

**Eye Care Skills: Presentations for Physicians  
and Other Health Care Professionals Version 3.0**

# **Glaucoma: Diagnosis and Management**

**Speaker Notes**

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 **AMERICAN ACADEMY  
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# A GUIDE TO PRESENTING

## Glaucoma: Diagnosis and Management

Glaucoma is the second most common cause of blindness in the United States and the single most important cause of blindness among African-Americans. Although approximately 3 million people have glaucoma, only 1 million are aware that they have the disease. About 80,000 Americans are blind from glaucoma.

*Glaucoma: Diagnosis and Management* provides primary care physicians with the latest information available about glaucoma. This program describes the types of glaucoma, with an emphasis on primary open-angle glaucoma and a secondary emphasis on angle-closure glaucoma. Also discussed are glaucoma risk factors, characteristic optic disc and visual field changes in POAG, and medical and surgical therapy of both primary open-angle and angle-closure glaucoma. This slide presentation also informs primary care physicians about the American Academy of Ophthalmology's EyeCare America Project, a program of professional and public education and public service that helps provide ophthalmologic examinations and, if needed, medical care to at-risk patients at no out-of-pocket cost to the patient.

### **Approximate Running Time**

50 to 90 minutes

### **Suggested Audience**

- Internists
- Family physicians
- Gerontologists
- Emergency physicians
- Medical students, interns, residents
- Emergency-room personnel (non-MD)
- State and local meetings of national medical societies: AAFP, AAP, ACP, ACEP
- Any primary care provider, nurse practitioner, etc.

# INTRODUCTION

## SLIDE

1

Glaucoma is the second most common cause of blindness in the United States and the single most important cause of blindness among African-Americans. Although approximately 3 million Americans have glaucoma, only 1 million are aware that they have the disease. About 80,000 Americans are blind from glaucoma.

## SLIDE

2

The prevalence of glaucoma is greatest in the elderly: 10% of African-Americans and 2% of Caucasians age 70 years or older have glaucoma. Age-adjusted prevalence rates are three to four times greater in African-Americans than in Caucasians. Other groups at increased risk for glaucoma are those with elevated intraocular pressures (ocular hypertension); those with first-degree family members, such as siblings, who have glaucoma; possibly those who have high myopia; and possibly those who have diabetes.

## SLIDE

3

Glaucoma may be defined as an optic neuropathy with characteristic optic nerve head and nerve fiber layer changes. With disease progression, characteristic visual field defects develop.

Introduction

## GLAUCOMA: PREVALENCE IN USA

- 3 million individuals with glaucoma
- 1 million unaware they have glaucoma
- 80,000 blind from glaucoma

Introduction

## GLAUCOMA: GROUPS AT RISK

- Elderly
- African-Americans
- Individuals with
  - Elevated IOP
  - 1st-degree relatives with glaucoma
  - High myopia?
  - Diabetes?

Introduction

## GLAUCOMA

Definition: an optic neuropathy with characteristic optic nerve head and nerve fiber layer changes

## PROGRESSION



## SLIDE

# 4

The characteristic optic nerve head changes include increased size of the central cup, thinning of the disc rim, progressive loss of neural rim tissue, disc hemorrhages, and loss of nerve fibers.

## SLIDE

# 5

The changes in the visual field may manifest as visual loss either in the nasal field, near the central field, or in the midperipheral field.

## SLIDE

# 6

Although elevated intraocular pressure (IOP) is a risk factor for glaucoma, the sensitivity, specificity, and positive predictive value of IOP screening for glaucoma is poor. Up to 50% of all patients with glaucoma may have pressures below 22 mm Hg at any given screening; therefore, glaucoma may remain undetected if these patients are screened on the basis of intraocular pressure alone. Because one-sixth of individuals with glaucoma may have intraocular pressures consistently below 22 mm Hg, an IOP of 22 mm Hg or higher is no longer part of the definition of glaucoma. In the past, a measurement of 21 mm Hg was considered the cutoff point for glaucoma because it is two standard deviations away from the mean IOP for the population in the United States, 16 mm Hg (assuming a normal, ie, Gaussian, distribution).

Introduction

## GLAUCOMA: OPTIC NERVE HEAD CHANGES

- Increased size of the cup
- Thinning of disc rim
- Progressive loss of neural rim tissue
- Disc hemorrhages
- Loss of nerve fibers

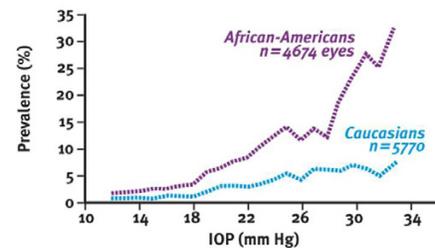
Introduction

## GLAUCOMA: VISUAL FIELD CHANGES

- Nasal field loss
- Paracentral field loss
- Midperipheral field loss

Introduction

## POAG: PREVALENCE IN RELATION TO SCREENING IOP



## SLIDE

7

The purpose of this presentation is to provide primary care physicians with the latest information about glaucoma and, through the American Academy of Ophthalmology's EyeCare America project, to enlist their support in our effort to eliminate unnecessary blindness due to undetected glaucoma.

Introduction



Reduce avoidable blindness and severe visual impairment.

# TYPES OF GLAUCOMA

## SLIDE

8

There are several types of glaucoma: primary open-angle glaucoma (POAG), angle-closure glaucoma, congenital glaucoma, childhood glaucoma, and secondary glaucoma. This program principally discusses primary open-angle glaucoma and angle-closure glaucoma.

Types of Glaucoma

## GLAUCOMA: TYPES

- Primary open-angle (POAG)
- Angle-closure
- Congenital
- Childhood
- Secondary

## SLIDE

9

Primary open-angle glaucoma is the most common type of glaucoma, accounting for 60% to 70% of all glaucoma cases. It is generally bilateral but not necessarily symmetric. POAG is distinguished from other glaucomas by the evidence of characteristic glaucomatous optic nerve and visual field damage; adult onset; open, normal-appearing anterior chamber angles; and the absence of secondary causes of open-angle glaucoma, such as trauma.

Types of Glaucoma

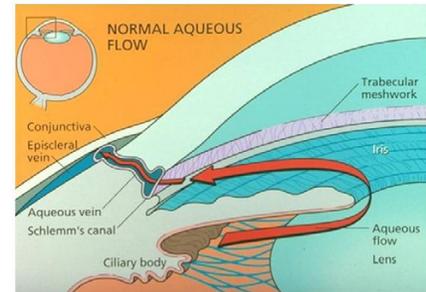
## POAG: CHARACTERISTICS

- Most common type of glaucoma
- Bilateral but not always symmetric
- Characteristic optic nerve and visual field damage
- Adult onset
- Open, normal-appearing anterior chamber angles
- Absence of secondary causes

SLIDE  
10

In the normal eye, aqueous humor is made by the ciliary body and flows through the pupil into the anterior chamber, where it is drained primarily through the trabecular meshwork to a canal leading to the venous system.

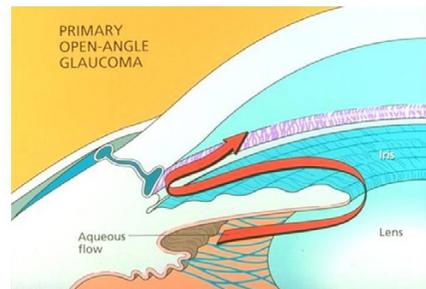
Types of Glaucoma



SLIDE  
11

Although the exact cause of primary open-angle glaucoma is not known, it seems to be related to increased resistance to outflow of aqueous through the trabecular meshwork. This progressive resistance to outflow results in a gradual increase in intraocular pressure, which may result in glaucomatous optic nerve damage in susceptible patients.

