**

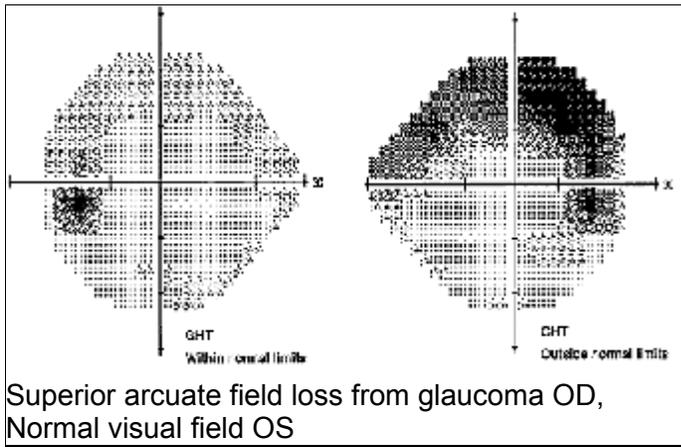
### **Neovascular Glaucoma**

Please read your text for descriptions of these types of glaucoma.

### **Visual field testing and glaucoma**

Early optic nerve damage results in characteristic patterns of peripheral vision loss. This visual field loss is usually too subtle to be noticed by the patient or to be picked up by “finger counting” visual field testing. Visual field testing is typically done using an automated *perimeter*. The patient sits in front of a dome. Lights of varying intensities appear around the dome. The patient is asked to press a button each time they detect a light. The perimeter keeps track of the patient's

threshold at each point in the visual field and presents it graphically for review and interpretation.

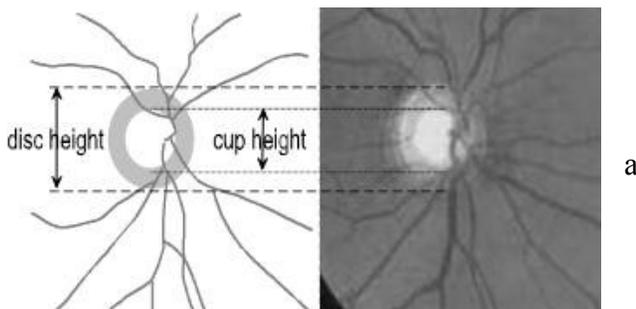


### Examination of the optic nerve

Examination of the optic nerve is the single most sensitive and specific way to detect glaucoma. Furthermore, it is currently the only practical way to screen for glaucoma in primary care setting.

Patients with cup to disc ratios of 0.4 or more and patients with cup to disc asymmetry of 0.2 or more require further evaluation.

Hemorrhage on the optic nerve may also be a sign of glaucoma. More subtle signs of a glaucomatous nerve include notching and pallor.



### Screening

The Glaucoma Foundation recommends this schedule to determine how often a patient should have an ophthalmologic exam to check for glaucoma.

Risk factors include : Family history, African-American heritage, myopia, diabetes, hypertension, long term steroid use, previous eye injury.

	No risk factors	Risk factors present
<b>Under age 45</b>	Every 4 years	Every 2 years
<b>45 and older</b>	Every 2 years	Every year

### Glaucoma Medications

Medication class	Examples	Mechanism of Action	Selected Side Effects
$\beta$ blockers	timolol, levobunolol, carteolol, metpranolol, betaxalol	decrease aqueous formation	asthma exacerbation, CHF exacerbation, bradycardia, heart block, fatigue, impotence, alopecia, lipid profile changes
miotics	pilocarpine	increase trabecular outflow	headache, poor dark adaptation, retinal detachment
$\alpha$ -adrenergic agents	apraclonidine, brimonidine	decrease aqueous formation	high topical allergy rate, drowsiness
carbonic anhydrase inhibitors	dorzolamide, brinzolamide	decrease aqueous formation	renal stones
prostaglandins	latanoprost, travaprost, bimatoprost	increase non trabecular outflow	red eye, turns some blue eyes brown, potentiates uveitis