PREFERRED PRACTICE PATTERN®





Primary Open-Angle Glaucoma

AMERICAN ACADEMY OF OPHTHALMOLOGY
The Eye M.D. Association

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As a service to its members and the public, the American Academy of Ophthalmology has developed a series of guidelines called Preferred Practice Patterns that **identify characteristics and components of quality eye care.** (See Appendix 1.)

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INTRODUCTION

The Preferred Practice Pattern® (PPP) guidelines have been written on the basis of three principles.

- Each PPP should be clinically relevant and specific enough to provide useful information to practitioners.
- Each recommendation that is made should be given an explicit rating that shows its importance to the care process.
- Each recommendation should also be given an explicit rating that shows the strength of evidence that supports the recommendation and reflects the best evidence available.

In the process of revising this document, a literature search of the Cochrane Library and PubMed was conducted on December 3, 2008 and April 28, 2009 on the subject of primary open-angle glaucoma (POAG) for the years 2004 to the date of the search. In addition, the evidence synthesis[1] prepared by the British National Collaborating Centre for Acute Care for the National Institute for Health and Clinical Excellence clinical guideline on Glaucoma: diagnosis and management of chronic open-angle glaucoma and ocular hypertension clinical guideline was reviewed.[2] Details of the literature search are available at www.aao.org/ppp. The results were reviewed by the Glaucoma Panel and used to prepare the recommendations, which they rated in two ways. The panel first rated each recommendation according to its importance to the care process. This "importance to the care process" rating represents care that the panel thought would improve the quality of the patient's care in a meaningful way. The ratings of importance are divided into three levels.

- Level A, defined as most important
- Level B, defined as moderately important
- Level C, defined as relevant but not critical

The panel also rated each recommendation on the strength of evidence in the available literature to support the recommendation made. The "ratings of strength of evidence" also are divided into three levels.

- Level I includes evidence obtained from at least one properly conducted, well-designed, randomized, controlled trial. It could include meta-analyses of randomized controlled trials.
- Level II includes evidence obtained from the following:
  - Well-designed controlled trials without randomization
  - Well-designed cohort or case-control analytic studies, preferably from more than one center
  - Multiple-time series with or without the intervention
- Level III includes evidence obtained from one of the following:
  - Descriptive studies
  - Case reports
  - Reports of expert committees/organizations (e.g., PPP panel consensus with peer review)

Evidence is that which supports the value of the recommendation as it relates to the quality of care. The committee believes that it is important to make available the strength of the evidence underlying the recommendation. In this way, readers can appreciate the degree of importance the committee attached to each recommendation, and they can understand what type of evidence supports the recommendation.

The ratings of importance and the ratings of strength of evidence are given in bracketed superscripts after each recommendation. For instance, "[A:II]" indicates a recommendation with high importance to clinical care [A], supported by sufficiently rigorous published evidence, though not by a randomized controlled trial [II].

The sections entitled "Orientation" and "Background" do not include recommendations; rather they are designed to educate and provide summary background information and rationale for the recommendations that are presented in the Care Process section. A summary of the major

recommendations for care is included in Appendix 2. Appendix 3 has an algorithm for the management of POAG. Appendix 4 contains the International Statistical Classification of Diseases and Related Health Problems (ICD) codes for the disease entities that the PPP covers.

ORIENTATION

DISEASE DEFINITION

Primary open-angle glaucoma is a progressive, chronic optic neuropathy in adults in which intraocular pressure (IOP) and other currently unknown factors contribute to damage and in which, in the absence of other identifiable causes, there is a characteristic acquired atrophy of the optic nerve and loss of retinal ganglion cells and their axons. This condition is associated with an anterior chamber angle that is open by gonioscopic appearance.

CLINICAL FINDINGS CHARACTERISTIC OF PRIMARY OPEN-ANGLE GLAUCOMA

Primary open-angle glaucoma is a chronic ocular disease process that is progressive, generally bilateral, but often asymmetric. It is associated with the following characteristics.

- Evidence of optic nerve damage from either, or both, of the following:
  - *Optic disc or retinal nerve fiber layer structural abnormalities*
    - Diffuse thinning, focal narrowing, or notching of the optic disc rim, especially at the inferior or superior poles
    - Documented, progressive thinning of the neuroretinal rim with an associated increase in cupping of the optic disc
    - Diffuse or localized abnormalities of the peripapillary retinal nerve fiber layer, especially at the inferior or superior poles
    - Disc rim or peripapillary retinal nerve fiber layer hemorrhages
    - Optic disc neural rim asymmetry of the two eyes consistent with loss of neural tissue
  - *Reliable and reproducible visual field abnormality* considered a valid representation of the subject's functional status
    - Visual field damage consistent with retinal nerve fiber layer damage (e.g., nasal step, arcuate field defect, or paracentral depression in clusters of test sites)[3]
    - Visual field loss in one hemifield that is different from the other hemifield, i.e., across the horizontal midline (in early/moderate cases)
    - Absence of other known explanations
- Adult onset
- Open anterior chamber angles
- Absence of other known explanations (i.e., secondary glaucoma) for progressive glaucomatous optic nerve change (e.g., pigment dispersion, pseudoexfoliation [exfoliation syndrome], uveitis, trauma, and corticosteroid use)

Primary open-angle glaucoma represents a spectrum of disease in adults in which the susceptibility of the optic nerve to damage varies among patients. While many POAG patients present with elevated IOP, a substantial minority with otherwise characteristic POAG may not have elevated IOP measurements.[4] The vast majority of patients with POAG have disc changes or disc and visual field changes,[5] but there are rare cases where there may be early visual field changes before there are detectable changes to the optic nerve.

The severity of glaucoma damage can be estimated using the following:

- Mild: optic nerve abnormalities consistent with glaucoma as detailed above and a normal visual field as tested with standard automated perimetry

- Moderate: optic nerve abnormalities consistent with glaucoma as detailed above, and visual field abnormalities in one hemifield that are not within 5 degrees of fixation as tested with standard automated perimetry
- Severe: optic nerve abnormalities consistent with glaucoma as detailed above, and visual field abnormalities in both hemifields and/or loss within 5 degrees of fixation in at least one hemifield as tested with standard automated perimetry

PATIENT POPULATION

The patient population consists of adults 18 or older with POAG.

ACTIVITY

Identification and management of a patient with POAG.

PURPOSE

To identify and treat POAG and to preserve visual function while minimizing adverse effects of therapy, thereby enhancing the patient's health and quality of life.

GOALS

- Document the status of optic nerve structure and function on presentation
- Estimate an IOP below which further optic nerve damage is unlikely to occur (see discussion of target pressure in the Care Process section)
- Attempt to maintain IOP at or below this target level by initiating appropriate therapeutic intervention(s)
- Monitor the structure and function of the optic nerve for further damage and adjust the target IOP to a lower level if deterioration occurs
- Minimize the side effects of treatment and their impact on the patient's vision, general health, and quality of life
- Educate and involve the patient and appropriate family members/caregivers in the management of the disease



BACKGROUND

EPIDEMIOLOGY

Primary open-angle glaucoma is a significant public health problem. It is estimated that 45 million people in the world have open-angle glaucoma (OAG).[6] Glaucoma (both open-angle and angle-closure) is the second leading cause of blindness worldwide, with approximately 8.4 million people blind from glaucoma.[6] Overall in 2004, the prevalence of POAG for adults 40 and older in the United States was estimated to be about 2%.[7] Open-angle glaucoma affects an estimated 2.2 million people in the United States, and that number is likely to increase to 3.3 million in 2020 as the population ages. However, large differences exist in the prevalence of glaucoma among different ethnic groups (see Table 1 and Figure 1). Overall, there appears to be a threefold higher prevalence of OAG in African Americans relative to non-Hispanic Whites in the United States.[7,8] It is also the leading cause of blindness in African Americans.[8] Further, the prevalence of OAG is even higher in Afro-Caribbeans relative to African Americans. Recent evidence on Hispanics/Latinos suggests that they have high prevalence rates of OAG that are comparable to African Americans.[9] There are no data on the prevalence of OAG in Asians in the United States.

TABLE 1 THE PREVALENCE OF DEFINITE OPEN-ANGLE GLAUCOMA AS REPORTED IN OTHER STUDIES

Study	Racial/Ethnic Group	Age-Specific Prevalence					
		Age Groups (yrs)					
		40–49	50–59	60–69	70–79	80+	Total
Baltimore Eye Study[1]	African American	1.27	4.15	6.19	8.88	12.87	4.97
Barbados Eye Study[2]	Afro-Caribbean	1.4	4.1	6.7	14.8	23.2	6.8
LALES	Latino	1.32	2.92	7.36	14.72	21.76	4.74
Proyecto VER[3]	Latino	0.50	0.59	1.73	5.66	12.63	1.97
Baltimore Eye Study[1]	NHW	0.18	0.32	1.53	3.33	1.94	1.44
Blue Mountains Eye Study[4]	NHW	0.4*		1.3	4.7	11.4	3.0
Visual Impairment Project[5]	NHW	0.5	1.5	4.5	8.6	9.9	3.4
Beaver Dam Eye Study[6]	NHW						2.1
Roscommon[7]	NHW		0.72	1.76	3.2	3.05	1.88

LALES = Los Angeles Latino Eye Study; NHW = non-Hispanic White

* The study combined ages 40–59 into one group.

NOTE: The studies reporting prevalence used different definitions of disease; therefore, caution should be exercised when comparing these studies.

SOURCE: Adapted with permission from Varma R, Ying-Lai M, Francis B, et al, Los Angeles Latino Eye Study Group. Prevalence of open-angle glaucoma and ocular hypertension in Latinos: the Los Angeles Latino Eye Study. Ophthalmology 2004;111:1445.

1. Tielsch JM, Sommer A, Katz J, et al. Racial variations in the prevalence of primary open-angle glaucoma. The Baltimore Eye Survey. JAMA 1991;266:369-74.
2. Leske MC, Connell AM, Schachat AP, Hyman L. The Barbados Eye Study. Prevalence of open angle glaucoma. Arch Ophthalmol 1994;112:821-9.
3. Quigley HA, West S, Rodriguez J, et al. The prevalence of glaucoma in a population-based study of Hispanic subjects: Proyecto VER. Arch Ophthalmol 2001;119:1819-26.
4. Mitchell P, Smith W, Attebo K, Healey PR. Prevalence of open-angle glaucoma in Australia. The Blue Mountains Eye Study. Ophthalmology 1996;103:1661-9.
5. Wensor MD, McCarty CA, Stanislavsky YL, et al. The prevalence of glaucoma in the Melbourne Visual Impairment Project. Ophthalmology 1998;105:733-9.
6. Klein BE, Klein R, Sponsel WE, et al. Prevalence of glaucoma: the Beaver Dam Eye Study. Ophthalmology 1992;99:1499-1504.
7. Coffey M, Reidy A, Wormald R, et al. Prevalence of glaucoma in the west of Ireland. Br J Ophthalmol 1993;77:17-21.

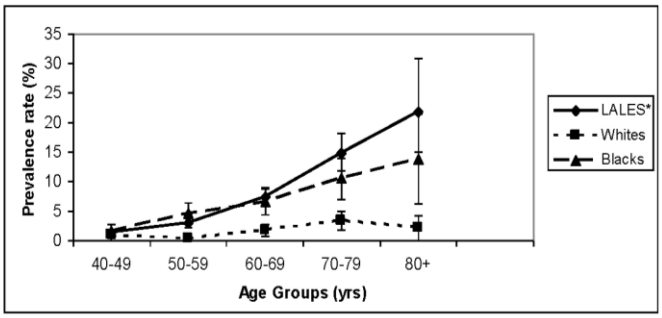


FIGURE 1. Comparison of age-specific prevalence of open-angle glaucoma in Latinos (Los Angeles Latino Eye Study), African Americans/Blacks and non-Hispanic Whites (the Baltimore Eye Study)[1]

* The data shown from LALES is from a different study.

SOURCE: Adapted with permission from Varma R, Ying-Lai M, Francis B, et al, Los Angeles Latino Eye Study Group. Prevalence of open-angle glaucoma and ocular hypertension in Latinos: the Los Angeles Latino Eye Study. Ophthalmology 2004;111:1446.

1. Tielsch JM, Sommer A, Katz J, et al. Racial variations in the prevalence of primary open-angle glaucoma. The Baltimore Eye Survey. JAMA 1991;266:369-74.

RISK FACTORS

The findings of epidemiological investigations and clinical trials provide a framework for assessing the risk factors associated with POAG. The important risk factors associated with POAG are as follows:

- Intraocular pressure level
- Older age
- Family history of glaucoma
- African ancestry or Latino/Hispanic ethnicity
- Thinner central cornea[10]
- Low ocular perfusion pressures[11,12]
- Type 2 diabetes mellitus[13-15]
- Myopia[12,16-18]
- Genetic mutations[19]

Intraocular Pressure

Several population-based studies have demonstrated that the prevalence of POAG[4,20-26] increases as the level of IOP increases (see Figures 2 and 3). These studies provide strong evidence that IOP plays an important role in the neuropathy of POAG. Furthermore, studies have demonstrated that reduction in the level of IOP lessens the risk of visual field progression in OAG (see Table 2).[27-32] In addition, treated eyes that have a greater IOP fluctuation may be at increased risk of progression, although this has not been shown consistently.[33-37]

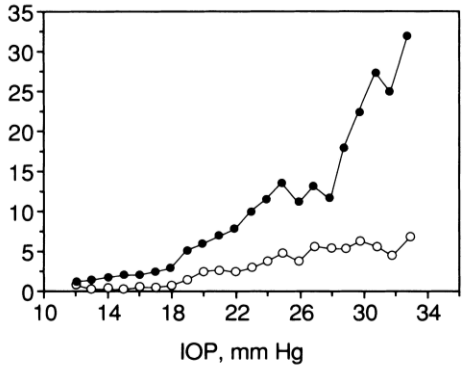


FIGURE 2. Prevalence of Primary Open-Angle Glaucoma in Relation to Screening Intraocular Pressure

African American subjects, n = 4,674 eyes (closed circles);

Caucasian American subjects, n = 5,700 eyes (open circles).

SOURCE: Sommer AE, Tielsch JM, Katz J, et al. Relationship between intraocular pressure and primary open angle glaucoma among white and black Americans. Arch Ophthalmol 1991;109:1092. Copyright 1991. Reprinted with permission from the American Medical Association. All rights reserved.

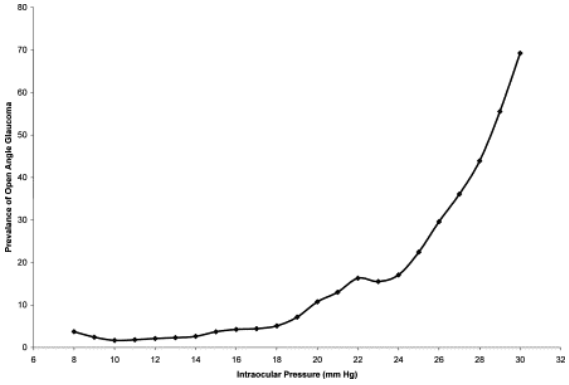


FIGURE 3. The relationship between prevalence of open-angle glaucoma and intraocular pressure (measured using Goldmann applanation tonometry) in Latinos (n=5970) in the Los Angeles Latino Eye Study.

SOURCE: Adapted with permission from Francis B, Varma R, Chopra V, et al, Los Angeles Latino Eye Study Group. Intraocular pressure, central corneal thickness, and prevalence of open-angle glaucoma: the Los Angeles Latino Eye Study. Am J Ophthalmol 2008;146:743.

TABLE 2 **RANDOMIZED CLINICAL TRIALS WITH PUBLISHED RESULTS**

Name	Study Design	No. of Patients	Follow-up Duration (years)	Finding
Scottish Glaucoma Trial[1,2]	Newly diagnosed POAG: medical therapy vs. trabeculectomy	116	4.6 (mean)	Trabeculectomy lowered IOP (-58%) more than medicine (-42%); medical therapy group had more deterioration in visual fields than trabeculectomy group.
Moorfields Primary Treatment Trial[3]	Newly diagnosed POAG: medical therapy vs. laser trabeculoplasty vs. trabeculectomy	168	5+	Trabeculectomy lowered IOP the most (-60%); laser trabeculoplasty (-38%) and medical therapy groups (-49%) had more deterioration in visual fields than trabeculectomy group.
Collaborative Normal-Tension Glaucoma Study[4]	POAG in eyes with normal IOP: rate of progression, effect of IOP reduction on progression rate	230	5+	Lowering IOP (-37%) retarded the progression rate of visual field loss compared with untreated eyes (-1%).
Early Manifest Glaucoma Trial[5,6,7]	Newly diagnosed POAG: medical therapy and laser trabeculoplasty vs. no treatment	255	8 (median)	Lowering IOP with medical therapy and trabeculoplasty (-25%) slowed progression of optic disc and visual field damage.
Collaborative Initial Glaucoma Treatment Study[8]	Newly diagnosed POAG: medicine vs. trabeculectomy	607	5+	Lowering IOP with initial filtering as surgery (-46%) was as effective as medical therapy (-38%) to inhibit progression of visual field damage, though the amount of reduction was slightly greater after surgery.
Advanced Glaucoma Intervention Study (AGIS)[9,10]	POAG after medical therapy failure with no previous surgery: laser trabeculoplasty vs. trabeculectomy	591	10–13	Surgical outcome varied by race; patients with African ancestry did better with laser trabeculoplasty as first surgery (-30% IOP), while in the longer term (4+ years) Caucasian American patients did better with trabeculectomy as first surgery (-48% IOP). Lowest IOP group during follow-up after surgical interventions (-47%) prevented further visual field deterioration in advanced glaucoma patients.

IOP = intraocular pressure; POAG = primary open-angle glaucoma

1. Jay JL, Allan D. The benefit of early trabeculectomy versus conventional management in primary open angle glaucoma relative to severity of disease. Eye 1989;3:528-35.
2. Jay JL, Murray SB. Early trabeculectomy versus conventional management in primary open angle glaucoma. Br J Ophthalmol 1988;72:881-9.
3. Migdal C, Gregory W, Hitchings R. Long term functional outcome after early surgery compared with laser and medicine in open-angle glaucoma. Ophthalmology 1994;101:1651-7.
4. Collaborative Normal-Tension Study Group. Comparison of glaucomatous progression between untreated patients with normal-tension glaucoma and patients with therapeutically reduced intraocular pressures. Am J Ophthalmol 1998;126:487-97.
5. Heijl A, Leske MC, Bengtsson B, et al, Early Manifest Glaucoma Trial Group. Reduction of intraocular pressure and glaucoma progression. Results from the Early Manifest Glaucoma Trial. Arch Ophthalmol 2002;120:1268-79.
6. Leske MC, Heijl A, Hussein M, et al, Early Manifest Glaucoma Trial Group. Factors for glaucoma progression and the effect of treatment. The Early Manifest Glaucoma Trial. Arch Ophthalmol 2003;121:48-56.
7. Leske MC, Heijl A, Hyman L, et al, Early Manifest Glaucoma Trial Group. Predictors of long-term progression in the Early Manifest Glaucoma Trial. Ophthalmology 2007;114:1965-72.
8. Lichter PR, Musch DC, Gillespie BW, et al, CIGTS Study Group. Interim clinical outcomes in the Collaborative Initial Glaucoma Treatment Study comparing initial treatment randomized to medications or surgery. Ophthalmology 2001;108:1943-53.
9. The AGIS Investigators. The Advanced Glaucoma Intervention Study (AGIS): 7. The relationship between control of intraocular pressure and visual field deterioration. Am J Ophthalmol 2000;130:429-40.
10. The AGIS Investigators. The Advanced Glaucoma Intervention Study (AGIS): 13. Comparison of treatment outcomes within race. Ten-year results. Ophthalmology 2004;111:651-64.