Types of Glaucoma



SLIDE  
12

In the early stages of the disease, the intraocular pressures are usually not high enough to cause noticeable symptoms, so most patients do not seek treatment. By the time the patient begins to notice visual loss the loss is usually marked. The speed with which optic nerve damage progresses varies with the patient, but left untreated, a large proportion of patients will suffer total optic nerve atrophy and blindness.

Types of Glaucoma

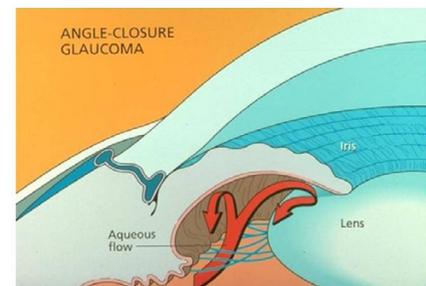
**POAG: PROGRESSION**

- Asymptomatic in early stages
- Often marked visual loss has occurred when patient presents with vision symptoms
- Can result in blindness

SLIDE  
13

Angle-closure glaucoma is the second most common type of glaucoma in the United States, accounting for approximately 10% of all glaucoma cases. It is characterized by appositional or synechial closure of the drainage angle of the eye. Because the angle closure prevents the aqueous from leaving the eye, the intraocular pressure may rise rapidly to high levels and damage the optic nerve.

Types of Glaucoma



**SLIDE**  
**14**

Congenital glaucoma presents in an infant as tearing, photophobia, an enlarged eye, and a hazy cornea. Infants may show signs of congenital glaucoma within days of birth, although some cases manifest later in the first year of life. It is more common in male infants and may be unilateral or bilateral.



Congenital glaucoma

**SLIDE**  
**15**

Childhood glaucoma is defined as glaucoma that presents in childhood or in adolescence. It includes cases of congenital glaucoma. Like primary open-angle glaucoma, it is asymptomatic in the early stages of disease and can result in total optic atrophy and blindness if untreated.

Types of Glaucoma

### CHILDHOOD GLAUCOMA

- Includes congenital glaucoma
- Asymptomatic in early stages
- Can result in total optic nerve atrophy and blindness

**SLIDE**  
**16**

Secondary glaucoma is glaucoma that occurs in patients with other ocular conditions. It can occur following severe ocular trauma or in conjunction with uveitis, chronic steroid use by systemic or topical administration, diabetic retinopathy, vascular occlusions, and other ocular conditions. These glaucomas require not only the management of intraocular pressure but also the management of the underlying condition.

Types of Glaucoma

### SECONDARY GLAUCOMAS: SOME CAUSES

- Trauma
- Uveitis
- Chronic steroid use
- Diabetic retinopathy
- Ocular vascular occlusion

# PRIMARY OPEN-ANGLE GLAUCOMA

## Risk Factors

SLIDE  
17

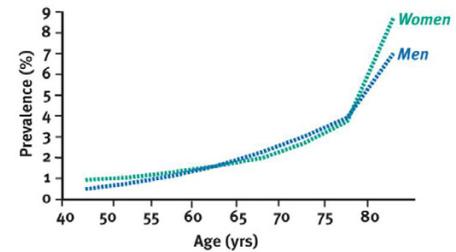
Age, race, and elevated intraocular pressure are all risk factors for glaucoma. The prevalence of primary open-angle glaucoma rises dramatically with age in all population-based epidemiologic studies. It is approximately 0.7% in people 40 to 49 years old and 8% for those over 80 years old. In addition, the proportion of Americans with intraocular pressures over 21 mm Hg becomes greater with age. Thus, the elderly are a high-risk group for high intraocular pressures and glaucoma

SLIDE  
18

Race is a major risk factor both for the presence of elevated intraocular pressures and for glaucomatous damage. Blindness from glaucoma is three to four times more common in African-Americans than in Caucasians.

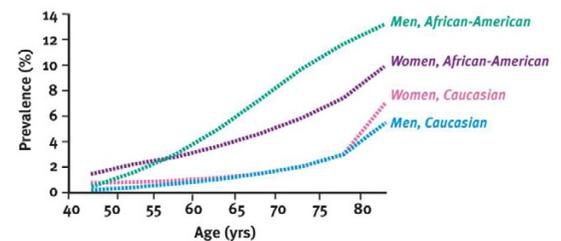
Primary Open-Angle Glaucoma

### POAG: PREVALENCE RELATED TO AGE



Primary Open-Angle Glaucoma

### POAG: PREVALENCE BY AGE, GENDER, AND RACE



SLIDE  
19

Glaucoma is not only more prevalent among African-Americans than Caucasians, but it also may occur at an earlier age and be more advanced when diagnosed in African-Americans. The reasons for the higher rate of glaucomatous blindness among African-Americans are as yet unknown, but they probably include some or all of the following: a greater susceptibility to damage of the optic nerve, a higher prevalence and earlier onset of elevated intraocular pressure, lower utilization of resources for detection and treatment, and less successful response to therapy.

SLIDE  
20

Although elevated intraocular pressure is a risk factor for developing glaucoma, there is no cutoff level of intraocular pressure that leads to glaucoma. Some individuals develop glaucomatous optic nerve damage with so-called normal intraocular pressures, while others with consistently elevated intraocular pressures do not develop damage. Although in population-based studies the presence of isolated, elevated intraocular pressures correlates poorly with the presence of glaucomatous optic nerve damage, the prevalence of glaucoma increases with increasing intraocular pressure, regardless of race. Thus, individuals with elevated intraocular pressures are at increased risk for developing glaucomatous optic nerve damage.

**POAG RISK FACTOR:  
AFRICAN-AMERICAN HERITAGE**

- Blindness = 3–4 times more common
- Age  $\rightarrow$  70 = 10% prevalence (2% for Caucasians  $\rightarrow$  70)
- POAG occurs at earlier age
- POAG more advanced when discovered

**POAG RISK FACTOR: ELEVATED  
IOP**

- High IOP correlates poorly with presence of optic nerve damage
- IOP level is related to POAG prevalence, regardless of race

**SLIDE**  
**21**

In addition to age, race, and elevated IOP, a positive family history of glaucoma is a major risk factor for the development of POAG. The mechanism is often complex and may be multifactorial. Listed on this slide are examples of the more than 10 genetic loci that have been mapped in families of patients with POAG, but only one gene has been identified for POAG, myocilin, which causes 3 to 5% of cases. The genetics of glaucoma is an area of intense ongoing research and may lead to strategies for early detection and treatment.

**SLIDE**  
**22**

In addition to Age and race, other risk factors for primary open-angle glaucoma include a family history of glaucoma in first-degree relatives; myopia; and decreased corneal thickness.

**POAG: EXAMPLES OF GENETIC FACTORS**

Genetic Locus	Chromosome
MYOC or TIGR gene (GLC1A)	1
GLC1B	2
GLC1C	3
GLC1D	8
GLC1E	10
GLC1F	7

**POAG: OTHER RISK FACTORS**

Factor	Relative Risk
Age (per decade >40)	2
African-American vs. Caucasian	4
Family history (1° relative)	2-4
Myopia	1.5-3
Decreased corneal thickness	3

# Screening for POAG

SLIDE  
23

As mentioned earlier, screening for glaucoma based only on intraocular pressure has low sensitivity, low specificity, and low positive predictive value. Screening can be made more effective by including an assessment of optic nerve status through examination of the optic disc, nerve fiber layer, and/or by visual field testing. Unfortunately, evaluation of the optic disc and visual field testing can be overly cumbersome and complex for routine population screening.

SLIDE  
24

Screening for glaucoma is necessary to give glaucoma patients the advantage of early treatment. In a study by the Baltimore Eye Survey Research Group at Johns Hopkins University, 50% of subjects with undiagnosed glaucoma were being seen regularly by primary care physicians. Many of these patients either had glaucoma risk factors that were unrecognized by their primary care physicians or could not obtain an ophthalmologic examination. Periodic, routine, comprehensive eye examinations of patients at risk may be the most efficient and cost-effective approach to detection and diagnosis of glaucoma. The American Academy of Ophthalmology recommends adult comprehensive eye examinations by an ophthalmologist at the frequencies shown here, with more frequent examinations in those at higher risk, including African-Americans and those with a family history of glaucoma in a first-degree relative.

Primary Open-Angle Glaucoma

## POAG: OTHER RISK FACTORS

Factor	Relative Risk
Age (per decade >40)	2
African-American vs. Caucasian	4
Family history (1° relative)	2-4
Myopia	1.5-3
Decreased corneal thickness	3

Primary Open-Angle Glaucoma

## RECOMMENDED FREQUENCY OF PERIODIC EYE EXAMINATIONS

Age	No Risk Factors	Risk Factors
20-29	At least once during interval	Every 3-5 years
30-39	At least twice during interval	Every 2-4 years
40-64	Every 2-4 years	Every 2-4 years
65+	Every 1-2 years	Every 1-2 years

**SLIDE**  
**25**

To help identify those individuals who should be screened for glaucoma, the AAO has developed a relative risk-factor weighting based on the best available epidemiologic data. This risk-factor analysis considers the variables of age, race, family history, and date of last complete eye examination and assigns a weighting value to each category of variable. Other historical variables, such as high myopia or hyperopia, systemic hypertension, steroid use, and, perhaps, diabetes, are not strong enough to be assigned a weight, but they may be considered in the overall assessment of glaucoma risk.

**SLIDE**  
**26**

The summing of the weights for each of the four risk categories results in an overall risk score. A score of 4 or greater signifies an individual at high risk for glaucoma, while a weight of 2 or lower signifies an individual at low risk for glaucoma. Subject to the physician's clinical judgment, all individuals with a score of 3 or higher would benefit from a comprehensive eye examination.

**RISK FACTOR WEIGHTING**

Variable	Category	Weight
Age	<40 years	0
	40-49 years	1
	50-59 years	2
	>60 years	3
Race	Caucasian/other	0
	Hispanic	1
	African-American	2
Family History of Glaucoma	- or + in non-1 <sup>st</sup> relatives	0
	+ for parents	2
	+ for siblings	4
Last Complete Eye Examination	Within past 2 years	0
	2-5 years ago	1
	>5 years ago	2

**RISK FACTOR ANALYSIS**

Level of Glaucoma Risk	Weighting Score
High	4 or greater
Moderate	3
Low	2 or less

**SLIDE**  
**27**

A comprehensive eye examination can also identify patients who are primary open-angle glaucoma suspects. A glaucoma suspect is defined as an adult who has normal visual fields but also has (1) elevated intraocular pressure, or (2) optic disc and/or nerve fiber layer with an appearance that is consistent with glaucomatous optic nerve damage, or (3) both of these attributes. Glaucoma suspects have open anterior chamber angles that appear normal. The susceptibility of the optic nerve to damage from glaucoma varies greatly from individual to individual and depends on the level of IOP and the presence and degree of other risk factors. Because we are currently unable to predict which glaucoma suspects will develop glaucomatous optic nerve damage, all glaucoma suspects should be reevaluated with an eye examination every 3 to 18 months.

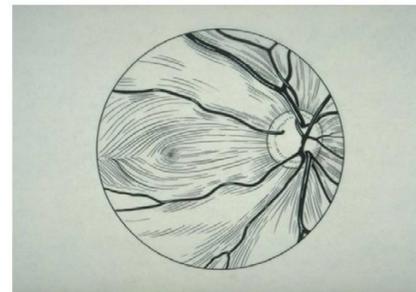
### GLAUCOMA SUSPECTS

Normal visual fields;  
Open, normal-appearing angles  
+  
Elevated IOP and/or  
abnormal optic disc/nerve fiber layer

## Optic Disc Evaluation

**SLIDE**  
**28**

Retinal nerve fibers, or axons, originate in the ganglion cells of the retina. These nerve fibers radiate in a linear or curvilinear pattern toward the optic nerve, with the majority of the fibers entering the optic nerve via the superior and inferior poles.

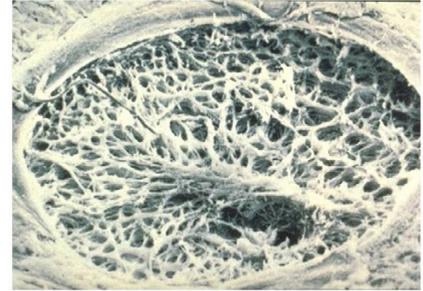


Pattern of retinal nerve fibers

SLIDE  
29

A healthy optic nerve has approximately 1.2 million nerve fibers. These nerve fibers pass through a sieve-like portion of the posterior sclera, called the lamina cribrosa, before exiting the eye. It is here that glaucomatous damage is thought to occur.

Primary Open-Angle Glaucoma

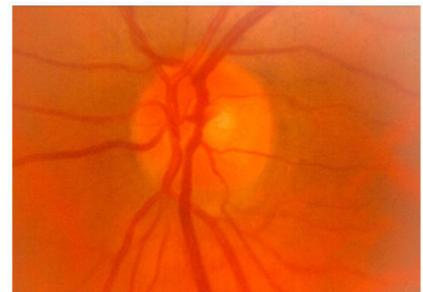


Lamina cribrosa

SLIDE  
30

The vast majority of optic nerves have a recognizable central depression or cup. An estimate of the ratio of cup diameter to disc diameter is best made vertically along an imaginary line drawn from the 12-o'clock to the 6-o'clock position through the center of the disc. The vertical disc diameter is arbitrarily set at 1.0 and then the relative cup diameter is estimated. The cup-to-disc ratio shown in this slide is 0.3. A cup-to-disc ratio of 0.5 or less has a lower level of glaucoma suspicion.

Primary Open-Angle Glaucoma

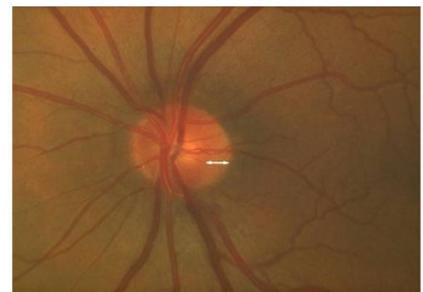


C:D ratio = 0.3

SLIDE  
31

The tissue between the border of the cup and the disc is the neuroretinal rim. This tissue consists mainly of nerve fibers with some glial cells and is usually pink. It tends to be symmetric at the superior and inferior margins of each disc.