In spite of the relationship between the level of IOP and POAG, there is great interindividual variation in the susceptibility of the optic nerve to IOP-related damage. Population-based studies indicate that a variable proportion of patients with IOP greater than 21 mmHg (Northern Italy [13%],[38] Los Angeles [18%],[9] Arizona [20%],[23] Blue Mountains [25%],[21] Melbourne [39%],[39] Baltimore [45%],[40] Rotterdam [61%],[4] Barbados [71%][12]) have glaucomatous optic nerve damage.[20] This suggests that an IOP level of greater than 21 mmHg is an arbitrarily defined level and highlights the poor value of utilizing a specific IOP cutoff as a measure for screening and diagnosing POAG.

Age

Older age is another important risk factor for the presence of POAG.[21-23,39,40] A number of epidemiological studies demonstrate that the prevalence of glaucoma increases dramatically with age, particularly among individuals of Latino/Hispanic and African descent (see Table 1, Figures 1 and 4). African Americans 73 to 74 years old and 75 and older had a prevalence of 5.7% and 23.2%, respectively. Similarly, the prevalence of OAG was 3.4% for white individuals 73 and 74 years old and 9.4% for those 75 and older.[41]

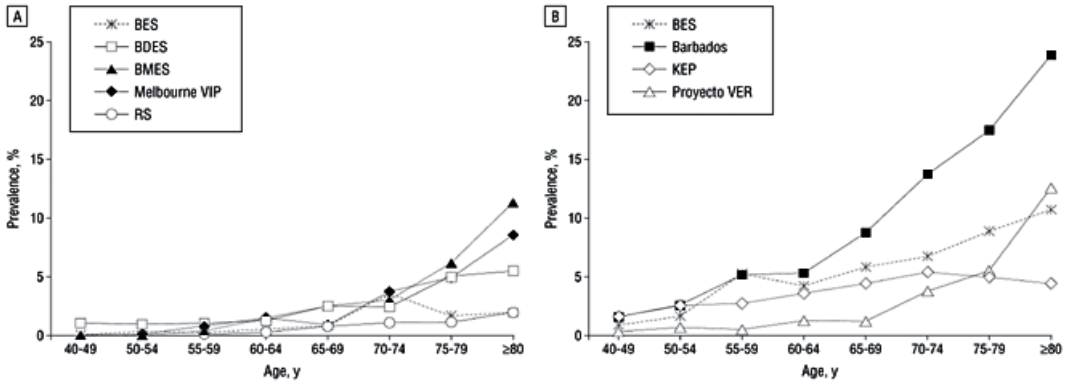


FIGURE 4. Prevalence of glaucoma in white (A) and black and Hispanic (B) subjects. BES indicates Baltimore Eye Survey,[1] Baltimore, Md; BDES, Beaver Dam Eye Study,[2] Beaver Dam, Wis; BMES, Blue Mountains Eye Study,[3] Sydney, New South Wales; Melbourne VIP, Melbourne Visual Impairment Project,[4] Melbourne, Victoria; RS, Rotterdam Study,[5] Rotterdam, the Netherlands; Barbados, Barbados Eye Study,[6] Barbados, West Indies; KEP, Kongwa Eye Project,[7] Tanzania; and Proyecto VER, Vision Evaluation Research,[8] Nogales and Tucson, Ariz.

SOURCE: Friedman DS, Wolfs RC, O'Colmain BJ, et al. Prevalence of open-angle glaucoma among adults in the United States. Arch Ophthalmol 2004;122:535. Copyright 2004. Reprinted with permission from the American Medical Association. All rights reserved.

1. Tielsch JM, Sommer A, Katz J, Royall RM, Quigley HA, Javitt J. Racial variations in the prevalence of primary open-angle glaucoma: the Baltimore Eye Survey. JAMA 1991;266:369-374.
2. Klein BE, Klein R, Sponsel WE, et al. Prevalence of glaucoma: the Beaver Dam Eye Study. Ophthalmology 1992;99:1499-1504.
3. Mitchell P, Smith W, Attebo K, Healey PR. Prevalence of open-angle glaucoma in Australia: the Blue Mountains Eye Study. Ophthalmology 1996;103:1661-9.
4. Wensor MD, McCarty CA, Stanislavsky YL, Livingston PM, Taylor HR. The prevalence of glaucoma in the Melbourne Visual Impairment Project. Ophthalmology 1998;105:733-739.
5. Wolfs RC, Borger PH, Ramrattan RS, et al. Changing views on open-angle glaucoma: definitions and prevalences: the Rotterdam Study. Invest Ophthalmol Vis Sci 2000;41:3309-3321.
6. Leske M, Connell A, Schachat A, Hyman L. The Barbados Eye Study: prevalence of open angle glaucoma. Arch Ophthalmol 1994;112:821-829.
7. Buhrmann RR, Quigley HA, Barron Y, West SK, Oliva MS, Mmbaga BB. Prevalence of glaucoma in a rural East African population. Invest Ophthalmol Vis Sci 2000;41:40-48.
8. Quigley HA, West SK, Rodriguez J, Munoz B, Klein R, Snyder R. The prevalence of glaucoma in a population-based study of Hispanic subjects: Proyecto VER. Arch Ophthalmol 2001;119:1819-1826.

Family History

Family history is a risk factor for glaucoma. In the Rotterdam Eye Study, where all siblings of glaucoma cases and controls were examined, the odds of having POAG was 9.2-fold higher for individuals who have a first-degree relative (sibling or parent) with medically confirmed POAG.[42] Other studies in which family members were not physically examined depend on patient reports of the status of family members, and these are known to be subject to several biases. Nonetheless, they support the concept that first degree relatives of those with OAG are at greater risk. For example, in the Baltimore Eye Survey and the Los Angeles Latino Eye Study (LALES), for individuals with POAG the odds were twice as high (1.92 and 2.85, respectively) of reporting a first-degree relative (parent, child, or sibling) with glaucoma compared with individuals who did not have glaucoma. However, the odds increased to over three times as high if they reported that they had a sibling with glaucoma (LALES 3.47,[43] Baltimore 3.7[44]). Interestingly, the odds rose to fivefold higher if there were two or more siblings who were reported to have a history of glaucoma.

Race or Ethnicity

For POAG, ethnicity is an important risk factor (see Figures 1 and 4). The prevalence of POAG is higher in individuals of West African, Afro-Caribbean, or Latino/Hispanic origin than of other groups.[9,22,23,40,45,46] The prevalence is three times higher in African Americans and Hispanics of Mexican ancestry compared with non-Hispanic Whites.[9,40] Blindness from glaucoma is at least six times more prevalent in African Americans than in Caucasian Americans.[8]

Central Corneal Thickness

Because applanation tonometry measurements are derived from resistance to corneal indentation and corneal stiffness, differences in central corneal thickness (CCT) may introduce artifacts in IOP measurement.[10,29,47-53] The mean CCT in healthy human eyes varies by race/ethnicity. The average CCT measured ultrasonically in Caucasian Americans is 556 μm, in Latinos it is 546 μm[54] and in African Americans it is 534 μm.[52] If IOP is underestimated in those with thinner CCT, the relationship between IOP level and OAG damage may be underestimated, since the IOP is actually higher than measured. Conversely, if IOP is overestimated in those with a nonedematous thicker CCT, the relationship between IOP level and OAG damage may be overestimated, since the IOP is actually lower than measured. Although several tables and figures have been published, no standard nomogram correcting applanation IOP measurements for CCT has yet been fully validated.[55,56]

A thinner central cornea has been reported as an independent risk factor (independent of IOP) associated with POAG,[57] though not in all studies. In LALES, the risk of having OAG was higher in persons with thinner CCT compared with those with normal or thicker CCT even after adjusting the IOP (see Figure 5).[58] Studies show that differences in corneal biomechanics across individuals may have a greater impact on IOP measurement errors than CCT.[59,60]

Low Ocular Perfusion Pressure

Ocular perfusion pressure is the difference between blood pressure (at systole or diastole) and the IOP. It has been hypothesized that low ocular perfusion pressures lead to alterations in blood flow at the optic nerve head and contribute to progressive glaucomatous optic nerve damage. Population-based studies in African Americans, Hispanics, and non-Hispanic Whites have provided evidence that low diastolic perfusion pressure (<50 mmHg) is associated with a higher prevalence of POAG.[11,23,61,62] In addition, in the Early Manifest Glaucoma Treatment Study, low systolic perfusion pressure (≤125 mmHg) was associated with a higher risk of glaucoma progression (relative risk of 1.42) over an 8-year period.[36]

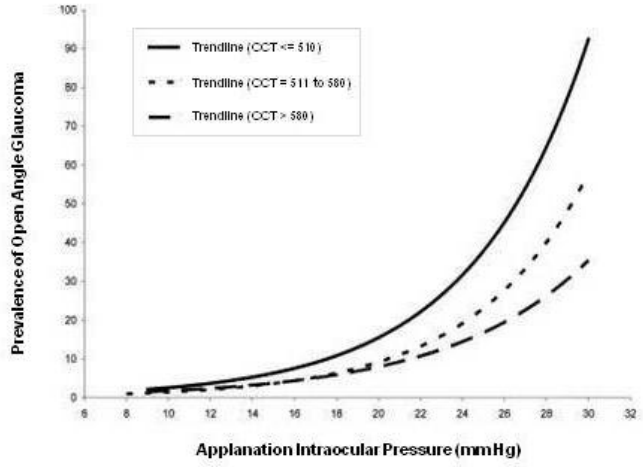


FIGURE 5. The relationship between the prevalence of open-angle glaucoma and applanation intraocular pressure stratified by central corneal thickness (CCT) in micrometers in the Latinos (n=5970) in the Los Angeles Latino Eye Study.

SOURCE: Adapted with permission from Francis B, Varma R, Chopra V, et al, Los Angeles Latino Eye Study Group. Intraocular pressure, central corneal thickness, and prevalence of open-angle glaucoma: the Los Angeles Latino Eye Study. Am J Ophthalmol 2008;146:743.

Type 2 Diabetes Mellitus

While there are some conflicting data on the association between type 2 diabetes mellitus (type 2 DM) and POAG,[13-15,63-69] there is increasing evidence from population-based studies suggesting that type 2 DM is an important risk factor for POAG.[13-15,65,67] Population-based assessments of Hispanics (in Los Angeles, California),[14] non-Hispanic Whites (in Beaver Dam, Wisconsin and Blue Mountains, Australia),[13,67] and a large cohort enrolled in the Nurses' Health Study[65] have shown that persons with type 2 DM are more likely (40% higher odds in Hispanics, twofold higher odds in non-Hispanic Whites) to have POAG. Further, in the LALES,[14] longer duration of type 2 DM was associated with a higher risk of having POAG. One explanation for this observation is that microvascular changes in the optic nerve may contribute to the greater susceptibility of optic nerve damage in persons with type 2 DM.[66]

Myopia

Large cross-sectional epidemiologic studies in Afro-Caribbeans, Hispanics, non-Hispanic Whites, Chinese, Asian Indians, and Japanese suggest that persons with myopia have a higher prevalence of OAG than those without myopia.[12,16-18,70-73] More recently, data from the LALES have provided evidence of an independent relationship between longer axial length (axial myopia) and a higher prevalence of OAG.[74] The underlying hypothesis is that individuals with axial myopia have weaker scleral support at the optic nerve, and this contributes to a greater susceptibility of the optic nerve to glaucomatous damage.

Genetic Factors

The first gene to be linked to POAG was the myocilin gene (*MYOC)* on chromosome 1.[75] In one study, 3% to 4% of cases with POAG were found to have mutations in the myocilin gene.[19] Several chromosomal regions have now been linked to POAG and additional genes identified.[76,77] However, a majority of the cases with POAG do not have an identified genetic abnormality, suggesting that glaucomatous optic nerve damage may be multifactorial in its development, with different genes modifying the impact of various factors such as age, IOP, and blood flow.

Other Factors

In addition, migraine headache and peripheral vasospasm have been identified as risk factors for glaucomatous optic nerve damage.[78-80] The association between POAG and factors such as concurrent cardiovascular disease and systemic hypertension has not been demonstrated consistently.[12,16,61,62,81-85]



POPULATION SCREENING FOR GLAUCOMA

Population-based screening for glaucoma is currently not cost-effective.[86] Screening may be more useful and cost-effective when it is targeted at populations at high risk for glaucoma, such as older adults,[7] those with a family history of glaucoma,[42,44,87-89] and African Americans and Hispanics.[7] Screening for glaucoma could be included in general screening for eye disease, especially among older populations. Once screening technologies improve, screening may be indicated for a wider population. In 2009, the U.S. Agency for Healthcare Research and Quality Effective Health Care Program funded a review of the comparative effectiveness of screening for glaucoma. These results, when available, will assist in identifying which screening methods should be used.

It is important to consider methods for screening the general population for POAG, because patients are asymptomatic until late in the disease process. It is possible to treat patients with POAG and to either slow or prevent the progression of visual field loss. Even mild visual field loss decreases health-related quality of life.[90,91] There are three main approaches to screening patients for POAG—measuring the IOP, assessing the optic nerve head and retinal nerve fiber layer, and evaluating the visual field. The IOP, appearance of the optic nerve, and status of visual function provide complementary clues, but to evaluate all in a single session would be impractical in population-based screening programs.[92]

Measuring IOP is not an effective method for screening populations for glaucoma. Using an IOP above 21 mmHg, the sensitivity for the diagnosis of POAG by tonometry was 47.1% and the specificity was 92.4% in one population survey.[93] Population-based studies suggest that half of all individuals with POAG have IOP levels consistently below 22 mmHg, the usual screening cutoff.[4,21] Furthermore, half of all individuals with POAG have IOP below 22 mmHg at a single screening.[20] Additionally, most individuals with elevated pressures at a screening measurement do not have, and may never develop, optic nerve damage, although risk increases with higher IOP.[20,21] Studies show that approximately 1 of every 10 to 15 individuals with elevated IOP at screening can have demonstrable optic nerve damage, and half of these (1 in 20 to 30 individuals) may not previously have been diagnosed with glaucoma.[20,21,94,95]

A second method of screening for glaucoma is to assess the optic nerve head and retinal nerve fiber layer. Current approaches to do this are technology dependent; they require examination expertise and, therefore, do not have ideal screening characteristics. While some authors report high sensitivity and specificity for subjective optic disc examination, others have found poor agreement and high interobserver variation.[96-98]

A third method of screening for glaucoma is to evaluate the visual field. Visual field testing has been used in mass screening but at unknown rates of sensitivity and specificity.[99] Perimetry based on frequency doubling technology shows promise as a screening tool to detect moderate glaucomatous damage. In a clinic-based population, frequency doubling technology correctly identified 91% of eyes with an abnormal Glaucoma Hemifield Test and 94% of glaucoma suspects with normal visual fields on Humphrey Field Analyzer (Carl Zeiss Meditec, Inc., Dublin, CA) threshold testing.[100] Test time with this technique was about 1 minute per eye. When used in a population-based glaucoma screening in Japan, the frequency doubling technology alone showed a positive predictive value ranging between 32.6% and 45.1% based on 14,814 subjects, while the negative predictive value was estimated at 98.7% based on a subset of 4141 subjects.[101]

In January 2002, the Centers for Medicare and Medicaid Services initiated coverage for glaucoma examinations by eye care professionals in the office for beneficiaries with diabetes mellitus, those with a family history of glaucoma, African Americans 50 or older, and Hispanic Americans 65 or older. While this is referred to as a screening benefit or examination, it is not applicable to examination of individuals in the community at random.

In 2005, the National Committee for Quality Assurance introduced a new quality measure for health plans that offer Medicare Advantage coverage in recognition of the importance of identifying patients with glaucoma and the difficulties of screening. The measure is based on a comprehensive eye examination conducted in the previous 2 years for older adults. The intent of the quality measure is to allow purchasers and consumers to compare the performance of managed health plans reliably.



CARE PROCESS

PATIENT OUTCOME CRITERIA

- Preservation of visual function
- Maintenance of quality of life

DIAGNOSIS

The comprehensive initial glaucoma evaluation (history and physical examination) includes all components of the comprehensive adult medical eye evaluation[102] in addition to, and with special attention to, those factors that specifically bear upon the diagnosis, course, and treatment of POAG. The examination may require more than one visit. For instance, an individual might be suspected of having glaucoma on one visit but may return for further evaluation to confirm the diagnosis, including additional IOP measurements, gonioscopy, CCT determination, visual field assessment, and optic nerve head and retinal nerve fiber layer evaluation and documentation.

Evaluation of Visual Function

Self-reported functional status or difficulty with vision can be assessed either by patient complaints or by using specific questionnaires, including the National Eye Institute - Visual Function Questionnaire-25.[90,103-109 [A:III]] Patients with glaucoma may have sufficient visual field loss to impair night driving, near vision, and outdoor mobility.[91,110-112]

Ophthalmic Evaluation

In completing the elements in the comprehensive adult medical eye evaluation,[102] the ophthalmic evaluation specifically focuses on the following elements:

- History[A:III]
- Visual acuity measurement[A:III]
- Pupil examination[B:II]
- Anterior segment examination[A:III]
- Intraocular pressure measurement[A:I]
- Gonioscopy[A:III]
- Optic nerve head and retinal nerve fiber layer examination[A:III]
- Fundus examination[A:III]

History

- Ocular,[A:III] family,[4,42,44 [A:II]] and systemic history (e.g., asthma).[A:III] The severity and outcome of glaucoma in family members, including history of visual loss from glaucoma, should be obtained during initial evaluation.[42,44 [B:III]]

- Review of pertinent records,[A:III] with particular reference to the past IOP levels, status of the optic nerve, and visual field.[A:III]
- Current ocular and systemic medications (e.g., corticosteroids) and known local or systemic intolerance to ocular or systemic medications.[A:III]
- Ocular surgery.[A:III]

  A history of LASIK or photorefractive keratectomy is associated with a falsely low IOP measurement due to thinning of the cornea.[113] Cataract surgery may also lower the IOP compared with the presurgical baseline.[114] A history of prior glaucoma laser or incisional surgical procedures should be elicited.

Visual acuity measurement

Visual acuity with current correction (the power of the present correction recorded) at distance and, when appropriate, at near should be measured. [A:III] Refraction may be indicated to obtain the best-corrected visual acuity.

Pupil examination

The pupils are examined for reactivity and an afferent pupillary defect.[115-117] [B:II]

Anterior segment examination

A slit-lamp biomicroscopic examination of the anterior segment can provide evidence of physical findings associated with narrow angles, such as shallow peripheral anterior chamber depth and crowded anterior chamber angle anatomy,[118,119] corneal pathology, or a secondary mechanism for elevated IOP such as pseudoexfoliation (exfoliation syndrome), pigment dispersion with Krukenberg spindle and/or iris transillumination defects, iris and angle neovascularization, or inflammation.[A:III]

Intraocular pressure measurement

Intraocular pressure is measured in each eye, preferably by Goldmann applanation tonometry, before gonioscopy or dilation of the pupil.[5,27,30-32,120-128] [A:I] Recording time of day of IOP measurements may be helpful to assess diurnal variation. Unrecognized fluctuations in IOP may lead to progression of POAG.[129-132] Therefore, additional measurements may be indicated, either at different hours of the day on the same day or on different days.

Gonioscopy

The diagnosis of POAG requires careful evaluation of the anterior chamber angle to exclude angle closure or secondary causes of IOP elevation, such as angle recession, pigment dispersion, peripheral anterior synechiae, angle neovascularization, and inflammatory precipitates.[133] [A:III] (See www.gonioscopy.org and Selected Reference Texts section for discussion of the techniques of gonioscopy.)