Primary Open-Angle Glaucoma



Healthy neuroretinal rim

**SLIDE**  
**32**

Glaucomatous optic nerve damage involves the loss of axons. This nerve fiber loss is irreversible, and thus the blindness caused by glaucoma is irreversible. The nerve fibers most susceptible in glaucoma are those entering the optic nerve in the superior and inferior poles. Thus, one early sign of glaucoma is a thinning of the neuroretinal rim at the vertical poles, resulting in a vertical elongation of the cup.

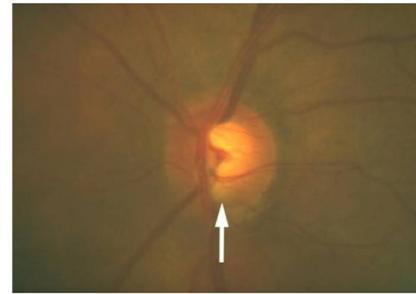
**SLIDE**  
**33**

Defects in the nerve fiber layer are among the earliest signs of glaucoma damage and may precede detectable changes in the nerve. Damage may be diffuse or focal, resulting in a groove or wedge defect, as seen here, or may be diffuse. The nerve fiber layer can be visualized in photographs or by ophthalmoscopy.

**SLIDE**  
**34**

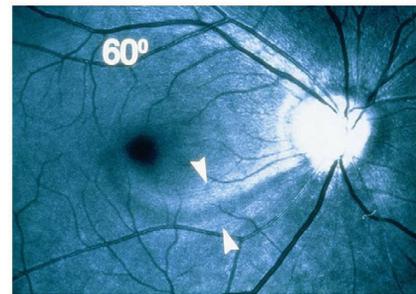
When evaluating patients for glaucoma it is important to evaluate both eyes, because the optic nerves of a healthy individual usually are symmetric, as shown here. Cup-to-disc ratios greater than 0.9 indicate a high level of glaucoma suspicion, while those of 0.6 to 0.8 indicate a moderate level. When using cup-to-disc ratios to assess a level of glaucoma suspicion, it is important to remember that African-Americans tend physiologically to have larger cup-to-disc ratios than Caucasians (based on a larger absolute disc size).

Primary Open-Angle Glaucoma



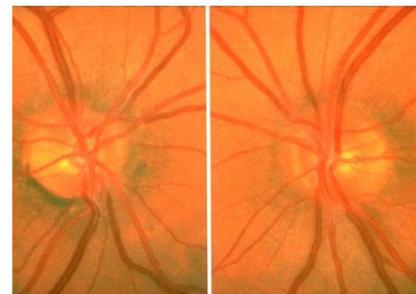
Thinning neuroretinal rim, elongated cup

Primary Open-Angle Glaucoma



Groove or wedge defect in nerve fiber layer

Primary Open-Angle Glaucoma

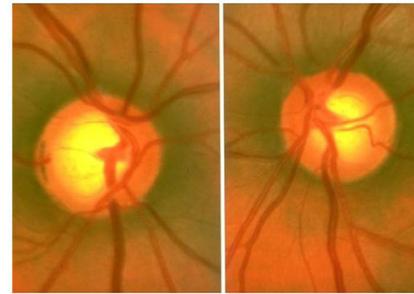


Healthy, symmetric optic nerves

**SLIDE**  
**35**

The left side of this slide shows a cup-to-disc ratio of 0.9 and the right side shows a cup-to-disc ratio of 0.7. Significant asymmetric cupping of the optic discs, as shown here, occurs in less than 2% of the normal population. Therefore, asymmetric optic nerve cupping may indicate glaucomatous optic nerve damage in the eye with the larger cup.

Primary Open-Angle Glaucoma



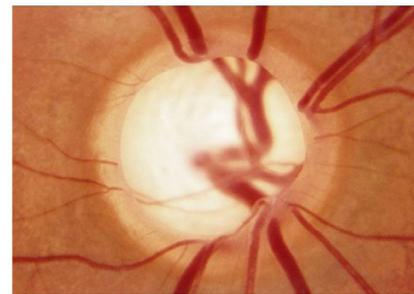
C:D ratio = 0.9

C:D ratio = 0.7

**SLIDE**  
**36**

More extensive glaucomatous damage shows increased cupping, further narrowing of the rim, increased pallor of the remaining neural tissue, heightened visibility of the pores of the lamina cribrosa, and displacement of the retinal vessels to the margin of the disc.

Primary Open-Angle Glaucoma



Extensive glaucomatous damage

**SLIDE**  
**37**

A disc hemorrhage is a flame-shaped hemorrhage near the disc margin. Although a disc hemorrhage can occur in a normal eye and in other disease states, it may suggest focal glaucomatous damage.

Primary Open-Angle Glaucoma



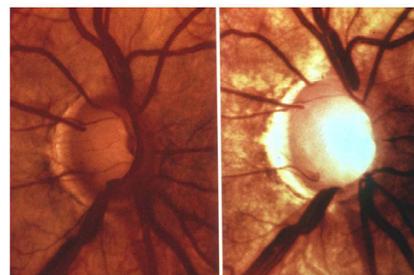
Flame-shaped disc hemorrhage

**SLIDE**  
**38**

An important feature of glaucomatous optic nerve damage is progressive neural tissue loss over time. These two photographs of the same optic nerve head were taken 12 years apart. One can detect a change in the cup and narrowing of the rim in the later optic nerve photograph shown on the right. In addition, there is increased displacement of the vessels toward the nasal disc margin.

Primary Open-Angle Glaucoma

**PROGRESSIVE NEURAL TISSUE LOSS**



Year 1

Year 12

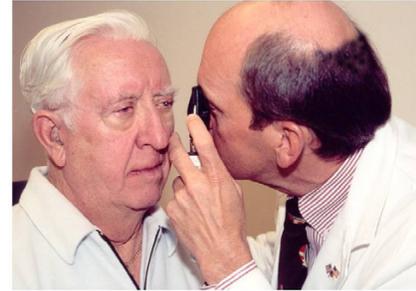
**SLIDE**  
**39**

The best way to begin evaluating the optic disc is to dilate the patient's pupils with, for example, 2.5% phenylephrine or 0.5% tropicamide. If pupillary dilation is not feasible, the room lights should be darkened. Set the dioptic power on the direct ophthalmoscope to zero, instruct the patient to fixate on a distant target, and elevate the patient's upper lid with your thumb.

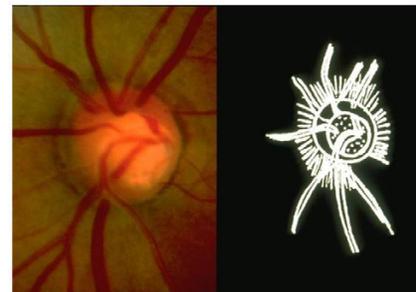
Gradually approach the patient's eye and aim the beam 15 degrees nasal to the patient's line of sight, toward the optic disc. As you approach, dial in whatever dioptic power is needed to keep the optic nerve in focus. If you cannot locate the optic nerve, ask the patient to look directly at or past your ear. You should assess cup-to-disc ratios and look for thin areas in the rim, disc hemorrhages, and asymmetry.

**SLIDE**  
**40**

Your evaluation of the optic disc should be recorded. This record can be a drawing of the appearance of the optic disc or a verbal description. In patients at high risk for glaucoma, baseline optic nerve photographs or images may be desirable and can be obtained in conjunction with a comprehensive ophthalmologic examination. These images are useful for future comparison to document change.