Optic nerve head and retinal nerve fiber layer examination

Examination of the optic nerve head and retinal nerve fiber layer provides valuable structural information about glaucomatous optic nerve damage.[134-136] Visible structural alterations of the optic nerve head or retinal nerve fiber layer and development of peripapillary choroidal atrophy frequently occur before visual field defects can be detected.[135,137-144] Careful study of the optic disc neural rim for small hemorrhages is important, since these hemorrhages often precede visual field loss and further optic nerve damage in patients with glaucoma.[27,28,30,36,145-149] In the Ocular Hypertension Treatment Study, the incidence of POAG in eyes with disc hemorrhage was 13.6% compared with 5.2% in eyes without disc hemorrhage over 8 years.[146] In the Early Manifest Glaucoma Trial, 13% of patients had disc hemorrhages at baseline examination, and hemorrhages were associated with progression.[30]

The preferred technique for optic nerve head and retinal nerve fiber layer evaluation involves magnified stereoscopic visualization (as with the slit-lamp biomicroscope), preferably through a

dilated pupil.[A:III] In some cases, direct ophthalmoscopy complements magnified stereoscopic visualization, providing additional information of optic nerve detail due to the greater magnification of the direct ophthalmoscope. Red-free illumination of the posterior pole by stereo-biomicroscopy with an indirect lens at the slit lamp, the direct ophthalmoscope, or with digital red-free photography may aid in evaluating the retinal nerve fiber layer.[150]

Fundus examination

Examination of the fundus, through a dilated pupil whenever feasible, includes a search for other abnormalities that may account for optic nerve changes and/or visual field defects (e.g., optic nerve pallor, disc drusen, optic nerve pits, disc edema from central nervous system disease, macular degeneration, retinovascular occlusion, and other retinal disease).[A:III]

Supplemental Ophthalmic Testing

Supplemental ophthalmic testing includes the following components:

- Central corneal thickness measurement[A:II]
- Visual field evaluation[A:III]
- Optic nerve head and retinal nerve fiber layer analysis[A:II]

Central corneal thickness measurement

Measurement of CCT aids the interpretation of IOP readings and helps to stratify patient risk for ocular damage.[29,53,57,151 [A:II]] An overestimation of the real IOP may occur in eyes with corneas that are thicker than average, while an underestimation of the real IOP tends to occur in eyes with corneas that are thinner than average. Several studies have sought to quantify the relationship between measured IOP level and CCT, but there is no generally accepted correction formula. There is a controversy over whether CCT represents a risk factor for glaucoma due to its effect on IOP measurement or whether CCT is a risk factor itself, unrelated to IOP.[50,152,153] While it is clear that thinner CCT is a risk factor for the development of POAG, studies of progression have had variable findings. Some studies have found an association while others have not (see Table 3).

Visual field evaluation

Automated static threshold perimetry is the preferred technique for evaluating the visual field.[154 [A:III]] The frequency doubling technology (FDT) method with the central 20-degree test program (C-20) and short-wavelength automated perimetry (SWAP) with the central 24-degree test program (24-2) are two of several alternative testing methods to screen for a defect before conducting more definitive threshold testing.[154] Visual field testing based on SWAP[155] and FDT[156] may detect defects or progression of defects earlier than conventional white-on-white perimetry in some patients.[157] Careful manual combined kinetic and static threshold testing (e.g., Goldmann visual fields) is an acceptable alternative when patients cannot perform automated perimetry reliably or if it is not available.[A:III] Repeat, confirmatory visual field examinations may be required for test results that are unreliable or show a new glaucomatous defect before changing management.[27,124 [A:III]] It is best to use a consistent examination strategy for visual field testing.

TABLE 3 SUMMARY OF RESULTS FOR CENTRAL CORNEAL THICKNESS AS A RISK FACTOR FOR PROGRESSION OF GLAUCOMA

Study	No. of Patients	Level of Evidence	Risk	Comments
Early Manifest Glaucoma Trial[1]	255	I	+	Thin CCT is a risk factor for progression of glaucoma (in those with baseline IOP ≥21 mmHg)
Kim[2]	88	II	+	Thin CCT is associated with visual field progression in glaucoma
Chauhan[3]	54	II	-	CCT did not predict visual field or optic disc progression
Jonas[4]	454	II	-	CCT is not associated with progression of visual field damage
Jonas[5]	390	II	-	CCT is not associated with optic disc hemorrhages
Congdon[6]	230	II	-	CCT is not associated with glaucoma progression (though corneal hysteresis was)
Stewart[7]	310	III	+/-	CCT is associated with progression on univariate analysis but is not associated on multivariate analysis

CCT = central corneal thickness

SOURCE: Adapted with permission from Dueker D, Singh K, Lin SC, et al. Corneal thickness measurement in the management of primary open-angle glaucoma: a report by the American Academy of Ophthalmology. Ophthalmology 2007;114:1784.

1. Leske MC, Heijl A, Hyman L, et al, Early Manifest Glaucoma Trial Group. Predictors of long-term progression in the Early Manifest Glaucoma Trial. Ophthalmology 2007;114:1965-72.
2. Kim JW, Chen PP. Central corneal pachymetry and visual field progression in patients with open-angle glaucoma. Ophthalmology 2004;111:2126-32.
3. Chauhan BC, Hutchison DM, LeBlanc RP, et al. Central corneal thickness and progression of the visual field and optic disc in glaucoma. Br J Ophthalmol 2005;89:1008-12.
4. Jonas JB, Stroux A, Velten I, et al. Central corneal thickness correlated with glaucoma damage and rate of progression. Invest Ophthalmol Vis Sci 2005;46:1269-74.
5. Jonas JB, Stroux A, Oberacher-Velten IM, et al. Central corneal thickness and development of glaucomatous optic disk hemorrhages. Am J Ophthalmol 2005;140:1139-41.
6. Congdon NG, Broman AT, Bandeen-Roche K, et al. Central corneal thickness and corneal hysteresis associated with glaucoma damage. Am J Ophthalmol 2006;141:868-75.
7. Stewart WC, Day DG, Jenkins JN, et al. Mean intraocular pressure and progression based on corneal thickness in primary open-angle glaucoma. J Ocul Pharmacol Ther 2006;22:26-33.

Optic nerve head and retinal nerve fiber layer analysis

The appearance of the optic nerve should be documented.[136,158 [A:II]] Color stereophotography is an accepted method for documenting optic nerve head appearance. Computer-based image analysis of the optic nerve head and retinal nerve fiber layer is an alternative for documentation of the optic nerve. As improvements in these instruments continue, the capacity for them to help the clinician diagnose glaucoma and identify progressive nerve damage becomes more reliable.[159-161] Stereoscopic disc photographs and computerized images of the nerve are distinctly different methods for optic nerve documentation and analysis.[162] Each is complementary with regard to the information they provide the clinician who must manage the patient. In the absence of these technologies, a nonstereoscopic photograph or a drawing of the optic nerve head should be recorded, but these are less desirable alternatives to stereophotography or computer-based imaging.[163 [A:III]] In patients with advanced glaucomatous optic neuropathy, there is limited benefit of using stereophotography to identify progressive optic nerve change.[164,165]

There are three types of computer-based imaging devices currently available for glaucoma: confocal scanning laser ophthalmoscopy, optical coherence tomography, and scanning laser polarimetry. In a systematic review, the versions of these devices that were studied were similar in their ability to distinguish glaucoma from controls.[136,166] When examined for the ability of these devices to detect glaucoma progression, studies have shown a relative lack of concordance between the structural (the imaging devices) and functional (visual field) tests.[167,168] Taken together, computer-based imaging devices for glaucoma provide useful, quantitative information for the clinician when analyzed in

conjunction with other relevant clinical parameters. As device technology evolves (e.g., higher resolution spectral domain optical coherence tomography), the diagnostic performance is expected to improve accordingly.

MANAGEMENT

Goals

The goals of managing patients with POAG are to achieve the following:

- Controlled IOP in the target range
- Stable optic nerve/retinal nerve fiber layer status
- Stable visual fields

Because elevated IOP is a treatable cause of POAG damage, one can expect to reduce the risk of disease progression in many patients by lowering the IOP by means of medication, laser therapy, or incisional glaucoma surgery. Results from randomized controlled trials (summarized in Table 2) and other studies reinforce this expectation and provide evidence that the more the IOP is lowered, the more likely is it to slow the rate of progression of POAG.[5,27-32,36,120,122,123,126,169-180]

Management is a challenge for the patient and the doctor, because POAG is a chronic, often asymptomatic, condition that may require frequent use of multiple and expensive medications[181] that may cause side effects or may require laser or incisional surgery. The effects of treatment, the patient's quality of life, and the patient's life expectancy are important to consider when choosing therapy. The diagnosis, severity of the disease, prognosis and management plan, and likelihood of long-term therapy should be discussed with the patient.[A:III] Substantial field loss in glaucoma is associated with a decrease in quality of life measures.[90,91,182]

Target Intraocular Pressure for Patients with POAG

The goal of glaucoma treatment is to maintain the IOP in a range at which a patient is likely to remain stable or at which worsening of glaucoma will be slow enough that the risk of additional intervention is not justified.[183,184] The estimated upper limit of this range is considered the "target pressure." The initial target pressure is an estimate and a means toward the ultimate goal of protecting the patient's vision. The target pressure should be individualized and may need adjustment during the course of the disease.[A:III]

When initiating therapy, the ophthalmologist assumes that the measured pretreatment pressure range contributed to optic nerve damage and is likely to cause additional damage in the future. Lowering the pretreatment IOP by 25% or more has been shown to inhibit progression of POAG.[27,30-32,123,125 [A:II]] It is reasonable to select an initial target pressure at least 25% lower than pretreatment levels. Choosing an even lower target IOP can be justified if there is more severe optic nerve damage, if the damage is progressing rapidly, or if other risk factors such as family history, age, or disc hemorrhages are present (see Risk Factors for Progression). Choosing a less aggressive target IOP may be reasonable if the risks of aggressive treatment outweigh the benefits (e.g., if a patient does not tolerate medical therapy well and surgical intervention would be difficult or if the patient's life expectancy is short). In a study of newly diagnosed patients with moderate to advanced glaucoma, a subset of patients randomized to initial lowering of IOP by surgery did better than those assigned to medical treatment over an 8-year period, and this could be due to the lower mean posttreatment IOP in the surgery group.[185]

The adequacy and validity of the target pressure are periodically reassessed by comparing optic nerve status (by optic disc appearance, quantitative assessments of the disc and nerve fiber layer, and visual field tests) with previous examinations. If progression occurs at the target pressure, undetected IOP fluctuations and adherence to therapy can be re-evaluated before adjusting the target IOP. However, target pressure is an estimate, and all treatment decisions must be individualized according to the needs of the patient.

Therapeutic Choices

The IOP can be lowered by medical treatment, laser therapy, or incisional glaucoma surgery (alone or in combination). The choice of initial therapy depends on numerous considerations, and discussion of treatment with the patient should include the relative risks and benefits of the three options.[A:III]

Medical treatment

Unless contraindicated, medical therapy is presently the most common initial intervention to lower IOP. There are many drugs available for initial therapy, and medication choice may be influenced by potential cost, side effects, and dosing schedules (see Table 4 for an overview of options available). Patient adherence to therapy is enhanced by using eyedrops with the fewest side effects as infrequently as necessary to achieve the target IOP. If target IOP is not achieved by one medication, then additional separate medications, combination therapies, or switching of treatments may be considered to reach the target IOP.

Prostaglandin analogs and beta-blockers are the most frequently used initial eye drops for lowering IOP in patients with glaucoma.[186,187] Prostaglandin analogs are the most effective drugs at lowering IOP and can be considered as initial medical therapy unless other considerations such as cost, side effects, intolerance, or patient refusal preclude this.[188,189 [A:I]] Other agents in addition to prostaglandin analogs and beta-blockers include alpha$_2$ adrenergic agonists, parasympathomimetics, and topical and oral carbonic anhydrase inhibitors.[190,191]

The ophthalmologist should consider the balance between side effects and effectiveness in choosing a regimen of maximal effectiveness and tolerance to achieve the desired IOP reduction for each patient.[192-195 [A:III]] Frequent dosing and side effects (such as depression, exercise intolerance, and impotence with topical beta-blockers) may affect adherence to therapy.[193,196]

To determine the effectiveness of topical therapy, it is necessary to distinguish between the therapeutic impact of an agent on IOP and ordinary background fluctuations of IOP. It may be useful to begin by treating only one eye and comparing the relative change of the IOP in the two eyes at follow-up visits.[131] However, because the two eyes of an individual may not respond equally to the same medication, and because of the possibility of asymmetric spontaneous fluctuations and the potential for contralateral effect of monocular topical medications,[197] it is acceptable to compare the effect in one eye relative to multiple baseline measurements.[198] Additional studies are needed to compare directly monocular and binocular drug trials to find out whether a monocular trial is better at determining a nonresponder than a binocular trial. If a drug fails to reduce IOP sufficiently despite good adherence to therapy, it can be replaced with an alternate agent until effective medical treatment is established. If a single medication is effective in lowering IOP but the target pressure is not reached, combination therapy[199] or switching to an alternative therapy may be appropriate.

The patient and ophthalmologist together decide on a practical and feasible regimen to follow in terms of dosing, cost, and adherence in the context of the patient's age and preferences.[158] The ophthalmologist should assess the patient who is being treated with glaucoma medication for local ocular and systemic side effects; toxicity, including interactions with other medications; and potential life-threatening adverse reactions.[200 [A:III]] To reduce systemic absorption, patients should be educated about eyelid closure or nasolacrimal duct occlusion when applying topical medications (see Related Academy Materials section for public information brochures).[201]

TABLE 4 GLAUCOMA MEDICATIONS

Drug Classification	Methods of Action	IOP Reduction*	Side Effects	Contraindications
Prostaglandin analogs	Increase uveoscleral and/or trabecular outflow	25%–33%	- Cystoid macular edema - Conjunctival injection - Increased eyelash growth - Periocular hyperpigmentation - Iris color change - Uveitis - Possible herpes virus activation	- Macular edema - History of herpetic keratitis
Beta-adrenergic antagonists (beta-blockers)	Decrease aqueous production	20%–25%	- Corneal toxicity - Allergic reactions - CHF (classic teaching, although cardiologists use beta-blockers as first line treatment in CHF) - Bronchospasm (seen with nonselective) - Bradycardia - Depression - Impotence	- Chronic obstructive pulmonary disease (nonselective) - Asthma (nonselective) - CHF (check with cardiologist) - Bradycardia - Hypotension - Greater than first degree heart block
Alpha-adrenergic agonists	Nonselective: improve aqueous outflow Selective: decrease aqueous production; decrease episcleral venous pressure or increase uveoscleral outflow	20%–25%	- Conjunctival injection - Allergic reactions - Fatigue - Somnolence - Headache	- Monoamine oxidase inhibitor therapy - Infants and children younger than 2 years
Parasympathomimetic agents	Increase trabecular outflow	20%–25%	- Increased myopia - Eye or brow ache/pain - Decreased vision - Cataract - Periocular contact dermatitis - Corneal toxicity - Paradoxical angle closure	- Neovascular, uveitic, or malignant glaucoma - Need to regularly assess fundus
Carbonic anhydrase inhibitors (mainly with systemic use)	Decrease aqueous production	15%–20%	With topical route: - Metallic taste - Allergic dermatitis/conjunctivitis - Corneal edema With oral route: - Stevens-Johnson syndrome - Malaise, anorexia, depression - Serum electrolyte imbalance - Renal calculi - Blood dyscrasias (aplastic anemia, thrombocytopenia) - Metallic taste	- Sulfonamide allergy - Kidney stones - Aplastic anemia - Thrombocytopenia - Sickle cell disease

CHF = congestive heart failure; IOP = intraocular pressure

* Data from the European Glaucoma Society. Terminology and Guidelines for Glaucoma. 3rd ed. Savona, Italy:Editrice Dogma S.r.l.; 2008:127. Available at: www.eugs.org/eng/EGS_guidelines.asp. Accessed May 28, 2010.

SOURCE: Adapted with permission from the American Academy of Ophthalmology Practicing Ophthalmologists Curriculum (POC) Panel Chairs and Vice Chairs. MOC Exam Study Kit. Core ophthalmic knowledge. Core topics for glaucoma: Medical management of glaucoma. Available to Academy members only at: http://one.aao.org/CE/MOC/MOCStudyResources.aspx. Accessed May 28, 2010.

Adequate treatment of glaucoma requires a high level of adherence to therapy. Frequently this is not achieved; studies indicate relatively poor adherence to therapy.[192,202-204] Even with instruction, free medication, once-daily administration, use of a dosing aid, and electronic monitoring of adherence, nearly 45% of patients in one study took fewer than 75% of their prescribed doses.[204] Instilling eyedrops correctly is difficult for patients, and their ability to do so may worsen as glaucoma progresses.[205,206] Repeated instruction and counseling in proper techniques for using medication as

well as a clearly written medication regimen and follow-up telephone calls may improve adherence to therapy.[204,207,208] At each examination, medication dosage and frequency of use should be recorded.[A:III] Reviewing the time medication was taken may be useful. Adherence to the therapeutic regimen and recommendations for therapeutic alternatives or diagnostic procedures should be discussed.[A:III] Cost may be a factor in adherence, especially when multiple medications are used.[208] Patient education and informed participation in treatment decisions may improve adherence[208] and overall effectiveness of glaucoma management.

Laser trabeculoplasty

Laser trabeculoplasty can be considered as initial therapy in selected patients[122,209 [A:I]] or an alternative for patients who cannot or will not use medications reliably due to cost, memory problems, difficulty with instillation, or intolerance to the medication. Laser trabeculoplasty lowers IOP by improving aqueous outflow and can be performed using argon, diode, and frequency-doubled YAG lasers.

Argon and diode laser trabeculoplasty

Studies using continuous-wave argon laser with a wavelength spectrum that peaks at 488 nm (argon laser trabeculoplasty [ALT]) found that treatment increases aqueous outflow and provides a clinically significant reduction of IOP in more than 75% of initial treatments of previously unoperated eyes (see Table 5).[122,123] Since these initial studies, more compact solid-state diode lasers have mostly replaced the original argon laser with equal IOP lowering efficacy.

TABLE 5 **RANDOMIZED CLINICAL TRIALS OF LASER TRABECULOPLASTY WITH PUBLISHED RESULTS**

Name	Study Design	No. of Patients	Follow-up Duration (years)	Finding
Glaucoma Laser Trial (GLT)[1,2]	Newly diagnosed POAG: medical therapy vs laser trabeculoplasty	271	2.5–5.5	Initial laser trabeculoplasty lowered IOP more (-9 mmHg) than initial treatment with topical timolol maleate (-7 mmHg) over 2 years; initial laser trabeculoplasty was at least as effective in preserving visual field and optic disc status over 5.5 years.
Glaucoma Laser Trial Follow-up Study[2]	Participants in the GLT	203	6–9	Longer follow-up reinforced the earlier findings that initial laser trabeculoplasty lowered IOP more (-1.2 mmHg) than initial treatment with topical timolol maleate and was at least as effective in preserving visual field and optic disc status.
Moorfields Primary Treatment Trial[3]	Newly diagnosed POAG: medical therapy vs. laser trabeculoplasty vs. trabeculectomy	168	5+	Trabeculectomy lowered IOP the most (-60%); laser trabeculoplasty (-38%) and medical therapy groups (-49%) had more deterioration in visual fields than trabeculectomy group.
Early Manifest Glaucoma Trial[4,5,6]	Newly diagnosed POAG: medical therapy and laser trabeculoplasty vs. no treatment	255	4–10	Lowering IOP with medical therapy and trabeculoplasty (-25%) slowed progression of optic disc and visual field damage.
Advanced Glaucoma Intervention Study (AGIS)[7,8]	POAG after medical therapy failure with no previous surgery: laser trabeculoplasty vs. trabeculectomy	591	10–13	Surgical outcome varied by race; patients with African ancestry did better with trabeculoplasty as first surgery (-30% IOP), while in the longer term (4+ years) Caucasian American patients did better with trabeculectomy as first surgery (-48% IOP). Lowest IOP group during follow-up after surgical interventions (-47%) protected against further visual field deterioration in advanced glaucoma patients.

IOP = intraocular pressure; POAG = primary open-angle glaucoma

1. Glaucoma Laser Trial Research Group. The Glaucoma Laser Trial (GLT). 2. Results of argon laser trabeculoplasty versus topical medicines. Ophthalmology 1990;97:1403-13.
2. Glaucoma Laser Trial Research Group. The Glaucoma Laser Trial (GLT) and Glaucoma Laser Trial Follow-up Study: 7. Results. Am J Ophthalmol 1995;120:718-31.
3. Migdal C, Gregory W, Hitchings R. Long term functional outcome after early surgery compared with laser and medicine in open-angle glaucoma. Ophthalmology 1994;101:1651-7.
4. Heijl A, Leske MC, Bengtsson B, et al, Early Manifest Glaucoma Trial Group. Reduction of intraocular pressure and glaucoma progression: results from the Early Manifest Glaucoma Trial. Arch Ophthalmol 2002;120:1268-79.
5. Leske MC, Heijl A, Hussein M, et al, Early Manifest Glaucoma Trial Group. Factors for glaucoma progression and the effect of treatment: the Early Manifest Glaucoma Trial. Arch Ophthalmol 2003;121:48-56.
6. Leske MC, Heijl A, Hyman L, et al, Early Manifest Glaucoma Trial Group. Predictors of long-term progression in the Early Manifest Glaucoma Trial. Ophthalmology 2007;114:1965-72.
7. AGIS Investigators. The Advanced Glaucoma Intervention Study (AGIS): 7. The relationship between control of intraocular pressure and visual field deterioration. Am J Ophthalmol 2000;130:429-40.
8. AGIS Investigators. The Advanced Glaucoma Intervention Study (AGIS): 13. Comparison of treatment outcomes within race: 10-year results. Ophthalmology 2004;111:651-64.

For patients initially treated with ALT, the amount of medical treatment required for glaucoma control is reduced.[121,122] Results from long-term studies of patients receiving maximum medical therapy who subsequently had laser and incisional surgery indicate that 30% to more than 50% of eyes require additional surgical treatment within 5 years after ALT.[123,210-213] For eyes that have failed to maintain a previously adequate response, repeat ALT has a low long-term rate of success, with failure occurring in nearly 90% of these eyes by 2 years.[214-218] After previous applications to the full circumference of the anterior chamber angle, repeat ALT has a lower success rate than initial therapy[216,217] in eyes that have not had a reduction in IOP for at least a year following the first laser surgery.[217] Compared with initial laser trabeculoplasty, there is an increased risk of problems and complications such as IOP spikes after repeat laser trabeculoplasty.[214,215,218,219]

Selective laser trabeculoplasty

The introduction of selective laser trabeculoplasty (SLT) is most likely responsible for the increase in use of laser trabeculoplasty in 2001 after a previous decline.[220] Selective laser trabeculoplasty uses a Q-switched, frequency doubled, 532 nm Nd:YAG laser that delivers less energy and is purported to be selectively absorbed by pigmented cells in the trabecular meshwork.[221] These attributes appear purportedly to produce less thermal damage to the trabecular meshwork compared with ALT.[222] However, several prospective and retrospective studies indicate that SLT appears comparable to but not better than ALT in lowering IOP.[223-230] Selective laser trabeculoplasty also appears to be comparable in efficacy to medical therapy with prostaglandin analogs,[209,231] although in one prospective study, SLT was only comparable to latanoprost when 360 degrees of the trabecular meshwork was treated.[231] In this study, latanoprost had a better IOP-lowering effect compared with 90 and 180 degrees of treatment.

It has been claimed that SLT has greater success than ALT with repeated treatments, but no controlled, randomized clinical trial has shown that this is true. Only one study with a significant sample size has studied the results of SLT repeated after prior SLT treatment. This was a retrospective study of repeat 360 degree SLT after prior successful 360-degree SLT.[232] In the postoperative period, repeat SLT treatment lowered IOP, but the effect was less than that with initial SLT treatment at 1 to 3 months and 5 to 8 months. These results suggest that repeat SLT may be less efficacious compared with primary treatment, but further studies are needed to clarify this issue. The safety profile of SLT appears to be good, with mild anterior chamber inflammation after treatment and less ocular discomfort compared with ALT.[226] Intraocular pressure spikes have been noted after SLT in 4.5% to 27% of eyes in various studies.[224,231,233]

Perioperative care in laser trabeculoplasty

The ophthalmologist who performs the laser surgery has the following responsibilities:[234,235] [A:III]

- To obtain informed consent from the patient or the patient's surrogate decision maker after discussing the risks, benefits, and expected outcomes of surgery[A:III]
- To ensure that the preoperative evaluation confirms the need for surgery[A:III]

- To perform at least one IOP check within 30 minutes to 2 hours of surgery[236] [A:I]
- To perform a follow-up examination within 6 weeks of surgery or sooner if there is concern about IOP-related damage to the optic nerve during this time[210,237-239] [A:III]

Medications that are not being used chronically may be used perioperatively to avert temporary IOP elevations, particularly in those patients with severe disease.[236,240,241]

Incisional glaucoma surgery

Trabeculectomy

Filtering surgery is effective in lowering IOP; it is generally indicated when medicine or laser therapy is insufficient to control disease and can be considered in selected cases as initial therapy.[185,242] [A:I]

Filtering surgery provides an alternative path for the escape of aqueous humor, and it often reduces IOP and the need for medical treatment. Estimates of success rates over time range from 31% to 56% in different populations.[243-245] The failure rate of filtering surgery, without the use of adjunctive antifibrotic medications, alone or combined with medical therapy in a previously unoperated eye in the Advanced Glaucoma Intervention Study[123] reached approximately 30% in African American patients and 20% in Caucasian American patients over a 10-year period.[123] While long-term control is often achieved, many patients may require further therapy or a reoperation, which carries a higher failure rate.[123,246-249] Furthermore, filtering surgery increases the likelihood that phakic eyes may undergo cataract surgery.[125,250,251] In eyes that have undergone previous cataract surgery involving the conjunctiva, the success rate of initial glaucoma surgery is reduced.[170]

Antifibrotic agents may be used intraoperatively and postoperatively to reduce the subconjunctival scarring after filtration surgery that can result in failure of the operation. The use of intraoperative mitomycin-C reduces the risk of surgical failure both in eyes at high risk of surgical failure[252,253] and in eyes that have not undergone previous surgery.[253-256] Some studies have demonstrated a benefit of intraoperative 5-fluorouracil[257,258] and others have not.[259] The use of postoperative injections of 5-fluorouracil also reduces the likelihood of surgical failure in both high-risk eyes[170,260-262] and eyes that have not undergone previous surgery.[262-264]

The use of an antifibrotic agent carries with it an increased likelihood of bleb-related complications such as hypotony,[265-267] hypotony maculopathy,[265] late-onset bleb leak,[262,268] and late-onset infection[269,270] that must be weighed against the benefits when deciding whether or not to use these agents. These complications may be even more common in primary filtering surgery of phakic patients.[271-273]

Aqueous shunts

All aqueous shunts (also known as tube shunts, glaucoma drainage devices, and setons) consist of a tube that diverts aqueous humor to an end plate located in the equatorial region of the eye. The primary resistance to flow through these devices occurs across the fibrous capsule that develops around the end plate. Aqueous shunts differ in their design with respect to the size, shape, and material from which the end plate is made. They may be further subdivided into valved and nonvalved shunts, depending on whether or not a valve mechanism is present to limit flow through the shunt if the IOP becomes too low. Examples of nonvalved implants are the Baerveldt glaucoma implant (Abbott Medical Optics, Santa Ana, CA) and the Molteno implant (Molteno Ophthalmic Ltd., Dunedin, New Zealand). Examples of the valved implants are the Ahmed glaucoma valve (New World Medical, Inc., Rancho Cucamonga, CA) and the Krupin implant (Eagle Vision, Inc., Memphis, TN).

Aqueous shunts have traditionally been used to manage medically uncontrolled glaucoma when trabeculectomy has failed to control IOP or is deemed unlikely to succeed. This includes eyes with neovascular glaucoma, uveitic glaucoma, extensive conjunctival scarring from previous ocular surgery or cicatrizing diseases of the conjunctiva, and congenital glaucoma in which angle surgery has failed. However, the indications for using aqueous shunts have been broadening, and these devices are being increasingly utilized in the surgical management of glaucoma. Medicare data show a steady rise in the number of shunts placed from 1995 to 2004, while there has been a

concurrent decline in the number of trabeculectomies performed.[274] Recent surveys of the American Glaucoma Society membership have also demonstrated a progressive increase in the number of surgeons using tube shunts.[275,276]

A systematic review concluded that aqueous shunts are comparable with trabeculectomy for IOP control and duration of benefit, that larger end-plate surface area provides better IOP control, and that there appears to be no advantage to the use of antifibrotic agents or systemic corticosteroids as adjuncts to aqueous shunt procedures.[277,278] The need for comparative studies and long-term follow-up was identified by the authors.

Several studies have compared aqueous shunts with trabeculectomy. A retrospective study evaluating surgical results in matched patient groups reported similar IOP reduction with the single-plate Molteno implant and trabeculectomy with 5-fluorouracil.[279] However, another retrospective case-control study observed a higher 5-year success rate after trabeculectomy with mitomycin-C than with Ahmed glaucoma valve implantation.[280] A randomized clinical trial in Sri Lanka comparing the Ahmed implant and trabeculectomy in patients with primary open-angle and angle-closure glaucoma found comparable IOP reduction and success rates with a mean follow-up of 31 months.[281] The Tube Versus Trabeculectomy (TVT) Study is a multicenter, prospective, randomized clinical trial that compared the safety and efficacy of tube-shunt surgery using the 350-mm^2 Baerveldt glaucoma implant and trabeculectomy with mitomycin-C in patients with previous cataract extraction and/or failed trabeculectomy. Tube-shunt surgery had a higher success rate than trabeculectomy (85% vs. 69%) after 3 years of follow-up as defined by loss of vision, and/or IOP less than or equal to 5 mmHg or greater than or equal to 21 mmHg.[282] Both surgical procedures were associated with similar IOP reduction and use of supplemental medical therapy at 3 years. Postoperative complications occurred more frequently after trabeculectomy compared with tube-shunt surgery, but the rate of serious complications associated with vision loss and/or reoperation to manage the complication was similar with both procedures.

Aqueous shunts are associated with intraoperative and postoperative complications that are similar to those that occur with trabeculectomy. In addition, they have unique complications related to implantation of a foreign body. Erosion of the tube may occur through the conjunctiva, and this typically develops a few millimeters behind the limbus following anterior chamber insertion. Diplopia may result from extraocular muscle fibrosis or a mass effect of the bleb overlying the end plate. Tube-cornea touch can lead to progressive endothelial cell loss and persistent corneal edema. The risk of postoperative infection appears to be less with aqueous shunts than after trabeculectomy with an antifibrotic agent.

Combined surgeries

Patients with POAG who have a visually significant cataract have a range of options to consider. If IOP control is at target on one or few medications, cataract surgery alone may be adequate, with the additional benefit that it may lower IOP slightly. If IOP is markedly uncontrolled on several medications after laser trabeculoplasty and the patient has a moderate cataract, then glaucoma surgery may be indicated initially, with the plan to perform cataract surgery once IOP is adequately controlled. In between these two extremes, the decision of which procedure(s) to perform first or whether to combine cataract and glaucoma surgery is determined by the ophthalmologist and patient after discussion of the risks and benefits of each course of action.

Cataract surgery with IOL implantation alone results in a modest reduction in IOP of less than 2 mmHg on average.[114] Generally, combined cataract and glaucoma surgery is not as effective as glaucoma surgery alone in lowering IOP,[114,283] so patients who require filtration surgery who also have mild cataract may be better served by filtration surgery alone and cataract surgery later.[B:III] The use of mitomycin-C, but not 5-flurouracil, results in lower IOP in combined procedures.[114,253,283] A systematic review published in 2002 found moderate quality evidence that separating the cataract and glaucoma incisions results in lower IOP than a one-site combined procedure, but the differences in outcomes were small.[283] Subsequent publications have found no difference between the two approaches.[284-286]

Potential benefits of a combined procedure (cataract extraction with IOL implantation and trabeculectomy) are protection against the IOP rise that may complicate cataract surgery alone and

the possibility of achieving long-term glaucoma control with a single operation. Therefore, an ophthalmologist may reasonably choose to perform a combined surgery due to these perceived advantages to an individual patient. Despite these presumed advantages, the evidence to date does not support routinely combining cataract and glaucoma surgery for all patients, because IOP outcomes with two-stage surgery are likely similar.[114] Additionally, combined procedures are technically more complex. Ultimately, however, the decision of which surgical route to pursue is best left to the treating ophthalmologist and the individual patient.

Other types of glaucoma surgery can also be combined with cataract surgery, such as implantation of glaucoma drainage devices and endocyclophotocoagulation (see discussion in next section). Combined cataract and glaucoma drainage device surgery can also improve vision while providing IOP control.[287-289]

Other glaucoma surgeries

Nonpenetrating glaucoma surgery is an alternative to trabeculectomy. The precise role of nonpenetrating surgery in the surgical management of glaucoma remains to be determined. The two main types of nonpenetrating glaucoma surgery are viscocanalostomy and nonpenetrating deep sclerectomy. The rationale for nonpenetrating glaucoma surgery is that by avoiding a continuous passageway from the anterior chamber to the subconjunctival space, the incidence of complications such as bleb-related problems and hypotony can be reduced. The nonpenetrating procedures have a higher degree of surgical difficulty compared with trabeculectomy and require special instrumentation. Randomized clinical trials comparing viscocanalostomy with trabeculectomy generally suggest greater IOP reduction with trabeculectomy, but more complications with viscocanalostomy.[290-298]

One randomized clinical trial found that trabeculectomy was more effective than nonpenetrating deep sclerectomy at lowering IOP,[299] and several others found that the two surgeries were equally effective.[300-303]

Other glaucoma surgical procedures currently under evaluation[304] are canaloplasty with a tensioning suture[305] (Prolene [Ethicon Inc., Somerville, NJ]), ab interno trabeculotomy using the Trabectome (NeoMedix, Tustin, CA),[306,307] trabecular meshwork bypass stent,[308] and the Ex-PRESS mini glaucoma shunt (Alcon Laboratories, Inc., Ft. Worth, TX).

Perioperative care in incisional glaucoma surgery

The ophthalmologist who performs incisional glaucoma surgery has the following responsibilities:[234,235] [A:III]

- Obtain informed consent from the patient or the patient's surrogate decision maker after discussing the risks, benefits, and expected outcomes of surgery[309] [A:III]
- Ensure that the preoperative evaluation accurately documents the findings and indications for surgery[A:III]
- Prescribe topical corticosteroids in the postoperative period[310,311] [A:II]
- Perform a follow-up evaluation on the first postoperative day (12 to 36 hours after surgery) and at least once during the first 1 to 2 weeks to evaluate visual acuity, IOP, and status of the anterior segment[312-317] [A:II]
- In the absence of complications, perform additional postoperative visits during a 6-week period to evaluate visual acuity, IOP, and status of the anterior segment[312-317] [A:III]
- Schedule more frequent follow-up visits, as necessary, for patients with postoperative complications such as a flat or shallow anterior chamber or evidence of early bleb failure, increased inflammation, or Tenon's encapsulated bleb formation[312-317] [A:III]
- Undertake additional treatments as necessary, including injection of antifibrotic agents, repair of bleb leaks, bleb massage, suture lysis, bleb needling, or other surgical revisions of the bleb or surgical procedures to correct a flat anterior chamber to maximize the chances for a successful long-term result[318-320] [A:III]

- Explain that filtration surgery places the eye at risk for endophthalmitis for the duration of the patient's life, and that if the patient has symptoms of pain and decreased vision and the signs of redness and discharge, he or she should notify the ophthalmologist immediately[321] [A:III]

Cyclodestructive surgery

Cyclodestructive procedures reduce the rate of aqueous production. There are several ways to reduce ciliary body function, such as cyclocryotherapy, transscleral and noncontact Nd:YAG laser, and transscleral and noncontact endodiode laser cyclophotocoagulation.[322,323] Cyclodestructive procedures have traditionally been used for refractory glaucomas, and success rates have been reported in the range of 34% to 94%.[323] They have been associated with a subsequent decrease of visual acuity[324,325] and, rarely, cases of sympathetic ophthalmia.[326,327] Disadvantages of cyclodestructive procedures include postoperative inflammation, IOP spike, and the frequent need for repeat treatment weeks or months later.[328] Compared with cyclocryotherapy, laser cyclophotocoagulation causes less postoperative pain and inflammation. Therefore, cyclocryotherapy is now rarely used. Laser cyclodestructive procedures have advantages over filtration surgery that include technical ease and reduced postoperative care.

In 2005, 47% of all Medicare cyclophotocoagulation procedures were performed endoscopically; in 2006, 58%; and in 2007, 65%.[329] Endoscopic cyclophotocoagulation (ECP) consists of a solid-state 810-nm laser, a video camera, aiming beam, and a xenon light source housed together in a fiberoptic cable[323] that can be introduced inside the eye for direct visualization and treatment of the ciliary processes. Theoretically, this allows better titration of laser treatment. The efficacy of ECP appears to be good, with IOP reduction reported in the range of 34% to 57%.[330-332] It appears that treating 270 to 360 degrees of the ciliary body is necessary to achieve significant IOP lowering.[330,332] Fibrin exudates, hyphema, cystoid macular edema, vision loss, hypotony, choroidal detachment,[330] and phthisis[333] have been noted after ECP in eyes with advanced glaucoma, but more recent studies involving eyes with less advanced glaucomatous damage seem to report fewer of these complications.[331]

Endoscopic cyclophotocoagulation[330,331,334] may be combined with cataract surgery or tube-shunt surgery. One randomized trial comparing cataract surgery combined with either ECP or trabeculectomy suggested that IOP lowering efficacy is similar for both,[335] and another study comparing ECP with the Ahmed drainage implant also showed comparable efficacy in IOP lowering, although the rate of complication with the latter surgery was higher.[336]

Other therapeutic considerations

Among patients there is a growing interest in complementary and alternative medicinal approaches to the treatment of glaucoma. There is a lack of scientific evidence that herbal medicines or nutritional supplements are beneficial in treating glaucoma.[337,338] One study based on patient questionnaires found an association between higher intake of certain fruits and vegetables (green collards, kale, and carrots) and reduced risk of glaucoma.[339] The American Academy of Ophthalmology Complementary Therapy Task Force found no scientific evidence of increased benefit or diminished risk with the use of marijuana to treat glaucoma compared with conventional medications.[340]

Follow-up Evaluation

Guidelines for follow-up of patients with POAG are summarized in Table 6. These recommendations apply to ongoing glaucoma management and not to visits for other purposes. Follow-up evaluation includes examination as well as optic nerve head and visual field assessment as indicated.

TABLE 6 RECOMMENDED GUIDELINES FOR FOLLOW-UP GLAUCOMA STATUS EVALUATIONS WITH OPTIC NERVE AND VISUAL FIELD ASSESSMENT[B:III] *

Target IOP Achieved	Progression of Damage	Duration of Control (months)	Approximate Follow-up Interval (months)**
Yes	No	≤6	6
Yes	No	>6	12
Yes	Yes	NA	1–2
No	Yes	NA	1–2
No	No	NA	3–6

IOP = intraocular pressure; NA = not applicable

* Evaluations consist of clinical examination of the patient, including optic nerve head assessment (with periodic color stereophotography or computerized imaging of the optic nerve and retinal nerve fiber layer structure) and visual field assessment.

** Patients with more advanced damage or greater lifetime risk from POAG may require more frequent evaluations. These intervals are the maximum recommended time between evaluations.

History

The following interval history can be elicited at POAG follow-up visits:

- Interval ocular history[A:III]
- Interval systemic medical history[B:III]
- Side effects of ocular medications[A:III]
- Frequency and time of last IOP-lowering medications and review of use of medications[B:III]

Ophthalmic examination

The following components of the ophthalmic examination should be performed at POAG follow-up visits:

- Visual acuity measurement[A:III]
- Slit-lamp biomicroscopy[A:III]
- Intraocular pressure measurement[A:I]

Based on the understanding of the effect of CCT on IOP measurements,[5,29,341] measurement of CCT should be repeated after any event (e.g., refractive surgery[342]) that may alter CCT.[A:II]

Gonioscopy

Gonioscopy is indicated when there is a suspicion of an angle-closure component, anterior chamber shallowing or anterior chamber angle abnormalities, or if there is an unexplained change in IOP.[A:III] Gonioscopy may also be performed periodically (e.g., 1 to 5 years).[A:III]

Optic nerve head and visual field evaluation[A:III]

Optic nerve head evaluation and documentation by imaging, photography, or drawing[139,163,343,344] and visual field evaluation[345-348] should be performed at the recommended intervals listed in Table 6.