Evaluating optic disc

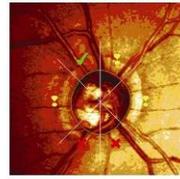


Record optic disc appearance in patient's chart.

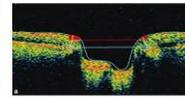
**SLIDE**  
**41**

Several imaging techniques have been developed to quantify the topography of the optic nerve head and to measure the thickness of the nerve fiber layer. This slide shows images of the optic nerve obtained with optical coherence tomography (OCT, middle). Other imaging techniques include confocal scanning laser ophthalmoscopy (left), and scanning laser polarimetry (right). The images from these devices are useful in the diagnosis of glaucoma and may be useful in the longitudinal follow-up of glaucoma patients.

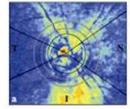
**OPTIC NERVE IMAGING: EXAMPLES OF NEW TECHNOLOGIES**



Confocal scanning laser ophthalmoscopy



Optical coherence tomography



Scanning laser polarimetry

## Evaluation of Visual Function

**SLIDE**  
**42**

Glaucoma is a disease of the optic nerve, and one of the best ways to measure disease of the optic nerve is to assess its function with visual field testing. A normal visual field of each eye usually spans over 120 degrees horizontally and 90 degrees vertically. This slide shows the field of view through a normal eye.



Normal visual field

**SLIDE**  
**43**

Characteristics of glaucomatous visual field damage include loss of vision in the nasal field (a nasal scotoma, or nasal step), loss of vision near the central field (a paracentral scotoma), or loss of vision in the midperiphery (arcuate scotomas). With undiagnosed and untreated glaucoma, these areas of lost vision can coalesce so that the patient loses all vision. This slide shows the field of view in an eye that has lost vision in the nasal field and midperiphery.



Nasal visual field and midperipheral vision loss

SLIDE  
44

In most cases, visual fields are tested using an automated perimeter, which can be used to standardize testing and print out the results.

SLIDE  
45

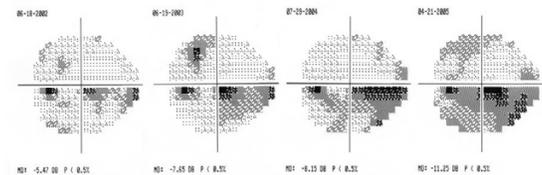
Patients diagnosed with glaucoma usually undergo visual field testing at regular intervals to assess optic nerve function. Depending on the severity and rapidity of progression of existing damage, visual field testing may be required as often as every 1 to 4 months, decreasing in frequency as stability of the visual field and optic nerve head appearance are demonstrated. This slide shows a printout of one patient's visual field test results over several years. Note the progressive enlargement of the scotoma (the dark areas), where vision is subnormal or absent.

SLIDE  
46

Additional functional tests, such as the pattern electroretinogram depicted here, are being developed to detect early glaucomatous damage. Tests that detect early glaucomatous damage need to be developed, since one-third or more of optic nerve fibers may already be destroyed by the time visual field abnormalities are reproducibly detected using current methods.



Automated perimetry



Visual field test results over time: progressive scotoma



Pattern electroretinogram

# TREATMENT OF POAG

## SLIDE 47

The goal of treatment of POAG is to stabilize visual field loss and optic nerve damage. To achieve this goal, we attempt to lower intraocular pressure—a *modifiable* risk factor, unlike gender, race, or age. In managing glaucoma, the ophthalmologist strives to achieve a stable range of pressures deemed clinically unlikely to cause further nerve damage in that particular patient. That range is referred to as the “target pressure.” The target pressure will vary among patients, and in the same patient it may vary during the course of disease. The target pressure needs to be periodically reassessed by comparing optic nerve status with previous data, since the target pressure is valid only if it actually prevents progression of POAG.

Treatment of POAG

## POAG: GOAL OF TREATMENT

- Halt further visual loss
- Halt further optic nerve damage

## Medical Therapy

## SLIDE 48

Topical glaucoma medications work by decreasing aqueous production or increasing aqueous outflow. Most are dosed one drop in the affected eye, once to three times a day. Side effects include local and systemic effects of the medications themselves, as well as local effects of the preservatives used.

Treatment of POAG

Glaucoma medications decrease  
intraocular pressure by  
decreasing aqueous production  
or  
increasing outflow.

SLIDE  
49

Topical beta-adrenergic antagonists decrease IOP significantly by decreasing aqueous production. Duration of action is 12 to 36 hours, so patients are treated with once-a-day or twice-a-day dosing. Most topical beta-adrenergic antagonists have yellow caps to make identification easier. Generic beta-adrenergic antagonists are frequently used.

SLIDE  
50

Commonly used beta blockers include timolol, levobunolol, carteolol, and metipranolol, which are nonselective, and the relatively selective beta-1 blocker betaxolol.

SLIDE  
51

Used as eye drops, beta blockers can produce serious systemic side effects, including worsening of congestive heart failure and bronchospasm, either of which can lead to death. In addition, bradycardia is not uncommon, and in the elderly, central nervous system side effects such as depression and confusion are commonly seen. Impotence and worsening of myasthenia gravis have been reported. Local ocular side effects are minimal.

SLIDE  
52

Topical adrenergic agonists lower resistance to outflow and may decrease aqueous production. Duration of action is 8 to 12 hours, so patients are treated with twice- or thrice-a-day dosing.

Treatment of POAG



Topical beta-adrenergic antagonists

Treatment of POAG

### TOPICAL BETA BLOCKERS

- Nonselective
  - Timolol (Timoptic, Betimol, Istalol)
  - Levobunolol (Betagan)
  - Carteolol
  - Metipranolol (OptiPranolol)
- Selective beta-1 blocker
  - Betaxolol (Betoptic-S)

Treatment of POAG

### TOPICAL BETA BLOCKERS: POTENTIAL SIDE EFFECTS

- Congestive heart failure
- Bronchospasm
- Bradycardia
- Depression, confusion
- Impotence
- Worsening of myasthenia gravis

Treatment of POAG



Topical adrenergic agonists

**SLIDE**  
**53**

Topical adrenergic agonists include epinephrine or derivatives, such as dipivefrin, and alpha-2 agonists, such as apraclonidine or brimonidine. Of these, brimonidine is the only one commonly used for long-term treatment

**SLIDE**  
**54**

Systemic side effects from topical adrenergic agonists can include decreased blood pressure, drowsiness, fatigue, and dry mouth. Ocular side effects include burning upon instillation, conjunctival injection, dilation of the pupils, and allergic reactions. Brimonidine is contraindicated in infants.

**SLIDE**  
**55**

Topical cholinergic agonists, or parasympathomimetics, increase aqueous outflow. Duration of action varies from 6 hours to 1 week, depending on the drug formulation.

**SLIDE**  
**56**

Cholinergic agonists are divided into two groups: short- and long-acting. Pilocarpine and carbachol are short-acting agents that affect the motor endplates in the same way as acetylcholine. Long-acting agents inhibit the enzyme acetylcholinesterase; echothiophate iodide, the representative agent in this class, is in limited availability.