Within each of the recommended intervals, factors that determine frequency of evaluations include the severity of damage (mild, moderate, severe, with more frequent evaluations for more severe disease), the rate of progression, the extent to which the IOP exceeds the target pressure, and the number and significance of other risk factors for damage to the optic nerve.[A:III] In certain cases, follow-up visual field testing may be required more frequently than the recommended intervals (e.g., a second test to establish a baseline for future comparisons, to clarify a suspicious test result, or to overcome an apparent testing artifact). For example, a patient with glaucomatous damage who has shown long-term stability can be followed every 6 to 12 months, depending on how severe the damage is, while a patient with evidence of glaucomatous progression may receive a change in care plan with more frequent follow-up.

Risk Factors for Progression

The risk factors for progression in eyes already diagnosed with OAG are related to the level of IOP and factors independent of IOP:

- Intraocular pressure: Several multicenter randomized clinical trials have investigated the relationship between IOP and risk of glaucomatous progression. Higher baseline IOP,[30] higher mean IOP during follow-up,[32,349] and higher yearly average IOP[350] were associated with greater progression of glaucoma as measured by visual field or optic nerve changes. Greater IOP fluctuation in some, but not all studies, has also been shown to be related to visual field progression, but this strongly correlated with absolute IOP level and may not be an independent risk factor.[33-37,185]
- Beta-zone peripapillary atrophy: Either the baseline presence[351,352] or the size [137,353] of peripapillary atrophy adjacent to the optic nerve (beta zone) has been related to visual field or optic nerve progression in several large prospective and retrospective studies.
- Older age[30,36,185,349,353,354]
- Disc hemorrhage: Either presence of a disc hemorrhage[351,353] or percentage of visits with disc hemorrhage[30,36] have been associated with progression of visual field defect or optic nerve damage. The association has been reported in both normal-tension and in high-pressure glaucoma.
- Larger cup-to-disc ratio or small optic nerve rim area[352,355]
- Thinner central cornea: Strong evidence exists for thinner central cornea as a risk factor for progression from ocular hypertension to POAG, but evidence is mixed for thinner central cornea as a risk factor for progression in glaucoma.[53,57,60,341,356-361]

Damage in one eye is associated with an increased risk of future damage in the other eye.[36,362,363] A retrospective study in eyes with OAG and severe visual field damage in one eye showed a risk of progression in the other eye (Kaplan Meier estimate of visual field progression = 12.1%).[364] Risk factors for progression were larger initial cup-to-disc ratio and lower calculated ocular perfusion pressure. In a separate retrospective study, progression in visual field damage between eyes showed a significant correlation.[363] In a large retrospective study of eyes with normal-tension glaucoma and unilateral visual field damage, the risk factors for progression in the normal eye were greater visual field damage in the eye with glaucoma and smaller neuroretinal rim area.[365]

Adjustment of Therapy

The indications for adjusting therapy are as follows:[A:III]

- Target IOP is not achieved and the benefits of a change in therapy outweigh the risks for the patient
- A patient has progressive optic nerve damage despite achieving the target IOP
- The patient is intolerant of the prescribed medical regimen
- The patient does not adhere to the prescribed medical regimen because of cost or other issues
- Contraindications to individual medicines develop
- Stable optic nerve status and low IOP occurs for a prolonged period in a patient on glaucoma medications. Under these circumstances, a carefully monitored attempt to reduce the medical regimen may be appropriate.

Downward adjustment of target pressure can be made in the face of progressive optic disc or visual field change.[366-370] [A:III]

Upward adjustment of target pressure can be considered if the patient has been stable and if the patient either requires (because of side effects) or desires less medication. A follow-up visit in 2 to 8 weeks may help to assess the response and side effects from washout of the old medication or onset of maximum effect of the new medication.

PROVIDER AND SETTING

The performance of certain diagnostic procedures (e.g., tonometry, pachymetry, perimetry, optic disc imaging, and photography) may be delegated to appropriately trained and supervised personnel.

However, the interpretation of results and medical and surgical management of the disease require the medical training, clinical judgment, and experience of the ophthalmologist.

Most diagnostic and therapeutic procedures can be safely undertaken on an outpatient basis. In some instances, however, hospitalization may be required. This includes, for example, patients who have special medical or social needs.

PHYSICIAN QUALITY REPORTING INITIATIVE

The Physician Quality Reporting Initiative program, initially launched by the Centers for Medicare and Medicaid Services in July 2007, encourages quality improvement through the use of clinical performance measures on a variety of clinical conditions. There are two measures in the 2010 Physician Quality Reporting Initiative program for POAG.[371] One measure is reduction of IOP by at least 15% from the preintervention level or documentation of a plan of care if treatment has not been successful in reducing IOP by at least 15%. The second measure is an optic nerve head evaluation within 12 months.[371]

COUNSELING/REFERRAL

It is important to educate and engage patients in the management of their condition. Patients should be educated about the disease process, the rationale and goals of intervention, the status of their condition, and the relative benefits and risks of alternative interventions so that they can participate meaningfully in developing an appropriate plan of action.[A:III] Patients should be encouraged to alert their ophthalmologists to physical or emotional changes that occur when taking glaucoma medications.[A:III] The diagnosis of glaucoma can itself lead to negative psychological effects and to fear of blindness.[372-376]

Numerous studies have been performed to characterize the psychological profile of the glaucoma patient, and some have shown the prevalence of anxiety to be higher in this population.[372,375,377] It has been much harder to demonstrate a consistent presence of depression in glaucoma patients; numerous studies have been unable to do so[106,372,378,379] and only a minority do.[375,376]

Glaucoma affects the patient's visual and health-related quality of life in many ways[91,380]; this includes employment issues (e.g., fear of loss of job and insurance from diminished ability to read and drive), social issues (e.g., fear of negative impact on relationships and sexuality), and loss of independence and activities that require good visual acuity (e.g., sports and other hobbies). The ophthalmologist should be sensitive to these problems and provide support and encouragement.[A:III] Some patients may find peer-support groups or counseling helpful.

Patients considering keratorefractive surgery should be informed about the possible impact laser vision correction has on reducing contrast sensitivity, altering visual field testing results, and decreasing the accuracy of IOP measurements.[A:III] During the conduct of LASIK the IOP will briefly increase from the effect of the suction ring to make the eye rigid during creation of the superficial flap. This effect may cause additional damage in patients whose optic nerves already have advanced damage.[381] Therefore, LASIK may be relatively contraindicated in such individuals, but photorefractive keratectomy may be possible. In addition, postoperative fluid may develop in the corneal flap-stromal interface and lead to temporary underestimation of the applanation IOP in patients treated aggressively with topical corticosteroids to resolve interface inflammation, who may actually have an undetected, corticosteroid-induced elevation of IOP.[382] Conversely, corticosteroid-induced IOP elevation may cause interface fluid that mimics interface inflammation and leads to IOP underestimation.[383,384] Patients with glaucomatous optic neuropathy considering implantation of a multifocal intraocular lens should be informed of the risk of reduced contrast sensitivity.[385] [A:III] It is important to establish preoperative and baseline documentation of optic nerve head status and visual field to facilitate subsequent glaucoma management.

If the diagnosis or management of POAG is in question or if the condition is refractory to treatment, consultation with or referral to an ophthalmologist with special training or experience in managing glaucoma should be considered. Patients with substantial visual impairment or blindness can be referred for and encouraged to use appropriate vision rehabilitation and social services.[386] [A:III] More information on vision rehabilitation, including materials for patients, is available at www.aao.org/smartsight.

APPENDIX 1. QUALITY OF OPHTHALMIC CARE CORE CRITERIA

Providing quality care
is the physician's foremost ethical obligation, and is
the basis of public trust in physicians.
AMA Board of Trustees, 1986

Quality ophthalmic care is provided in a manner and with the skill that is consistent with the best interests of the patient. The discussion that follows characterizes the core elements of such care.

The ophthalmologist is first and foremost a physician. As such, the ophthalmologist demonstrates compassion and concern for the individual, and utilizes the science and art of medicine to help alleviate patient fear and suffering. The ophthalmologist strives to develop and maintain clinical skills at the highest feasible level, consistent with the needs of patients, through training and continuing education. The ophthalmologist evaluates those skills and medical knowledge in relation to the needs of the patient and responds accordingly. The ophthalmologist also ensures that needy patients receive necessary care directly or through referral to appropriate persons and facilities that will provide such care, and he or she supports activities that promote health and prevent disease and disability.

The ophthalmologist recognizes that disease places patients in a disadvantaged, dependent state. The ophthalmologist respects the dignity and integrity of his or her patients, and does not exploit their vulnerability.

Quality ophthalmic care has the following optimal attributes, among others.

- The essence of quality care is a meaningful partnership relationship between patient and physician. The ophthalmologist strives to communicate effectively with his or her patients, listening carefully to their needs and concerns. In turn, the ophthalmologist educates his or her patients about the nature and prognosis of their condition and about proper and appropriate therapeutic modalities. This is to ensure their meaningful participation (appropriate to their unique physical, intellectual and emotional state) in decisions affecting their management and care, to improve their motivation and compliance with the agreed plan of treatment, and to help alleviate their fears and concerns.
- The ophthalmologist uses his or her best judgment in choosing and timing appropriate diagnostic and therapeutic modalities as well as the frequency of evaluation and follow-up, with due regard to the urgency and nature of the patient's condition and unique needs and desires.
- The ophthalmologist carries out only those procedures for which he or she is adequately trained, experienced and competent, or, when necessary, is assisted by someone who is, depending on the urgency of the problem and availability and accessibility of alternative providers.
- Patients are assured access to, and continuity of, needed and appropriate ophthalmic care, which can be described as follows.
  - The ophthalmologist treats patients with due regard to timeliness, appropriateness, and his or her own ability to provide such care.
  - The operating ophthalmologist makes adequate provision for appropriate pre- and postoperative patient care.
  - When the ophthalmologist is unavailable for his or her patient, he or she provides appropriate alternate ophthalmic care, with adequate mechanisms for informing patients of the existence of such care and procedures for obtaining it.
  - The ophthalmologist refers patients to other ophthalmologists and eye care providers based on the timeliness and appropriateness of such referral, the patient's needs, the competence and qualifications of the person to whom the referral is made, and access and availability.
  - The ophthalmologist seeks appropriate consultation with due regard to the nature of the ocular or other medical or surgical problem. Consultants are suggested for their skill, competence, and accessibility.

They receive as complete and accurate an accounting of the problem as necessary to provide efficient and effective advice or intervention, and in turn respond in an adequate and timely manner.

- The ophthalmologist maintains complete and accurate medical records.
- On appropriate request, the ophthalmologist provides a full and accurate rendering of the patient's records in his or her possession.
- The ophthalmologist reviews the results of consultations and laboratory tests in a timely and effective manner and takes appropriate actions.
- The ophthalmologist and those who assist in providing care identify themselves and their profession.
- For patients whose conditions fail to respond to treatment and for whom further treatment is unavailable, the ophthalmologist provides proper professional support, counseling, rehabilitative and social services, and referral as appropriate and accessible.

- Prior to therapeutic or invasive diagnostic procedures, the ophthalmologist becomes appropriately conversant with the patient's condition by collecting pertinent historical information and performing relevant preoperative examinations. Additionally, he or she enables the patient to reach a fully informed decision by providing an accurate and truthful explanation of the diagnosis; the nature, purpose, risks, benefits, and probability of success of the proposed treatment and of alternative treatment; and the risks and benefits of no treatment.
- The ophthalmologist adopts new technology (e.g., drugs, devices, surgical techniques) in judicious fashion, appropriate to the cost and potential benefit relative to existing alternatives and to its demonstrated safety and efficacy.
- The ophthalmologist enhances the quality of care he or she provides by periodically reviewing and assessing his or her personal performance in relation to established standards, and by revising or altering his or her practices and techniques appropriately.
- The ophthalmologist improves ophthalmic care by communicating to colleagues, through appropriate professional channels, knowledge gained through clinical research and practice. This includes alerting colleagues of instances of unusual or unexpected rates of complications and problems related to new drugs, devices or procedures.
- The ophthalmologist provides care in suitably staffed and equipped facilities adequate to deal with potential ocular and systemic complications requiring immediate attention.
- The ophthalmologist also provides ophthalmic care in a manner that is cost effective without unacceptably compromising accepted standards of quality.

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APPENDIX 2. MAJOR RECOMMENDATIONS FOR CARE

DIAGNOSIS

The comprehensive initial glaucoma evaluation (history and physical examination) includes all components of the comprehensive adult medical eye evaluation[102] in addition to, and with special attention to, those factors that specifically bear upon the diagnosis, course, and treatment of POAG.

Evaluation of Visual Function

Self-reported functional status or difficulty with vision can be assessed either by patient complaints or by using specific questionnaires, including the National Eye Institute - Visual Function Questionnaire-25.[90,103-109] [A:III]

Ophthalmic Evaluation

History

- Ocular,[A:III] family,[4,42,44] [A:II] and systemic history (e.g., asthma).[A:III] The severity and outcome of glaucoma in family members, including history of visual loss from glaucoma, should be obtained during initial evaluation.[42,44] [B:III]
- Review of pertinent records,[A:III] with particular reference to the past IOP levels, status of the optic nerve, and visual field.[A:III]
- Current ocular and systemic medications (e.g., corticosteroids) and known local or systemic intolerance to ocular or systemic medications.[A:III]
- Ocular surgery.[A:III]

Visual acuity measurement

Visual acuity with current correction (the power of the present correction recorded) at distance and, when appropriate, at near should be measured. [A:III] Refraction may be indicated to obtain the best-corrected visual acuity.

Pupil examination

The pupils are examined for reactivity and an afferent pupillary defect.[115-117] [B:II]

Anterior segment examination

A slit-lamp biomicroscopic examination of the anterior segment can provide evidence of physical findings associated with narrow angles, such as shallow peripheral anterior chamber depth and crowded anterior chamber angle anatomy,[118,119] corneal pathology, or a secondary mechanism for elevated IOP such as pseudoexfoliation (exfoliation syndrome), pigment dispersion with Krukenberg spindle and/or iris transillumination defects, iris and angle neovascularization, or inflammation.[A:III]

Intraocular pressure measurement

Intraocular pressure is measured in each eye, preferably by Goldmann applanation tonometry, before gonioscopy or dilation of the pupil. [5,27,30-32,120-128] [A:I] Recording time of day of IOP measurements may be helpful to assess diurnal variation. Unrecognized fluctuations in IOP may lead to progression of POAG.[129-132] Therefore, additional measurements may be indicated, either at different hours of the day on the same day or on different days.

Gonioscopy

The diagnosis of POAG requires careful evaluation of the anterior chamber angle to exclude angle closure or secondary causes of IOP elevation, such as angle recession, pigment dispersion, peripheral anterior synechiae, angle neovascularization, and inflammatory precipitates.[133] [A:III]

Optic nerve head and retinal nerve fiber layer examination

The preferred technique for optic nerve head and retinal nerve fiber layer evaluation involves magnified stereoscopic visualization (as with the slit-lamp biomicroscope), preferably through a dilated pupil.[A:III]

Fundus examination

Examination of the fundus, through a dilated pupil whenever feasible, includes a search for other abnormalities that may account for optic nerve changes and/or visual field defects (e.g., optic nerve pallor, disc drusen, optic nerve pits, disc edema from central nervous system disease, macular degeneration, retinovascular occlusion, and other retinal disease).[A:III]

Supplemental Ophthalmic Testing

Central corneal thickness measurement

Measurement of CCT aids the interpretation of IOP readings and helps to stratify patient risk for ocular damage.[29,53,57,151] [A:II]

Visual field evaluation

Automated static threshold perimetry is the preferred technique for evaluating the visual field.[154] [A:III] Careful manual combined kinetic and static threshold testing (e.g., Goldmann visual fields) is an acceptable alternative when patients cannot perform automated perimetry reliably or if it is not available.[A:III] Repeat, confirmatory visual field examinations may be required for test results that are unreliable or show a new glaucomatous defect before changing management.[27,124] [A:III]

Optic nerve head and retinal nerve fiber layer analysis

The appearance of the optic nerve should be documented.[136,158] [A:II] Color stereophotography is an accepted method for documenting optic nerve head appearance. Computer-based image analysis of the optic nerve head and retinal nerve fiber layer is an alternative for documentation of the optic nerve. In the absence of these technologies, a nonstereoscopic photograph or a drawing of the optic nerve head should be recorded, but these are less desirable alternatives to stereophotography or computer-based imaging.[163] [A:III]

Management recommendations are described in the main body of the text.

Follow-up Evaluation

Guidelines for follow-up of patients with POAG are summarized in Table 6. These recommendations apply to ongoing glaucoma management and not to visits for other purposes. Follow-up evaluation includes examination as well as optic nerve head and visual field assessment as indicated.

TABLE 6 RECOMMENDED GUIDELINES FOR FOLLOW-UP GLAUCOMA STATUS EVALUATIONS WITH OPTIC NERVE AND VISUAL FIELD ASSESSMENT[B:III] *

Target IOP Achieved	Progression of Damage	Duration of Control (months)	Approximate Follow-up Interval (months)**
Yes	No	≤6	6
Yes	No	>6	12
Yes	Yes	NA	1–2
No	Yes	NA	1–2
No	No	NA	3–6

IOP = intraocular pressure; NA = not applicable

* Evaluations consist of clinical examination of the patient, including optic nerve head assessment (with periodic color stereophotography or computerized imaging of the optic nerve and retinal nerve fiber layer structure) and visual field assessment.

** Patients with more advanced damage or greater lifetime risk from POAG may require more frequent evaluations. These intervals are the maximum recommended time between evaluations.

History

- Interval ocular history[A:III]
- Interval systemic medical history[B:III]
- Side effects of ocular medications[A:III]
- Frequency and time of last IOP-lowering medications and review of use of medications[B:III]

Ophthalmic examination

- Visual acuity measurement[A:III]
- Slit-lamp biomicroscopy[A:III]
- Intraocular pressure measurement[A:I]

Based on the understanding of the effect of CCT on IOP measurements,[5,29,341] measurement of CCT should be repeated after any event (e.g., refractive surgery[342]) that may alter CCT.[A:II]

Gonioscopy

Gonioscopy is indicated when there is a suspicion of an angle-closure component, anterior chamber shallowing or anterior chamber angle abnormalities, or if there is an unexplained change in IOP.[A:III] Gonioscopy may also be performed periodically (e.g., 1 to 5 years).[A:III]

Optic nerve head and visual field evaluation[A:III]

Optic nerve head evaluation and documentation by imaging, photography, or drawing[139,163,343,344] and visual field evaluation[345-348] should be performed at the recommended intervals listed in Table 6.

Within each of the recommended intervals, factors that determine frequency of evaluations include the severity of damage (mild, moderate, severe, with more frequent evaluations for more severe disease), the rate of progression, the extent to which the IOP exceeds the target pressure, and the number and significance of other risk factors for damage to the optic nerve.[A:III]

COUNSELING/REFERRAL

Patients should be educated about the disease process, the rationale and goals of intervention, the status of their condition, and the relative benefits and risks of alternative interventions so that they can participate meaningfully in developing an appropriate plan of action.[A:III] Patients should be encouraged to alert their ophthalmologists to physical or emotional changes that occur when taking glaucoma medications.[A:III]

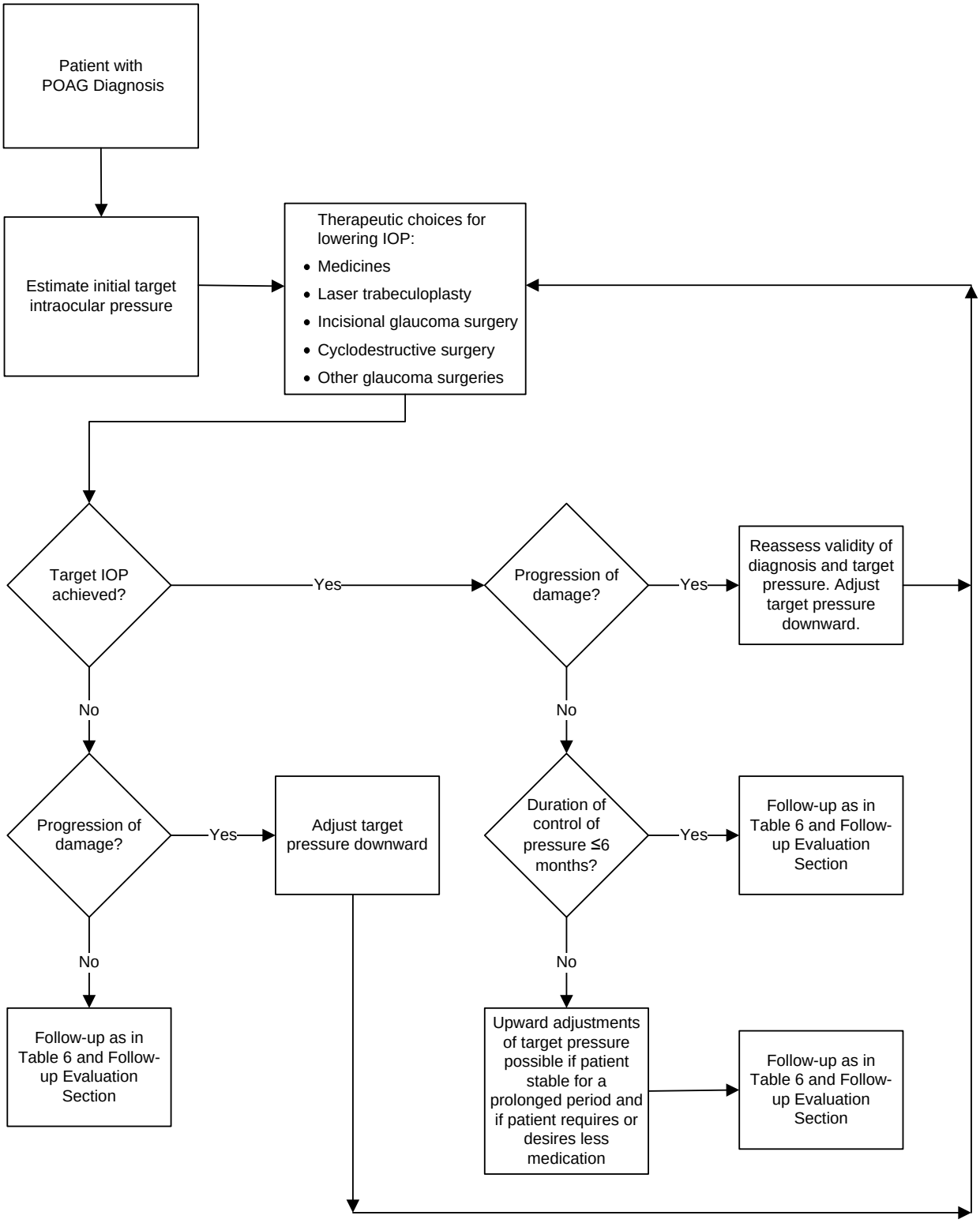
Glaucoma affects the patient's visual and health-related quality of life in many ways[91,380]; this includes employment issues (e.g., fear of loss of job and insurance from diminished ability to read

and drive), social issues (e.g., fear of negative impact on relationships and sexuality), and loss of independence and activities that require good visual acuity (e.g., sports and other hobbies). The ophthalmologist should be sensitive to these problems and provide support and encouragement.[A:III] Some patients may find peer-support groups or counseling helpful.

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Patients with substantial visual impairment or blindness can be referred for and encouraged to use appropriate vision rehabilitation and social services.[386] [A:III] More information on vision rehabilitation, including materials for patients, is available at www.aao.org/smartsight.

APPENDIX 3. TREATMENT ALGORITHM FOR PATIENTS WITH PRIMARY OPEN-ANGLE GLAUCOMA



APPENDIX 4. INTERNATIONAL STATISTICAL CLASSIFICATION OF DISEASES AND RELATED HEALTH PROBLEMS (ICD) CODES

The POAG PPP covers the entity of open-angle glaucoma (ICD-9 #365.10) and related entities with the following ICD-9 classifications:

- Primary open-angle glaucoma (365.11)
- Low-tension glaucoma (365.12)
- Residual stage of open-angle glaucoma (365.15)
- Glaucomatous atrophy of the optic disc (377.14)

SUGGESTED REFERENCE TEXTS

- Allingham RR, Damji KF, Freedman S, Moroi SE, Shafranov G, Shields MB, eds. Shields' Textbook of Glaucoma. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2005.
- Alward WLM. www.gonioscopy.org. Accessed September 16, 2010.
- Alward WLM, Longmuir RA. Color Atlas of Gonioscopy. 2nd ed. San Francisco, CA: American Academy of Ophthalmology; 2008.
- Anderson DR, Patella VM. Automated Static Perimetry. 2nd ed. St. Louis, MO: CV Mosby Co.; 1999.
- Epstein DL, Allingham RR, Shuman JS, eds. Chandler and Grant's Glaucoma. 4th ed. Baltimore, MD: Lippincott Williams and Wilkins; 1997.
- Ritch R, Shields MB, Krupin T, eds. The Glaucomas. 2nd ed. St. Louis, MO: CV Mosby Co.; 1996.
- Stamper RL, Lieberman MF, Drake MV. Becker-Shaffer's Diagnosis and Therapy of the Glaucomas. 8th ed. Philadelphia, PA: Mosby Elsevier; 2009.
- Tasman W, Jaeger EA, eds. Duane's Ophthalmology. 15th edition. Philadelphia, PA: Lippincott Williams & Wilkins; 2009.
- Weinreb RN, Greve EL, eds. Glaucoma Diagnosis: Structure and Function. World Glaucoma Association Consensus Series - 1. The Netherlands: Kugler Publications; 2004.
- Weinreb RN, Crowston JG, eds. Glaucoma Surgery: Open Angle Glaucoma. World Glaucoma Association Consensus Series - 2. The Netherlands: Kugler Publications; 2005.
- Weinreb RN, Brandt JD, Garway-Heath D, Medeiros FA, eds. Intraocular Pressure. World Glaucoma Association Consensus Series - 4. The Netherlands: Kugler Publications; 2007.
- Weinreb RN, Healy PR, Topouzis F, eds. Glaucoma Screening. World Glaucoma Association Consensus Series - 5. The Netherlands: Kugler Publications; 2008.

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REFERENCES

1. National Collaborating Centre for Acute Care. Glaucoma: diagnosis and management of chronic open angle glaucoma and ocular hypertension. Appendices A-G. Available at: www.nice.org.uk/nicemedia/live/12145/43888/43888.pdf. Accessed April 2, 2010.
2. National Collaborating Centre for Acute Care. Glaucoma: diagnosis and management of chronic open angle glaucoma and ocular hypertension. Methods, evidence & guidance. Available at: www.nice.org.uk/nicemedia/live/12145/43887/43887.pdf. Accessed April 2, 2010.
3. Foster PJ, Buhrmann R, Quigley HA, Johnson GJ. The definition and classification of glaucoma in prevalence surveys. Br J Ophthalmol 2002;86:238-42.
4. Dielemans I, Vingerling JR, Wolfs RC, et al. The prevalence of primary open-angle glaucoma in a population-based study in The Netherlands. The Rotterdam Study. Ophthalmology 1994;101:1851-5.
5. Kass MA, Heuer DK, Higginbotham EJ, et al. The Ocular Hypertension Treatment Study: a randomized trial determines that topical ocular hypotensive medication delays or prevents the onset of primary open-angle glaucoma. Arch Ophthalmol 2002;120:701-13; discussion 829-30.
6. Quigley HA, Broman AT. The number of people with glaucoma worldwide in 2010 and 2020. Br J Ophthalmol 2006;90:262-7.
7. Friedman DS, Wolfs RC, O'Colmain BJ, et al. Prevalence of open-angle glaucoma among adults in the United States. Arch Ophthalmol 2004;122:532-8.
8. Sommer A, Tielsch JM, Katz J, et al. Racial differences in the cause-specific prevalence of blindness in east Baltimore. N Engl J Med 1991;325:1412-7.
9. Varma R, Ying-Lai M, Francis BA, et al, Los Angeles Latino Eye Study Group. Prevalence of open-angle glaucoma and ocular hypertension in Latinos: the Los Angeles Latino Eye Study. Ophthalmology 2004;111:1439-48.
10. Brandt JD, Beiser JA, Kass MA, Gordon MO. Central corneal thickness in the Ocular Hypertension Treatment Study (OHTS). Ophthalmology 2001;108:1779-88.
11. Tielsch JM, Katz J, Sommer A, et al. Hypertension, perfusion pressure, and primary open-angle glaucoma. A population-based assessment. Arch Ophthalmol 1995;113:216-21.
12. Leske MC, Connell AM, Wu SY, et al. Risk factors for open-angle glaucoma. The Barbados Eye Study. Arch Ophthalmol 1995;113:918-24.
13. Mitchell P, Smith W, Chey T, Healey PR. Open-angle glaucoma and diabetes: the Blue Mountains Eye Study, Australia. Ophthalmology 1997;104:712-8.
14. Chopra V, Varma R, Francis BA, et al. Los Angeles Latino Eye Study Group. Type 2 diabetes mellitus and the risk of open-angle glaucoma: the Los Angeles Latino Eye Study. Ophthalmology 2008;115:227-32.
15. Bonovas S, Peponis V, Filioussi K. Diabetes mellitus as a risk factor for primary open-angle glaucoma: a meta-analysis. Diabet Med 2004;21:609-14.
16. Mitchell P, Hourihan F, Sandbach J, Wang JJ. The relationship between glaucoma and myopia: the Blue Mountains Eye Study. Ophthalmology 1999;106:2010-5.
17. Grodum K, Heijl A, Bengtsson B. Refractive error and glaucoma. Acta Ophthalmol Scand 2001;79:560-6.
18. Xu L, Wang Y, Wang S, Jonas JB. High myopia and glaucoma susceptibility the Beijing Eye Study. Ophthalmology 2007;114:216-20.
19. Fingert JH, Heon E, Liebmann JM, et al. Analysis of myocilin mutations in 1703 glaucoma patients from five different populations. Hum Mol Genet 1999;8:899-905.
20. Sommer A, Tielsch JM, Katz J, et al. Relationship between intraocular pressure and primary open angle glaucoma among white and black Americans. The Baltimore Eye Survey. Arch Ophthalmol 1991;109:1090-5.

21. Mitchell P, Smith W, Attebo K, Healey PR. Prevalence of open-angle glaucoma in Australia. The Blue Mountains Eye Study. Ophthalmology 1996;103:1661-9.
22. Leske MC, Connell AM, Schachat AP, Hyman L. The Barbados Eye Study. Prevalence of open angle glaucoma. Arch Ophthalmol 1994;112:821-9.
23. Quigley HA, West SK, Rodriguez J, et al. The prevalence of glaucoma in a population-based study of Hispanic subjects: Proyecto VER. Arch Ophthalmol 2001;119:1819-26.
24. Leibowitz HM, Krueger DE, Maunder LR, et al. The Framingham Eye Study monograph: An ophthalmological and epidemiological study of cataract, glaucoma, diabetic retinopathy, macular degeneration, and visual acuity in a general population of 2631 adults, 1973-1975. Surv Ophthalmol 1980;24:335-610.
25. Klein BE, Klein R, Sponsel WE, et al. Prevalence of glaucoma. The Beaver Dam Eye Study. Ophthalmology 1992;99:1499-504.
26. Weih LM, Nanjan M, McCarty CA, Taylor HR. Prevalence and predictors of open-angle glaucoma: results from the visual impairment project. Ophthalmology 2001;108:1966-72.
27. Collaborative Normal-Tension Glaucoma Study Group. Comparison of glaucomatous progression between untreated patients with normal-tension glaucoma and patients with therapeutically reduced intraocular pressures. Am J Ophthalmol 1998;126:487-97.
28. Collaborative Normal-Tension Glaucoma Study Group. The effectiveness of intraocular pressure reduction in the treatment of normal-tension glaucoma. Am J Ophthalmol 1998;126:498-505.
29. Gordon MO, Beiser JA, Brandt JD, et al. The Ocular Hypertension Treatment Study: baseline factors that predict the onset of primary open-angle glaucoma. Arch Ophthalmol 2002;120:714-20; discussion 829-30.
30. Leske MC, Heijl A, Hussein M, et al, Early Manifest Glaucoma Trial Group. Factors for glaucoma progression and the effect of treatment: the Early Manifest Glaucoma Trial. Arch Ophthalmol 2003;121:48-56.
31. Heijl A, Leske MC, Bengtsson B, et al, Early Manifest Glaucoma Trial Group. Reduction of intraocular pressure and glaucoma progression: results from the Early Manifest Glaucoma Trial. Arch Ophthalmol 2002;120:1268-79.
32. AGIS Investigators. The Advanced Glaucoma Intervention Study (AGIS): 7. The relationship between control of intraocular pressure and visual field deterioration. Am J Ophthalmol 2000;130:429-40.
33. Nouri-Mahdavi K, Hoffman D, Coleman AL, et al. Predictive factors for glaucomatous visual field progression in the Advanced Glaucoma Intervention Study. Ophthalmology 2004;111:1627-35.
34. Asrani S, Zeimer R, Wilensky J, et al. Large diurnal fluctuations in intraocular pressure are an independent risk factor in patients with glaucoma. J Glaucoma 2000;9:134-42.
35. Caprioli J, Coleman AL. Intraocular pressure fluctuation a risk factor for visual field progression at low intraocular pressures in the advanced glaucoma intervention study. Ophthalmology 2008;115:1123-9 e3.
36. Leske MC, Heijl A, Hyman L, et al. Predictors of long-term progression in the Early Manifest Glaucoma Trial. Ophthalmology 2007;114:1965-72.
37. Nouri-Mahdavi K, Medeiros FA, Weinreb RN. Fluctuation of intraocular pressure as a predictor of visual field progression. Arch Ophthalmol 2008;126:1168-9; author reply 9-70.
38. Bonomi L, Marchini G, Marraffa M, et al. Prevalence of glaucoma and intraocular pressure distribution in a defined population. The Egna-Neumarkt Study. Ophthalmology 1998;105:209-15.
39. Wensor MD, McCarty CA, Stanislavsky YL, et al. The prevalence of glaucoma in the Melbourne Visual Impairment Project. Ophthalmology 1998;105:733-9.
40. Tielsch JM, Sommer A, Katz J, et al. Racial variations in the prevalence of primary open-angle glaucoma. The Baltimore Eye Survey. JAMA 1991;266:369-74.
41. Friedman DS, Jampel HD, Munoz B, West SK. The prevalence of open-angle glaucoma among blacks and whites 73 years and older: the Salisbury Eye Evaluation Glaucoma Study. Arch Ophthalmol 2006;124:1625-30.

42. Wolfs RC, Klaver CC, Ramrattan RS, et al. Genetic risk of primary open-angle glaucoma. Population-based familial aggregation study. Arch Ophthalmol 1998;116:1640-5.
43. Doshi V, Ying-Lai M, Azen SP, Varma R, Los Angeles Latino Eye Study Group. Sociodemographic, family history, and lifestyle risk factors for open-angle glaucoma and ocular hypertension. The Los Angeles Latino Eye Study. Ophthalmology 2008;115:639-47.
44. Tielsch JM, Katz J, Sommer A, et al. Family history and risk of primary open angle glaucoma. The Baltimore Eye Survey. Arch Ophthalmol 1994;112:69-73.
45. Rotchford AP, Johnson GJ. Glaucoma in Zulus: a population-based cross-sectional survey in a rural district in South Africa. Arch Ophthalmol 2002;120:471-8.
46. Rotchford AP, Kirwan JF, Muller MA, et al. Temba glaucoma study: a population-based cross-sectional survey in urban South Africa. Ophthalmology 2003;110:376-82.
47. Shah S, Chatterjee A, Mathai M, et al. Relationship between corneal thickness and measured intraocular pressure in a general ophthalmology clinic. Ophthalmology 1999;106:2154-60.
48. Whitacre MM, Stein RA, Hassanein K. The effect of corneal thickness on applanation tonometry. Am J Ophthalmol 1993;115:592-6.
49. Goldmann H, Schmidt T. Applanation tonometry [in German]. Ophthalmologica 1957;134:221-42.
50. Ehlers N, Bramsen T, Sperling S. Applanation tonometry and central corneal thickness. Acta Ophthalmol (Copenh) 1975;53:34-43.
51. Stodtmeister R. Applanation tonometry and correction according to corneal thickness. Acta Ophthalmol Scand 1998;76:319-24.
52. Doughty MJ, Zaman ML. Human corneal thickness and its impact on intraocular pressure measures: a review and meta-analysis approach. Surv Ophthalmol 2000;44:367-408.
53. Medeiros FA, Sample PA, Zangwill LM, et al. Corneal thickness as a risk factor for visual field loss in patients with preperimetric glaucomatous optic neuropathy. Am J Ophthalmol 2003;136:805-13.
54. Hahn S, Azen S, Ying-Lai M, Varma R, Los Angeles Latino Eye Study Group. Central corneal thickness in Latinos. Invest Ophthalmol Vis Sci 2003;44:1508-12.
55. Shimmyo M, Ross AJ, Moy A, Mostafavi R. Intraocular pressure, Goldmann applanation tension, corneal thickness, and corneal curvature in Caucasians, Asians, Hispanics, and African Americans. Am J Ophthalmol 2003;136:603-13.
56. Orssengo GJ, Pye DC. Determination of the true intraocular pressure and modulus of elasticity of the human cornea in vivo. Bull Math Biol 1999;61:551-72.
57. Dueker DK, Singh K, Lin SC, et al. Corneal thickness measurement in the management of primary open-angle glaucoma: a report by the American Academy of Ophthalmology. Ophthalmology 2007;114:1779-87.
58. Francis BA, Varma R, Chopra V, et al, Los Angeles Latino Eye Study Group. Intraocular pressure, central corneal thickness, and prevalence of open-angle glaucoma: the Los Angeles Latino Eye Study. Am J Ophthalmol 2008;146:741-6.
59. Liu J, Roberts CJ. Influence of corneal biomechanical properties on intraocular pressure measurement: quantitative analysis. J Cataract Refract Surg 2005;31:146-55.
60. Congdon NG, Broman AT, Bandeen-Roche K, et al. Central corneal thickness and corneal hysteresis associated with glaucoma damage. Am J Ophthalmol 2006;141:868-75.
61. Bonomi L, Marchini G, Marraffa M, et al. Vascular risk factors for primary open angle glaucoma: the Egna-Neumarkt Study. Ophthalmology 2000;107:1287-93.
62. Memarzadeh F, Ying-Lai M, Chung J, et al, Los Angeles Latino Eye Study Group. Blood pressure, perfusion pressure, and open-angle glaucoma: the Los Angeles Latino Eye Study. Invest Ophthalmol Vis Sci 2010;51:2872-7.
63. Dielemans I, de Jong PT, Stolk R, et al. Primary open-angle glaucoma, intraocular pressure, and diabetes mellitus in the general elderly population. The Rotterdam Study. Ophthalmology 1996;103:1271-5.
64. Le A, Mukesh BN, McCarty CA, Taylor HR. Risk factors associated with the incidence of open-angle glaucoma: the visual impairment project. Invest Ophthalmol Vis Sci 2003;44:3783-9.