Treatment of POAG

## TOPICAL ADRENERGIC AGONISTS

- Epinephrine (Epifrin)
- Dipivefrin (Propine)
- Alpha-2 agonists
  - Apraclonidine (Iopidine)
  - Brimonidine (Alphagan-P)
  - Combination brimonidine and beta-blocker (Combigan)

Treatment of POAG

## TOPICAL ADRENERGIC AGONISTS: POSSIBLE SIDE EFFECTS

- Systemic
  - Decreased BP
  - Drowsiness
  - Fatigue
  - Dry mouth
- Ocular
  - Burning on instillation
  - Conjunctival injection
  - Pupillary dilation
  - Allergic or toxic reactions

Treatment of POAG



Topical cholinergic agonists

Treatment of POAG

## TOPICAL CHOLINERGIC AGONISTS

- Short-acting
  - Pilocarpine (drops and gel)
  - Carbachol
- Long-acting
  - Echothiophate iodide (Phospholine Iodide; limited availability)

SLIDE  
57

Systemic side effects due to cholinergic agonists are related to their cholinergic actions and are much less common than systemic side effects from beta blockers. These include diaphoresis, stimulation of exocrine gland secretion, and smooth-muscle contraction. These side effects can lead to increased bronchial secretion, nausea, vomiting, and diarrhea. Since acetylcholinesterase inhibitors deplete pseudocholinesterase, prolonged respiratory paralysis may result after general anesthesia when succinylcholine is used. Ocular side effects of topical cholinergic agonists include ciliary muscle spasm, which can cause increased nearsightedness and brow pain, and pupillary constriction (miosis), which can cause decreased or darkening vision. Because of the frequent systemic and ocular side effects, this class of drugs is currently infrequently used.

SLIDE  
58

Carbonic anhydrase inhibitors decrease aqueous production by inhibiting ion transport associated with the secretion of aqueous humor. Adequate inhibition of carbonic anhydrase with systemic administration has been shown to cause a decrease in aqueous humor production by approximately 30%.

Treatment of POAG

## TOPICAL CHOLINERGIC AGONISTS: SIDE EFFECTS

- Systemic
  - Increased bronchial secretion
  - Nausea, vomiting
  - Diarrhea
  - Apnea\*
- Ocular
  - Increased myopia
  - Eye or brow pain
  - Decreased vision

\*With acetylcholinesterase inhibitors after succinylcholine administration

Treatment of POAG



Systemic carbonic anhydrase inhibitors

SLIDE  
59

Carbonic anhydrase inhibitors are available as oral or topical agents. Oral carbonic anhydrase inhibitors include acetazolamide, methazolamide, and dichlorphenamide. Topical carbonic anhydrase inhibitors include dorzolamide and brinzolamide.

SLIDE  
60

Topical carbonic anhydrase inhibitors are instilled two or three times a day. They have been associated with blurred vision, bitter taste, and conjunctival hyperemia. They cause fewer systemic side effects than oral agents. One topical carbonic anhydrase inhibitor, dorzolamide, is available in combination with 0.5% timolol, called Cosopt. It is used twice a day and is as effective in lowering IOP as dorzolamide and timolol used separately.

SLIDE  
61

Treatment with full doses of carbonic anhydrase inhibitors often causes a symptom complex consisting of malaise, anorexia, depression, and paresthesias. Carbonic anhydrase inhibitors also cause mild alterations in serum electrolytes, including decreased carbon dioxide, increased chloride, and decreased potassium. They also can cause renal calculi and, like all sulfa drugs, can cause blood dyscrasias, such as thrombocytopenia, agranulocytosis, neutropenia, and aplastic anemia. All these side effects are extremely rare with topical carbonic anhydrase inhibitors.

Treatment of POAG

## CARBONIC ANHYDRASE INHIBITORS

- Oral
  - Acetazolamide (Diamox)
  - Methazolamide (Neptazane)
  - Dichlorphenamide (Daranide)
- Topical
  - Dorzolamide (Trusopt)
  - Brinzolamide (Azopt)

Treatment of POAG



Topical carbonic anhydrase inhibitors

Treatment of POAG

## CARBONIC ANHYDRASE INHIBITORS: SIDE EFFECTS

- Malaise, anorexia, depression, paresthesias
- Serum electrolyte abnormalities
- Renal calculi
- Blood dyscrasias

SLIDE  
62

Latanoprost, travoprost, and bimatoprost are analogs of prostaglandin F<sub>2α</sub>. They act on prostanoid receptors and increase aqueous outflow. Duration of action is at least 24 hours, so patients are treated with one dose a day. The prostaglandin analogs are the most commonly prescribed glaucoma medications today.

SLIDE  
63

Side effects of prostaglandin analogs include conjunctival hyperemia, increased iris pigmentation, eyelash growth, and pigmentation of the periocular skin. The iris pigmentation change may be irreversible but is not associated with an increase in iris malignancies. The prostaglandin analogs should be used with caution in patients with a history of or risk factors for cystoid macular edema, due to a reported association with this condition. It should also be used with caution in patients with a history of uveitis.

SLIDE  
64

Eyelid closure and nasolacrimal occlusion can help reduce systemic side effects of topical medications by preventing their absorption through the nasal mucosa. Tears and eye drops reach the nasal mucosa via the nasolacrimal duct. This pathway is blocked with either nasolacrimal occlusion or sustained eyelid closure for 3 to 5 minutes after instillation of topical medications.

Treatment of POAG



Prostaglandin analogs

Treatment of POAG

### PROSTAGLANDIN ANALOGS: SIDE EFFECTS

- Conjunctival hyperemia
- +Iris pigmentation
- Eyelash growth
- Periocular skin pigmentation

**Use with caution in patients with cystoid macular edema and history of uveitis.**

Treatment of POAG



Inhibiting systemic absorption of eye drops by occluding lacrimal puncta

**SLIDE**  
**65**

The ability to lower IOP varies with the therapeutic agent and the compliance and responsiveness of the individual patient. Adequate control of glaucoma requires a high level of compliance, which frequently is not achieved. Studies indicate that one-third or more of glaucoma patients comply relatively poorly with treatment. Compliance can be improved with patient education and informed participation in treatment decisions.

**SLIDE**  
**66**

Medical therapy may fail to halt progressive visual field loss; this may be due to several factors: (1) target pressures may not be low enough, (2) patients may be noncompliant, or (3) patients on medical therapy may be more subject to wide fluctuations in IOP.

## Surgical Therapy

**SLIDE**  
**67**

Surgical therapy may or may not be the first choice in the management of glaucoma. The decision whether to proceed with surgery or with medications depends on the patient's medical condition and on a discussion of risks, benefits, and alternatives with the patient. Surgical therapy for glaucoma is usually successful in lowering IOP, although it is associated with greater ocular risks than medical therapy. Surgical therapies include laser trabeculoplasty, filtering surgery (trabeculectomy), drainage implant surgery, and cyclophotocoagulation.

Treatment of POAG

### **PATIENT COMPLIANCE IMPROVED WITH**

- Patient education
- Informed participation in treatment decisions

Treatment of POAG

### **WHY MEDICAL THERAPY MAY FAIL**

- Target pressures not low enough
- Poor patient compliance
- Fluctuations of IOP with medicines

Treatment of POAG

### **GLAUCOMA: SURGICAL PROCEDURES**

- Laser trabeculoplasty
- Filtering surgery (trabeculectomy)
- Drainage implant surgery
- Cyclophotocoagulation

**SLIDE**  
**68**

Laser trabecular surgery provides a clinically significant reduction of IOP in about 75% of initial treatments, but many patients continue to need topical medications after the laser surgery. The laser energy is applied to the trabecular meshwork for 180 or 360 degrees. Postoperative complications include inflammation and elevated IOP. Within 2 to 5 years, about 50% of patients will need additional medical or surgical therapy because of decay of IOP control. It is unlikely that laser trabeculoplasty will allow for the discontinuation of topical medications.

**SLIDE**  
**69**

Filtering surgery usually provides a dramatic, stable reduction in IOP. The initial success rate in a previously unoperated eye of an elderly Caucasian patient averages 75% to 85%, with or without adjunctive topical medications. Although long-term control may be achieved, many patients require additional operations. The success rate of additional operations is lower, but the use of local antifibrotics like 5-fluorouracil or mitomycin-C improves the success rate.

**SLIDE**  
**70**

Glaucoma drainage implant surgery is designed to shunt aqueous from the anterior chamber to a reservoir located at the equator of the eye. It is traditionally used in eyes in which filtering surgery has failed or with types of glaucoma known to be resistant to filtering surgery. The slide shows an eye with a drainage implant. Implants have a 1-year success rate of approximately 80%.

Treatment of POAG



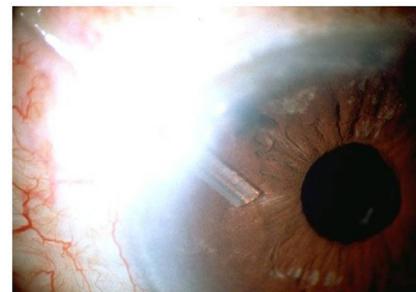
Laser trabecular surgery

Treatment of POAG

### **FILTERING SURGERY**



Treatment of POAG



Drainage implant

**SLIDE**  
**71**

When managing glaucoma patients, it is important to recognize the chronicity of the disease and the lack of a cure. Although vision loss from glaucoma is irreversible, aggressively lowering IOP can often halt disease progression. Recent large prospective clinical trials have demonstrated a 50% reduction in progression risk with a 20%–30% reduction in IOP.

**SLIDE**  
**72**

Some patients continue to lose vision despite the reduction of IOP to 5–10 mm Hg by medications and/or surgery. Researchers are currently looking for neuroprotective agents that will help protect ganglion cells and their axons from apoptosis.

Treatment of POAG

**Glaucoma can be controlled  
but not cured!**

Treatment of POAG

**POTENTIAL NEUROPROTECTIVE  
AGENTS**

- Protect ganglion cells from apoptosis
- Glutamate blockers

## ANGLE-CLOSURE GLAUCOMA

**SLIDE**  
**73**

Angle-closure glaucoma accounts for approximately 10% of glaucoma cases in the United States. The prevalence of angle closure differs among racial and ethnic groups. It is low among the Welsh in the United Kingdom and high among Eskimos and Asians. Prevalence also increases with age. It is more common in females and hyperopic patients, and a positive family history of angle-closure glaucoma is an additional risk factor.

Angle-Closure Glaucoma

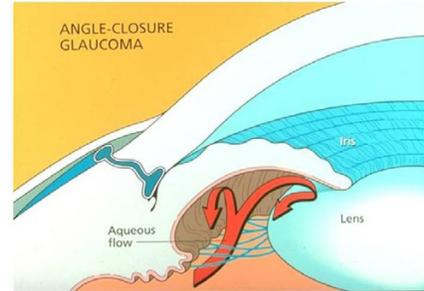
**ANGLE-CLOSURE GLAUCOMA:  
HIGH-RISK GROUPS**

- Elderly
- Hyperopic patients
- Positive family history of angle closure
- Females
- Eskimos
- Asians

**SLIDE**  
**74**

Angle-closure glaucoma occurs because of an excessive area of iris apposition to the lens, which impedes the flow of aqueous from the posterior chamber to the anterior chamber and thus to the trabecular meshwork. This is called relative pupillary block. The iris bows forward because of the higher pressure in the posterior chamber. The iris occludes the anterior chamber angle, aqueous does not reach the trabecular meshwork, the IOP rises, and the eye becomes inflamed.

Angle-Closure Glaucoma



**SLIDE**  
**75**

The individual who is predisposed to angle closure has anatomically narrow but still open angles. Narrow angles can be detected by directing the beam of a penlight parallel to the plane of the iris. If there is no shadow nasally, then the angles are most likely wide enough to dilate. Dilation with phenylephrine 2.5% is associated with minimal risk of precipitating angle-closure glaucoma. Systemic medications with anticholinergic side effects should be used with caution in patients with angle closure glaucoma or narrow angles because they cause the pupil to dilate and may precipitate an acute attack of angle closure.

Angle-Closure Glaucoma



Penlight examination of chamber angle

**SLIDE**  
**76**

The symptoms of acute angle-closure glaucoma are severe ocular pain and redness, blurred vision, halos appearing around lights (such as street lamps), headache, and nausea and vomiting. Angle-closure glaucoma is diagnosed with the aid of a gonioscope, which allows a view of the anterior chamber angle.

Angle-Closure Glaucoma

**ACUTE GLAUCOMA:  
SYMPTOMS AND DIAGNOSIS**

- Severe ocular pain, redness
- Blurred vision
- Halos around lights
- Headache
- Nausea and vomiting
- Diagnosis aided by gonioscope exam

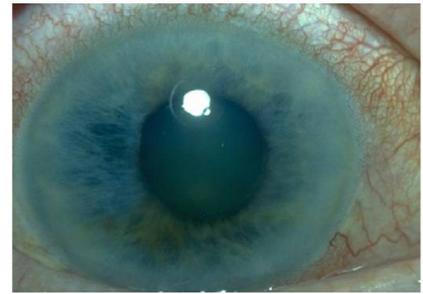


SLIDE

77

This slide shows an eye in acute angle closure. The conjunctiva is injected, the pupil is fixed and mid-dilated, and the cornea is hazy.

Angle-Closure Glaucoma



Acute angle-closure glaucoma

SLIDE

78

Whenever acute angle-closure glaucoma is suspected, the patient should be referred to an ophthalmologist immediately. The ophthalmologist can determine whether the elevated pressure, eye redness, and eye pain are due to angle closure or to another ocular disease.

Angle-Closure Glaucoma

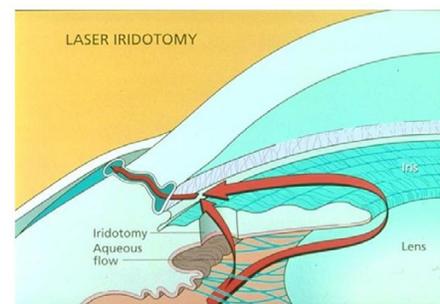
**Acute angle-closure glaucoma is an emergency!**

SLIDE

79

Once a diagnosis of angle-closure glaucoma has been established, the patient should receive medical treatment as soon as possible to lower the pressure and break the relative pupillary block. Acute angle-closure glaucoma is definitively treated, however, with a laser iridotomy, or a hole in the iris. This hole relieves the pupillary block mechanism by providing an alternate channel for aqueous to reach the anterior chamber angle and trabecular meshwork.

Angle-Closure Glaucoma



**SLIDE**  
**80**

Laser iridotomy may not be possible with uncooperative patients or patients with persistent corneal edema. With these patients, an incisional iridectomy can be done with the same result.