65. Pasquale LR, Kang JH, Manson JE, et al. Prospective study of type 2 diabetes mellitus and risk of primary open-angle glaucoma in women. Ophthalmology 2006;113:1081-6.
66. de Voogd S, Ikram MK, Wolfs RC, et al. Is diabetes mellitus a risk factor for open-angle glaucoma? The Rotterdam Study. Ophthalmology 2006;113:1827-31.
67. Klein BE, Klein R, Jensen SC. Open-angle glaucoma and older-onset diabetes. The Beaver Dam Eye Study. Ophthalmology 1994;101:1173-7.
68. Nakamura M, Kanamori A, Negi A. Diabetes mellitus as a risk factor for glaucomatous optic neuropathy. Ophthalmologica 2005;219:1-10.
69. Vijaya L, George R, Paul PG, et al. Prevalence of open-angle glaucoma in a rural south Indian population. Invest Ophthalmol Vis Sci 2005;46:4461-7.
70. Wong TY, Klein BE, Klein R, et al. Refractive errors, intraocular pressure, and glaucoma in a white population. Ophthalmology 2003;110:211-7.
71. Ramakrishnan R, Nirmalan PK, Krishnadas R, et al. Glaucoma in a rural population of southern India: the Aravind comprehensive eye survey. Ophthalmology 2003;110:1484-90.
72. Suzuki Y, Iwase A, Araie M, et al. Risk factors for open-angle glaucoma in a Japanese population: the Tajimi Study. Ophthalmology 2006;113:1613-7.
73. Wu SY, Nemesure B, Leske MC. Glaucoma and myopia. Ophthalmology 2000;107:1026-7.
74. Kuzin AA, Varma R, Reddy HS, et al. Ocular biometry and open-angle glaucoma: The Los Angeles Latino Eye Study. Ophthalmology 2010;117:1713-19.
75. Gong G, Kosoko-Lasaki O, Haynatzki GR, Wilson MR. Genetic dissection of myocilin glaucoma. Hum Mol Genet 2004;13 Spec No 1:R91-102.
76. Kwon YH, Fingert JH, Kuehn MH, Alward WL. Primary open-angle glaucoma. N Engl J Med 2009;360:1113-24.
77. Wiggs JL. Genetic etiologies of glaucoma. Arch Ophthalmol 2007;125:30-7.
78. Wang J, Mitchell P, Smith W. Is there an association between migraine headache and open-angle glaucoma? Findings from the Blue Mountains Eye Study. Ophthalmology 1997;104:1714-19.
79. Broadway DC, Drance SM. Glaucoma and vasospasm. Br J Ophthalmol 1998;82:862-70.
80. Cursiefen C, Wisse M, Cursiefen S, et al. Migraine and tension headache in high-pressure and normal-pressure glaucoma. Am J Ophthalmol 2000;129:102-4.
81. Armaly MF, Krueger DE, Maunder L, et al. Biostatistical analysis of the collaborative glaucoma study. I. Summary report of the risk factors for glaucomatous visual-field defects. Arch Ophthalmol 1980;98:2163-71.
82. AGIS Investigators. The Advanced Glaucoma Intervention Study (AGIS): 3. Baseline characteristics of black and white patients. Ophthalmology 1998;105:1137-45.
83. Dielemans I, Vingerling JR, Algra D, et al. Primary open-angle glaucoma, intraocular pressure, and systemic blood pressure in the general elderly population. The Rotterdam Study. Ophthalmology 1995;102:54-60.
84. Mitchell P, Lee AJ, Rochtchina E, Wang JJ. Open-angle glaucoma and systemic hypertension: the Blue Mountains Eye Study. J Glaucoma 2004;13:319-26.
85. Leske MC, Wu SY, Nemesure B, Hennis A. Incident open-angle glaucoma and blood pressure. Arch Ophthalmol 2002;120:954-9.
86. Hernandez RA, Burr JM, Vale LD. Economic evaluation of screening for open-angle glaucoma. Int J Technol Assess Health Care 2008;24:203-11.
87. Klein BE, Klein R, Lee KE. Heritability of risk factors for primary open-angle glaucoma: the Beaver Dam Eye Study. Invest Ophthalmol Vis Sci 2004;45:59-62.
88. Duggal P, Klein AP, Lee KE, et al. A genetic contribution to intraocular pressure: the Beaver Dam Eye Study. Invest Ophthalmol Vis Sci 2005;46:555-60.
89. Mitchell P, Rochtchina E, Lee AJ, Wang JJ. Bias in self-reported family history and relationship to glaucoma: the Blue Mountains Eye Study. Ophthalmic Epidemiol 2002;9:333-45.
90. Freeman EE, Munoz B, West SK, et al. Glaucoma and quality of life: the Salisbury Eye Evaluation. Ophthalmology 2008;115:233-8.

91. McKean-Cowdin R, Wang Y, Wu J, et al, Los Angeles Latino Eye Study Group. Impact of visual field loss on health-related quality of life in glaucoma: the Los Angeles Latino Eye Study. Ophthalmology 2008;115:941-8.
92. U.S. Preventive Services Task Force. Screening for Glaucoma: Recommendation Statement. AHRQ Publication No. 04-0548-A, March 2005. http://www.uspreventiveservicestaskforce.org/uspstf05/glaucoma/glaucrs.htm. Accessed August 17, 2010.
93. Tielsch JM, Katz J, Singh K, et al. A population-based evaluation of glaucoma screening: the Baltimore Eye Survey. Am J Epidemiol 1991;134:1102-10.
94. Hollows FC, Graham P. Intraocular pressure, glaucoma and glaucoma suspects in a defined population. Br J Ophthalmol 1966;50:570-86.
95. Coffey M, Reidy A, Wormald R, et al. Prevalence of glaucoma in the west of Ireland. Br J Ophthalmol 1993;77:17-21.
96. Sommer A. Disabling visual disorders. Norwalk: Appleton-Century-Crofts, 1986; 1297-311.
97. Lichter PR. Variability of expert observers in evaluating the optic disc. Trans Am Ophthalmol Soc 1976;74:532-72.
98. Kahn HA, Leibowitz HM, Ganley JP. Standardizing diagnostic procedures. Am J Ophthalmol 1975;79:768-75.
99. Johnson CA, Keltner JL. Incidence of visual field loss in 20,000 eyes and its relationship to driving performance. Arch Ophthalmol 1983;101:371-5.
100. Quigley HA. Identification of glaucoma-related visual field abnormality with the screening protocol of frequency doubling technology. Am J Ophthalmol 1998;125:819-29.
101. Tatemichi M, Nakano T, Tanaka K, et al. Performance of glaucoma mass screening with only a visual field test using frequency-doubling technology perimetry. Am J Ophthalmol 2002;134:529-37.
102. American Academy of Ophthalmology Preferred Practice Patterns Committee. Preferred Practice Pattern® Guidelines. Comprehensive Adult Medical Eye Evaluation. San Francisco, CA: American Academy of Ophthalmology; 2010. Available at: www.aao.org/ppp.
103. Gutierrez P, Wilson MR, Johnson C, et al. Influence of glaucomatous visual field loss on health-related quality of life. Arch Ophthalmol 1997;115:777-84.
104. Lee BL, Gutierrez P, Gordon M, et al. The Glaucoma Symptom Scale. A brief index of glaucoma-specific symptoms. Arch Ophthalmol 1998;116:861-6.
105. Parrish RK II, Gedde SJ, Scott IU, et al. Visual function and quality of life among patients with glaucoma. Arch Ophthalmol 1997;115:1447-55.
106. Wilson MR, Coleman AL, Yu F, et al. Functional status and well-being in patients with glaucoma as measured by the Medical Outcomes Study Short Form-36 questionnaire. Ophthalmology 1998;105:2112-6.
107. Aspinall PA, Johnson ZK, Azuara-Blanco A, et al. Evaluation of quality of life and priorities of patients with glaucoma. Invest Ophthalmol Vis Sci 2008;49:1907-15.
108. Goldberg I, Clement CI, Chiang TH, et al. Assessing quality of life in patients with glaucoma using the Glaucoma Quality of Life-15 (GQL-15) questionnaire. J Glaucoma 2009;18:6-12.
109. Spaeth G, Walt J, Keener J. Evaluation of quality of life for patients with glaucoma. Am J Ophthalmol 2006;141:S3-14.
110. McKean-Cowdin R, Varma R, Wu J, et al, Los Angeles Latino Eye Study Group. Severity of visual field loss and health-related quality of life. Am J Ophthalmol 2007;143:1013-23.
111. Ringsdorf L, McGwin G, Jr., Owsley C. Visual field defects and vision-specific health-related quality of life in African Americans and whites with glaucoma. J Glaucoma 2006;15:414-8.
112. Varma R, Wu J, Chong K, et al, Los Angeles Latino Eye Study Group. Impact of severity and bilaterality of visual impairment on health-related quality of life. Ophthalmology 2006;113:1846-53.

113. Svedberg H, Chen E, Hamberg-Nystrom H. Changes in corneal thickness and curvature after different excimer laser photorefractive procedures and their impact on intraocular pressure measurements. Graefes Arch Clin Exp Ophthalmol 2005;243:1218-20.
114. Friedman DS, Jampel HD, Lubomski LH, et al. Surgical strategies for coexisting glaucoma and cataract: an evidence-based update. Ophthalmology 2002;109:1902-13.
115. Kohn AN, Moss AP, Podos SM. Relative afferent pupillary defects in glaucoma without characteristic field loss. Arch Ophthalmol 1979;97:294-6.
116. Brown RH, Zilis JD, Lynch MG, Sanborn GE. The afferent pupillary defect in asymmetric glaucoma. Arch Ophthalmol 1987;105:1540-3.
117. Kerrison JB, Buchanan K, Rosenberg ML, et al. Quantification of optic nerve axon loss associated with a relative afferent pupillary defect in the monkey. Arch Ophthalmol 2001;119:1333-41.
118. Foster PJ, Devereux JG, Alsbirk PH, et al. Detection of gonioscopically occludable angles and primary angle closure glaucoma by estimation of limbal chamber depth in Asians: modified grading scheme. Br J Ophthalmol 2000;84:186-92.
119. Van Herick W, Shaffer RN, Schwartz A. Estimation of width of angle of anterior chamber. Incidence and significance of the narrow angle. Am J Ophthalmol 1969;68:626-9.
120. Jay JL, Allan D. The benefit of early trabeculectomy versus conventional management in primary open angle glaucoma relative to severity of disease. Eye (Lond) 1989;3 (Pt 5):528-35.
121. Glaucoma Laser Trial Research Group. The Glaucoma Laser Trial (GLT). 2. Results of argon laser trabeculoplasty versus topical medicines. Ophthalmology 1990;97:1403-13.
122. Glaucoma Laser Trial Research Group. The Glaucoma Laser Trial (GLT) and Glaucoma Laser Trial Follow-up Study: 7. Results. Am J Ophthalmol 1995;120:718-31.
123. AGIS Investigators. The Advanced Glaucoma Intervention Study (AGIS): 13. Comparison of treatment outcomes within race: 10-year results. Ophthalmology 2004;111:651-64.
124. Gordon MO, Kass MA. The Ocular Hypertension Treatment Study: design and baseline description of the participants. Arch Ophthalmol 1999;117:573-83.
125. Lichter PR, Musch DC, Gillespie BW, et al. Interim clinical outcomes in the Collaborative Initial Glaucoma Treatment Study comparing initial treatment randomized to medications or surgery. Ophthalmology 2001;108:1943-53.
126. Migdal C, Gregory W, Hitchings R. Long-term functional outcome after early surgery compared with laser and medicine in open-angle glaucoma. Ophthalmology 1994;101:1651-6; discussion 7.
127. Jay JL, Murray SB. Early trabeculectomy versus conventional management in primary open angle glaucoma. Br J Ophthalmol 1988;72:881-9.
128. Whitacre MM, Stein R. Sources of error with use of Goldmann-type tonometers. Surv Ophthalmol 1993;38:1-30.
129. Barkana Y, Anis S, Liebmann J, et al. Clinical utility of intraocular pressure monitoring outside of normal office hours in patients with glaucoma. Arch Ophthalmol 2006;124:793-7.
130. Hasegawa K, Ishida K, Sawada A, et al. Diurnal variation of intraocular pressure in suspected normal-tension glaucoma. Jpn J Ophthalmol 2006;50:449-54.
131. Dinn RB, Zimmerman MB, Shuba LM, et al. Concordance of diurnal intraocular pressure between fellow eyes in primary open-angle glaucoma. Ophthalmology 2007;114:915-20.
132. Bagga H, Liu JH, Weinreb RN. Intraocular pressure measurements throughout the 24 h. Curr Opin Ophthalmol 2009;20:79-83.
133. Tasman W, Jaeger EA, eds. Duane's Ophthalmology, 15th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2009.
134. Quigley HA, Enger C, Katz J, et al. Risk factors for the development of glaucomatous visual field loss in ocular hypertension. Arch Ophthalmol 1994;112:644-9.
135. Sommer A, Katz J, Quigley HA, et al. Clinically detectable nerve fiber atrophy precedes the onset of glaucomatous field loss. Arch Ophthalmol 1991;109:77-83.
136. Lin SC, Singh K, Jampel HD, et al. Optic nerve head and retinal nerve fiber layer analysis: a report by the American Academy of Ophthalmology. Ophthalmology 2007;114:1937-49.

137. Jonas JB, Martus P, Horn FK, et al. Predictive factors of the optic nerve head for development or progression of glaucomatous visual field loss. Invest Ophthalmol Vis Sci 2004;45:2613-8.
138. Uchida H, Ugurlu S, Caprioli J. Increasing peripapillary atrophy is associated with progressive glaucoma. Ophthalmology 1998;105:1541-5.
139. Zeyen TG, Caprioli J. Progression of disc and field damage in early glaucoma. Arch Ophthalmol 1993;111:62-5.
140. Sommer A, Pollack I, Maumenee AE. Optic disc parameters and onset of glaucomatous field loss. I. Methods and progressive changes in disc morphology. Arch Ophthalmol 1979;97:1444-8.
141. Pederson JE, Anderson DR. The mode of progressive disc cupping in ocular hypertension and glaucoma. Arch Ophthalmol 1980;98:490-5.
142. Hart WM, Jr., Yablonski M, Kass MA, Becker B. Multivariate analysis of the risk of glaucomatous visual field loss. Arch Ophthalmol 1979;97:1455-8.
143. Yablonski ME, Zimmerman TJ, Kass MA, Becker B. Prognostic significance of optic disk cupping in ocular hypertensive patients. Am J Ophthalmol 1980;89:585-92.
144. Odberg T, Riise D. Early diagnosis of glaucoma. The value of successive stereophotography of the optic disc. Acta Ophthalmol (Copenh) 1985;63:257-63.
145. Drance S, Anderson DR, Schulzer M. Risk factors for progression of visual field abnormalities in normal-tension glaucoma. Am J Ophthalmol 2001;131:699-708.
146. Budenz DL, Anderson DR, Feuer WJ, et al. Detection and prognostic significance of optic disc hemorrhages during the Ocular Hypertension Treatment Study. Ophthalmology 2006;113:2137-43.
147. Miglior S, Torri V, Zeyen T, et al. Intercurrent factors associated with the development of open-angle glaucoma in the European Glaucoma Prevention Study. Am J Ophthalmol 2007;144:266-75.
148. De Moraes CG, Prata TS, Liebmann CA, et al. Spatially consistent, localized visual field loss before and after disc hemorrhage. Invest Ophthalmol Vis Sci 2009;50:4727-33.
149. Jeoung JW, Park KH, Kim JM, et al. Optic disc hemorrhage may be associated with retinal nerve fiber loss in otherwise normal eyes. Ophthalmology 2008;115:2132-40.
150. Quigley HA, Sommer A. How to use nerve fiber layer examination in the management of glaucoma. Trans Am Ophthalmol Soc 1987;85:254-72.
151. Agudelo LM, Molina CA, Alvarez DL. Changes in intraocular pressure after laser in situ keratomileusis for myopia, hyperopia, and astigmatism. J Refract Surg 2002;18:472-4.
152. Manni G, Oddone F, Parisi V, et al. Intraocular pressure and central corneal thickness. Prog Brain Res 2008;173:25-30.
153. Ehlers N, Hansen FK. Central corneal thickness in low-tension glaucoma. Acta Ophthalmol (Copenh) 1974;52:740-6.
154. Delgado MF, Nguyen NT, Cox TA, et al. Automated perimetry: a report by the American Academy of Ophthalmology. Ophthalmology 2002;109:2362-74.
155. Demirel S, Johnson CA. Incidence and prevalence of short wavelength automated perimetry deficits in ocular hypertensive patients. Am J Ophthalmol 2001;131:709-15.
156. Medeiros FA, Sample PA, Weinreb RN. Frequency doubling technology perimetry abnormalities as predictors of glaucomatous visual field loss. Am J Ophthalmol 2004;137:863-71.
157. Landers JA, Goldberg I, Graham SL. Detection of early visual field loss in glaucoma using frequency-doubling perimetry and short-wavelength automated perimetry. Arch Ophthalmol 2003;121:1705-10.
158. Singh K, Lee BL, Wilson MR. A panel assessment of glaucoma management: modification of existing RAND-like methodology for consensus in ophthalmology. Part II: results and interpretation. Am J Ophthalmol 2008;145:575-81.
159. Alencar LM, Bowd C, Weinreb RN, et al. Comparison of HRT-3 glaucoma probability score and subjective stereophotograph assessment for prediction of progression in glaucoma. Invest Ophthalmol Vis Sci 2008;49:1898-906.

160. Baraibar B, Sanchez-Cano A, Pablo LE, Honrubia FM. Preperimetric glaucoma assessment with scanning laser polarimetry (GDx VCC): analysis of retinal nerve fiber layer by sectors. J Glaucoma 2007;16:659-64.
161. Lalezary M, Medeiros FA, Weinreb RN, et al. Baseline optical coherence tomography predicts the development of glaucomatous change in glaucoma suspects. Am J Ophthalmol 2006;142:576-82.
162. Vizzeri G, Weinreb RN, Martinez de la Casa JM, et al. Clinicians agreement in establishing glaucomatous progression using the Heidelberg retina tomograph. Ophthalmology 2009;116:14-24.
163. Shaffer RN, Ridgway WL, Brown R, Kramer SG. The use of diagrams to record changes in glaucomatous disks. Am J Ophthalmol 1975;80:460-4.
164. Jampel HD, Friedman D, Quigley H, et al. Agreement among glaucoma specialists in assessing progressive disc changes from photographs in open-angle glaucoma patients. Am J Ophthalmol 2009;147:39-44.
165. Gaasterland DE, Blackwell B, Dally LG, et al, Advanced Glaucoma Intervention Study Investigators. The Advanced Glaucoma Intervention Study (AGIS): 10. Variability among academic glaucoma subspecialists in assessing optic disc notching. Trans Am Ophthalmol Soc 2001;99:177-84; discussion 184-5.
166. Medeiros FA, Zangwill LM, Bowd C, Weinreb RN. Comparison of the GDx VCC scanning laser polarimeter, HRT II confocal scanning laser ophthalmoscope, and stratus OCT optical coherence tomograph for the detection of glaucoma. Arch Ophthalmol 2004;122:827-37.
167. Chauhan BC, McCormick TA, Nicolela MT, LeBlanc RP. Optic disc and visual field changes in a prospective longitudinal study of patients with glaucoma: comparison of scanning laser tomography with conventional perimetry and optic disc photography. Arch Ophthalmol 2001;119:1492-9.
168. Wollstein G, Schuman JS, Price LL, et al. Optical coherence tomography longitudinal evaluation of retinal nerve fiber layer thickness in glaucoma. Arch Ophthalmol 2005;123:464-70.
169. Higginbotham EJ, Gordon MO, Beiser JA, et al. The Ocular Hypertension Treatment Study: topical medication delays or prevents primary open-angle glaucoma in African American individuals. Arch Ophthalmol 2004;122:813-20.
170. Fluorouracil Filtering Surgery Study Group. Five-year follow-up of the Fluorouracil Filtering Surgery Study. Am J Ophthalmol 1996;121:349-66.
171. Odberg T. Visual field prognosis in advanced glaucoma. Acta Ophthalmol 1987;65 (Suppl):27-9.
172. Mao LK, Stewart WC, Shields MB. Correlation between intraocular pressure control and progressive glaucomatous damage in primary open-angle glaucoma. Am J Ophthalmol 1991;111:51-5.
173. Kolker AE. Visual prognosis in advanced glaucoma: a comparison of medical and surgical therapy for retention of vision in 101 eyes with advanced glaucoma. Trans Am Ophthalmol Soc 1977;75:539-55.
174. Quigley HA, Maumenee AE. Long-term follow-up of treated open-angle glaucoma. Am J Ophthalmol 1979;87:519-25.
175. Greve EL, Dake CL, Klaver J, Mutsaerts E. Ten year prospective follow-up of a glaucoma operation. The double flap Scheie in primary open angle glaucoma. In: Greve EL, Leydhecker W, Raitta C, eds. Second European Glaucoma Symposium, Helsinki 1984. Dordrecht, Netherlands: Dr. W Junk, 1985.
176. Werner EB, Drance SM, Schulzer M. Trabeculectomy and the progression of glaucomatous visual field loss. Arch Ophthalmol 1977;95:1374-7.
177. Kidd MN, O'Connor M. Progression of field loss after trabeculectomy: a five-year follow-up. Br J Ophthalmol 1985;69:827-31.
178. Rollins D, Drance S. Five-year follow-up of trabeculectomy in the management of chronic open-angle glaucoma. In: New Orleans Acad Ophthalmol, Symposium on Glaucoma, 1981.
179. Chandler PA. Long-term results in glaucoma therapy. Am J Ophthalmol 1960;49:221-46.
180. Abedin S, Simmons RJ, Grant WM. Progressive low-tension glaucoma: treatment to stop glaucomatous cupping and field loss when these progress despite normal intraocular pressure. Ophthalmology 1982;89:1-6.