Angle-Closure Glaucoma



Incisional iridectomy

**SLIDE**  
**81**

If an ophthalmologist is not readily available to provide emergency care, the primary care physician should begin the necessary emergency procedures. After carefully evaluating the patient's history, symptoms, and signs and measuring the intraocular pressure, medical treatment to lower the intraocular pressure can be initiated.

Angle-Closure Glaucoma

**ACUTE GLAUCOMA:  
EMERGENCY MEDICAL CARE**

- Evaluate patient
- Measure IOP
- Initiate medical treatment

**SLIDE**  
**82**

Intraocular pressures may be measured with a Goldmann tonometer mounted on a slit lamp as shown on the left, or with hand-held devices such as a TonoPen (Medtronic Xomed, Inc.), as shown on the right.

Measurement of Intraocular Pressure



Goldmann applanation tonometer



Tonopen

**SLIDE**  
**83**

Treatment should include administering topical 2% pilocarpine drops in two doses, 15 minutes apart; timolol maleate 0.5% drops; and apraclonidine 0.5% drops; and acetazolamide, 500 mg orally or parenterally. A 20% solution of IV mannitol, 1.5–2 g/kg/body weight infused over 30–60 minutes should be given if there are no medical contraindications. The longer the intraocular pressure remains high, the greater the risk of permanent visual loss.

**SLIDE**  
**84**

The fellow eye should receive a prophylactic iridotomy if its chamber angle is narrow, because 58% to 75% of fellow eyes will suffer acute attacks. Chronic miotic therapy either for prophylaxis of the fellow eye or for treatment of established angle closure is not a lasting substitute for an iridotomy.

**SLIDE**  
**85**

Even after an iridotomy, long-term follow up of angle-closure glaucoma is indicated. Visual fields and optic nerves should be monitored for progressive damage, regardless of IOP.

**ACUTE GLAUCOMA:  
INITIAL TREATMENT**

- Pilocarpine 2% gtt q 15 min x 2
- Timolol maleate 0.5% gtt
- Apraclonidine 0.5% gtt
- Acetazolamide 500 mg po or IV
- IV mannitol 20%, 1.5–2 g/kg/body weight, infused over 30–60 min

**FELLOW EYES: MANAGEMENT**

- At risk for acute angle-closure glaucoma
- Prophylactic iridotomy if chamber angle is narrow
- Miotics are not a substitute for iridotomy

**Angle-closure glaucoma  
requires long-term follow-up.**

# CONCLUSION

SLIDE

86

The role of the primary care provider is vital in the diagnosis and management of glaucoma. With risk factor analysis, primary care providers can help high-risk patients receive appropriate care and diagnostic work-up. In addition, primary care providers are essential in helping to ensure that patients comply with medical therapy. Patients often forget that topical medications should be taken as prescribed. In addition, primary care providers can help monitor patients for poorly tolerated systemic side effects due to topical and oral glaucoma medications.

Conclusion

**Primary care providers are essential in the diagnosis and management of glaucoma.**

# APPENDIX 1

## EyeCare America

### SLIDE 87

By age 65, one in three Americans has some form of vision-impairing eye disease. As the population of older adults grows larger, it is estimated that the number of people with visual impairment will increase. EyeCare America, the public service foundation of the American Academy of Ophthalmology, is committed to the preservation of sight, accomplishing its mission through public service and education.

EyeCare America's public service programs provide eye care to the medically underserved and for those at increased risk for eye disease through its corps of 7,500 volunteer ophthalmologists. Public service includes programs for seniors, glaucoma, diabetes, and children; it is the largest program of its kind in American medicine. Since its inception, EyeCare America has helped more than 700,000 people and treated more than 180,000 cases of eye disease.

EyeCare America

### EYECARE AMERICA: COMMITTED TO THE PRESERVATION OF SIGHT



**SLIDE**  
**88**

The goal of the Glaucoma EyeCare Program is to reduce the incidence of unnecessary blindness by making both physicians and the public more aware of glaucoma risk factors and by providing ophthalmologic examinations to patients at no out-of-pocket expense. Although elevated intraocular pressure is a major risk factor for glaucoma, a single intraocular pressure measurement is a poor predictive screening test. The Glaucoma EyeCare Program emphasizes evaluation of other risk factors for glaucoma as the best way to identify patients in need of more extensive testing. Besides elevated intraocular pressure, the strongest risk factors for glaucoma are age, family history of glaucoma, and African-American racial heritage.

**SLIDE**  
**89**

Glaucoma EyeCare aims to increase awareness of glaucoma risk factors and the diagnostic value of a comprehensive examination. Referred patients with health insurance will be billed according to the doctor's office procedures and are responsible for any fees not covered by insurance. Ophthalmologists participating in EyeCare America agree to accept insurance reimbursement as full payment for an initial eye examination on patients identified as being at risk for glaucoma. Patients without insurance will be seen without charge.

## THE GLAUCOMA EYECARE PROGRAM

### Designed for people who:

- Are US citizens or legal residents
- Have not had an eye exam in 12 months or more
- Are at increased risk for glaucoma (family history, race, age)
- Do not have eye care insurance through an HMO or the VA

## THE GLAUCOMA EYECARE PROGRAM

### Promotes early detection and treatment of glaucoma.

- Provides glaucoma eye exams to at-risk, eligible callers (family history, age, and race)
- Uninsured patients are examined at no charge
- Insured patients are billed and responsible for any co-payment and/or deductible

**SLIDE**  
**90**

Ophthalmologists participating in EyeCare America see qualified referred patients as soon as possible after the initial call. They personally provide or arrange provision of continuing care for uninsured patients diagnosed with glaucoma. Eligible seniors who have not seen an ophthalmologist in 3 or more years may be able to receive eye care at no out-of-pocket expense for up to 1 year through the Seniors Program.

**SLIDE**  
**91**

People may call the toll-free help line at **1-800-391-EYES (1-800-391-3937)** anytime, for themselves and/or family members and friends, to request free glaucoma educational materials and determine if they qualify for a glaucoma exam from one of EyeCare America's 7,500 volunteer ophthalmologists nationwide.

EyeCare America

**GLAUCOMA EYECARE PROGRAM:  
EXAM AND CONTINUING CARE**

- Patients seen as soon as possible after referral
- Continuing care for up to 1 year for eligible patients through Seniors Program

EyeCare America

**EYECARE AMERICA HELP LINE:  
INFORMATION AND PATIENT REFERRAL**

**1-800-391-EYES  
(1-800-391-3937)**

# APPENDIX 2

## Glaucoma Risk Factor Analysis

### **History-Based Risk Factor Weights**

<b>Variable</b>	<b>Category</b>	<b>Weight</b>
Age	<40 years	0
	40–49 years	1
	50–59 years	2
	>60 years	3
Race	Caucasian/other	0
	Hispanic	1
	African-American	3
Family History of Glaucoma	Negative or positive in non–first-degree relatives	0
	Positive for parent(s)	2
	Positive for sibling(s)	4
	Positive for parent(s) and sibling(s)	4
Last Complete Eye Examination	Within past 2 years	0
	2–5 years ago	1
	>5 years ago	2

Other historical variables, such as high myopia or hyperopia, systemic hypertension, steroid use, and perhaps diabetes, are not strong enough to be assigned a weight but may be considered in the overall assessment of glaucoma risk.

<b>Level of Glaucoma Risk</b>	<b>Weighting Score</b>
High	4 or greater
Moderate	3
Low	2 or less