181. Fiscella RG, Green A, Patuszynski DH, Wilensky J. Medical therapy cost considerations for glaucoma. Am J Ophthalmol 2003;136:18-25.
182. Nelson P, Aspinall P, Papasouliotis O, et al. Quality of life in glaucoma and its relationship with visual function. J Glaucoma 2003;12:139-50.
183. Anderson DR. Glaucoma: the damage caused by pressure. XLVI Edward Jackson memorial lecture. Am J Ophthalmol 1989;108:485-95.
184. Jampel HD. Target pressure in glaucoma therapy. J Glaucoma 1997;6:133-8.
185. Musch DC, Gillespie BW, Lichter PR, et al. Visual field progression in the Collaborative Initial Glaucoma Treatment Study the impact of treatment and other baseline factors. Ophthalmology 2009;116:200-7.
186. Whitson JT. Glaucoma: a review of adjunctive therapy and new management strategies. Expert Opin Pharmacother 2007;8:3237-49.
187. McKinnon SJ, Goldberg LD, Peeples P, et al. Current management of glaucoma and the need for complete therapy. Am J Manag Care 2008;14:S20-7.
188. Stewart WC, Konstas AG, Nelson LA, Kruft B. Meta-analysis of 24-hour intraocular pressure studies evaluating the efficacy of glaucoma medicines. Ophthalmology 2008;115:1117-22.
189. Bhosle MJ, Reardon G, Camacho FT, et al. Medication adherence and health care costs with the introduction of latanoprost therapy for glaucoma in a Medicare managed care population. Am J Geriatr Pharmacother 2007;5:100-11.
190. van der Valk R, Webers CA, Schouten JS, et al. Intraocular pressure-lowering effects of all commonly used glaucoma drugs: a meta-analysis of randomized clinical trials. Ophthalmology 2005;112:1177-85.
191. Cheng JW, Cai JP, Wei RL. Meta-analysis of medical intervention for normal tension glaucoma. Ophthalmology 2009;116:1243-9.
192. Nordstrom BL, Friedman DS, Mozaffari E, et al. Persistence and adherence with topical glaucoma therapy. Am J Ophthalmol 2005;140:598-606.
193. Robin AL, Novack GD, Covert DW, et al. Adherence in glaucoma: objective measurements of once-daily and adjunctive medication use. Am J Ophthalmol 2007;144:533-40.
194. Robin AL, Covert D. Does adjunctive glaucoma therapy affect adherence to the initial primary therapy? Ophthalmology 2005;112:863-8.
195. Sleath B, Robin AL, Covert D, et al. Patient-reported behavior and problems in using glaucoma medications. Ophthalmology 2006;113:431-6.
196. Tsai JC. A comprehensive perspective on patient adherence to topical glaucoma therapy. Ophthalmology 2009;116:S30-6.
197. Piltz J, Gross R, Shin DH, et al. Contralateral effect of topical beta-adrenergic antagonists in initial one-eyed trials in the ocular hypertension treatment study. Am J Ophthalmol 2000;130:441-53.
198. Realini T, Fechtner RD, Atreides SP, Gollance S. The uniocular drug trial and second-eye response to glaucoma medications. Ophthalmology 2004;111:421-6.
199. Khouri AS, Realini T, Fechtner RD. Use of fixed-dose combination drugs for the treatment of glaucoma. Drugs Aging 2007;24:1007-16.
200. Fung AT, Reid SE, Jones MP, et al. Meta-analysis of randomised controlled trials comparing latanoprost with brimonidine in the treatment of open-angle glaucoma, ocular hypertension or normal-tension glaucoma. Br J Ophthalmol 2007;91:62-8.
201. Zimmerman TJ, Kooner KS, Kandarakis AS, Ziegler LP. Improving the therapeutic index of topically applied ocular drugs. Arch Ophthalmol 1984;102:551-3.
202. Friedman DS, Quigley HA, Gelb L, et al. Using pharmacy claims data to study adherence to glaucoma medications: methodology and findings of the Glaucoma Adherence and Persistency Study (GAPS). Invest Ophthalmol Vis Sci 2007;48:5052-7.
203. Schwartz GF, Reardon G, Mozaffari E. Persistency with latanoprost or timolol in primary open-angle glaucoma suspects. Am J Ophthalmol 2004;137:S13-6.

204. Okeke CO, Quigley HA, Jampel HD, et al. Adherence with topical glaucoma medication monitored electronically the Travatan Dosing Aid study. Ophthalmology 2009;116:191-9.
205. Stone JL, Robin AL, Novack GD, et al. An objective evaluation of eyedrop instillation in patients with glaucoma. Arch Ophthalmol 2009;127:732-6.
206. Aptel F, Masset H, Burillon C, et al. The influence of disease severity on quality of eye-drop administration in patients with glaucoma or ocular hypertension. Br J Ophthalmol 2009;93:700-1.
207. Haynes R, McDonald H, Garg A, Montague P. Interventions for helping patients to follow prescriptions for medications. Cochrane Database of Syst Rev 2002, Issue 2. Art. No.: CD000011. DOI: 10.1002/14651858.CD000011.
208. Osterberg L, Blaschke T. Adherence to medication. N Engl J Med 2005;353:487-97.
209. McIlraith I, Strasfeld M, Colev G, Hutnik CM. Selective laser trabeculoplasty as initial and adjunctive treatment for open-angle glaucoma. J Glaucoma 2006;15:124-30.
210. Spaeth GL, Baez KA. Argon laser trabeculoplasty controls one third of cases of progressive, uncontrolled, open angle glaucoma for 5 years. Arch Ophthalmol 1992;110:491-4.
211. Schwartz AL, Love DC, Schwartz MA. Long-term follow-up of argon laser trabeculoplasty for uncontrolled open-angle glaucoma. Arch Ophthalmol 1985;103:1482-4.
212. Krupin T, Patkin R, Kurata FK, et al. Argon laser trabeculoplasty in black and white patients with primary open-angle glaucoma. Ophthalmology 1986;93:811-6.
213. Shingleton BJ, Richter CU, Dharma SK, et al. Long-term efficacy of argon laser trabeculoplasty. A 10-year follow-up study. Ophthalmology 1993;100:1324-9.
214. Starita RJ, Fellman RL, Spaeth GL, Poryzees E. The effect of repeating full-circumference argon laser trabeculoplasty. Ophthalmic Surg 1984;15:41-3.
215. Brown SV, Thomas JV, Simmons RJ. Laser trabeculoplasty re-treatment. Am J Ophthalmol 1985;99:8-10.
216. Richter CU, Shingleton BJ, Bellows AR, et al. Retreatment with argon laser trabeculoplasty. Ophthalmology 1987;94:1085-9.
217. Weber PA, Burton GD, Epitropoulos AT. Laser trabeculoplasty retreatment. Ophthalmic Surg 1989;20:702-6.
218. Feldman RM, Katz LJ, Spaeth GL, et al. Long-term efficacy of repeat argon laser trabeculoplasty. Ophthalmology 1991;98:1061-5.
219. Jorizzo PA, Samples JR, Van Buskirk EM. The effect of repeat argon laser trabeculoplasty. Am J Ophthalmol 1988;106:682-5.
220. Reynolds AC, Skuta GL. Current Trends and Challenges in Glaucoma Care. Focal Points: Clinical Modules for Ophthalmologists. Module 6. San Francisco, CA: American Academy of Ophthalmology; 2008:2.
221. Latina MA, Park C. Selective targeting of trabecular meshwork cells: in vitro studies of pulsed and CW laser interactions. Exp Eye Res 1995;60:359-71.
222. Kramer TR, Noecker RJ. Comparison of the morphologic changes after selective laser trabeculoplasty and argon laser trabeculoplasty in human eye bank eyes. Ophthalmology 2001;108:773-9.
223. Russo V, Barone A, Cosma A, et al. Selective laser trabeculoplasty versus argon laser trabeculoplasty in patients with uncontrolled open-angle glaucoma. Eur J Ophthalmol 2009;19:429-34.
224. Damji KF, Shah KC, Rock WJ, et al. Selective laser trabeculoplasty v argon laser trabeculoplasty: a prospective randomised clinical trial. Br J Ophthalmol 1999;83:718-22.
225. Popiela G, Muzyka M, Szelepin L, et al. Use of YAG-Selecta laser and argon laser in the treatment of open angle glaucoma [in Polish]. Klin Oczna 2000;102:129-33.
226. Martinez-de-la-Casa JM, Garcia-Feijoo J, Castillo A, et al. Selective vs argon laser trabeculoplasty: hypotensive efficacy, anterior chamber inflammation, and postoperative pain. Eye (Lond) 2004;18:498-502.

227. Damji KF, Bovell AM, Hodge WG, et al. Selective laser trabeculoplasty versus argon laser trabeculoplasty: results from a 1-year randomised clinical trial. Br J Ophthalmol 2006;90:1490-4.
228. Best UP, Domack H, Schmidt V. Pressure reduction after selective laser trabeculoplasty with two different laser systems and after argon laser trabeculoplasty--a controlled prospective clinical trial on 284 eyes [in German]. Klin Monatsbl Augenheilkd 2007;224:173-9.
229. Juzych MS, Chopra V, Banitt MR, et al. Comparison of long-term outcomes of selective laser trabeculoplasty versus argon laser trabeculoplasty in open-angle glaucoma. Ophthalmology 2004;111:1853-9.
230. Van de Veire S, Zeyen T, Stalmans I. Argon versus selective laser trabeculoplasty. Bull Soc Belge Ophtalmol 2006;299:5-10.
231. Nagar M, Ogunyomade A, O'Brart DP, et al. A randomised, prospective study comparing selective laser trabeculoplasty with latanoprost for the control of intraocular pressure in ocular hypertension and open angle glaucoma. Br J Ophthalmol 2005;89:1413-7.
232. Hong BK, Winer JC, Martone JF, et al. Repeat selective laser trabeculoplasty. J Glaucoma 2009;18:180-3.
233. Latina MA, Sibayan SA, Shin DH, et al. Q-switched 532-nm Nd:YAG laser trabeculoplasty (selective laser trabeculoplasty): a multicenter, pilot, clinical study. Ophthalmology 1998;105:2082-8; discussion 2089-90.
234. American Academy of Ophthalmology. Policy Statement. Pretreatment Assessment: Responsibilities of the Ophthalmologist. San Francisco, CA: American Academy of Ophthalmology; 2006. Available at: http://one.aao.org/CE/PracticeGuidelines/ClinicalStatements.aspx.
235. American Academy of Ophthalmology. Policy Statement. An Ophthalmologist's Duties Concerning Postoperative Care. San Francisco, CA: American Academy of Ophthalmology; 2006. Available at: http://one.aao.org/CE/PracticeGuidelines/ClinicalStatements.aspx.
236. Robin AL. Argon laser trabeculoplasty medical therapy to prevent the intraocular pressure rise associated with argon laser trabeculoplasty. Ophthalmic Surg 1991;22:31-7.
237. Wickham MG, Worthen DM. Argon laser trabeculotomy:long-term follow-up. Ophthalmology 1979;86:495-503.
238. Wise JB, Witter SL. Argon laser therapy for open-angle glaucoma. A pilot study. Arch Ophthalmol 1979;97:319-22.
239. Schwartz AL, Whitten ME, Bleiman B, Martin D. Argon laser trabecular surgery in uncontrolled phakic open angle glaucoma. Ophthalmology 1981;88:203-12.
240. Holmwood PC, Chase RD, Krupin T, et al. Apraclonidine and argon laser trabeculoplasty. Am J Ophthalmol 1992;114:19-22.
241. Robin A, Pollack I, House B, Enger C. Effects of ALO 2145 on intraocular pressure following argon laser trabeculectomy. Arch Ophtalmol 1987;105:646-50.
242. Parrish RK II, Feuer WJ, Schiffman JC, et al. Five-year follow-up optic disc findings of the Collaborative Initial Glaucoma Treatment Study. Am J Ophthalmol 2009;147:717-24.
243. Law SK, Modjtahedi SP, Mansury A, Caprioli J. Intermediate-term comparison of trabeculectomy with intraoperative mitomycin-C between Asian American and Caucasian glaucoma patients: a case-controlled comparison. Eye (Lond) 2007;21:71-8.
244. Kim HY, Egbert PR, Singh K. Long-term comparison of primary trabeculectomy with 5-fluorouracil versus mitomycin C in West Africa. J Glaucoma 2008;17:578-83.
245. Wong TT, Khaw PT, Aung T, et al. The Singapore 5-fluorouracil trabeculectomy study: effects on intraocular pressure control and disease progression at 3 years. Ophthalmology 2009;116:175-84.
246. Heuer DK, Gressel MG, Parrish RK II, et al. Trabeculectomy in aphakic eyes. Ophthalmology 1984;91:1045-51.
247. Gross RL, Feldman RM, Spaeth GL, et al. Surgical therapy of chronic glaucoma in aphakia and pseudophakia. Ophthalmology 1988;95:1195-201.
248. Shirato S, Kitazawa Y, Mishima S. A critical analysis of the trabeculectomy results by a prospective follow-up design. Jpn J Ophthalmol 1982;26:468-80.

249. Law SK, Shih K, Tran DH, et al. Long-term outcomes of repeat vs initial trabeculectomy in open-angle glaucoma. Am J Ophthalmol 2009;148:685-95.
250. AGIS Investigators. The Advanced Glaucoma Intervention Study: 8. Risk of cataract formation after trabeculectomy. Arch Ophthalmol 2001;119:1771-9.
251. Hylton C, Congdon N, Friedman D, et al. Cataract after glaucoma filtration surgery. Am J Ophthalmol 2003;135:231-2.
252. Andreanos D, Georgopoulos GT, Vergados J, et al. Clinical evaluation of the effect of mitomycin-C in re-operation for primary open angle glaucoma. Eur J Ophthalmol 1997;7:49-54.
253. Wilkins M, Indar A, Wormald R. Intra-operative Mitomycin C for glaucoma surgery. Cochrane Database of Syst Rev 2005, Issue 1. Art. No.: CD002897. DOI: 10.1002/14651858.CD002897.pub2. 2005.
254. Robin AL, Ramakrishnan R, Krishnadas R, et al. A long-term dose-response study of mitomycin in glaucoma filtration surgery. Arch Ophthalmol 1997;115:969-74.
255. Costa VP, Comegno PE, Vasconcelos JP, et al. Low-dose mitomycin C trabeculectomy in patients with advanced glaucoma. J Glaucoma 1996;5:193-9.
256. Martini E, Laffi GL, Sprovieri C, Scorolli L. Low-dosage mitomycin C as an adjunct to trabeculectomy. A prospective controlled study. Eur J Ophthalmol 1997;7:40-8.
257. WuDunn D, Cantor LB, Palanca-Capistrano AM, et al. A prospective randomized trial comparing intraoperative 5-fluorouracil vs mitomycin C in primary trabeculectomy. Am J Ophthalmol 2002;134:521-8.
258. Singh K, Mehta K, Shaikh N. Trabeculectomy with intraoperative mitomycin C versus 5-fluorouracil. Prospective randomized clinical trial. Ophthalmology 2000;107:2305-9.
259. Leyland M, Bloom P, Zinicola E, et al. Single intraoperative application of 5-Fluorouracil versus placebo in low-risk trabeculectomy surgery: a randomized trial. J Glaucoma 2001;10:452-7.
260. Ruderman JM, Welch DB, Smith MF, Shoch DE. A randomized study of 5-fluorouracil and filtration surgery. Am J Ophthalmol 1987;104:218-24.
261. Fluorouracil Filtering Surgery Study Group. Fluorouracil Filtering Surgery Study one-year follow-up. Am J Ophthalmol 1989;108:625-35.
262. Wormald R, Wilkins MR, Bunce C. Postoperative 5-Fluorouracil for glaucoma surgery. Cochrane Database of Syst Rev 2008, Issue 4. Art. No.: CD001132. DOI: 10.1002/14651858.CD001132.
263. Goldenfeld M, Krupin T, Ruderman JM, et al. 5-Fluorouracil in initial trabeculectomy. A prospective, randomized, multicenter study. Ophthalmology 1994;101:1024-9.
264. Ophir A, Ticho U. A randomized study of trabeculectomy and subconjunctival administration of fluorouracil in primary glaucomas. Arch Ophthalmol 1992;110:1072-5.
265. Costa VP, Wilson RP, Moster MR, et al. Hypotony maculopathy following the use of topical mitomycin C in glaucoma filtration surgery. Ophthalmic Surg 1993;24:389-94.
266. Zacharia PT, Deppermann SR, Schuman JS. Ocular hypotony after trabeculectomy with mitomycin C. Am J Ophthalmol 1993;116:314-26.
267. Kupin TH, Juzych MS, Shin DH, et al. Adjunctive mitomycin C in primary trabeculectomy in phakic eyes. Am J Ophthalmol 1995;119:30-9.
268. Greenfield DS, Liebmann JM, Jee J, Ritch R. Late-onset bleb leaks after glaucoma filtering surgery. Arch Ophthalmol 1998;116:443-7.
269. Soltau JB, Rothman RF, Budenz DL, et al. Risk factors for glaucoma filtering bleb infections. Arch Ophthalmol 2000;118:338-42.
270. Jampel HD, Quigley HA, Kerrigan-Baumrind LA, et al. Risk factors for late-onset infection following glaucoma filtration surgery. Arch Ophthalmol 2001;119:1001-8.
271. Whiteside-Michel J, Liebmann JM, Ritch R. Initial 5-fluorouracil trabeculectomy in young patients. Ophthalmology 1992;99:7-13.
272. Suner IJ, Greenfield DS, Miller MP, et al. Hypotony maculopathy after filtering surgery with mitomycin C. Incidence and treatment. Ophthalmology 1997;104:207-14; discussion 14-5.

273. Scott DR, Quigley HA. Medical management of a high bleb phase after trabeculectomies. Ophthalmology 1988;95:1169-73.
274. Ramulu PY, Corcoran KJ, Corcoran SL, Robin AL. Utilization of various glaucoma surgeries and procedures in Medicare beneficiaries from 1995 to 2004. Ophthalmology 2007;114:2265-70.
275. Chen PP, Yamamoto T, Sawada A, et al. Use of antifibrosis agents and glaucoma drainage devices in the American and Japanese Glaucoma Societies. J Glaucoma 1997;6:192-6.
276. Joshi AB, Parrish RK II, Feuer WF. 2002 survey of the American Glaucoma Society: practice preferences for glaucoma surgery and antifibrotic use. J Glaucoma 2005;14:172-4.
277. Minckler DS, Francis BA, Hodapp EA, et al. Aqueous shunts in glaucoma: a report by the American Academy of Ophthalmology. Ophthalmology 2008;115:1089-98.
278. Minckler D, Vedula SS, Li T, et al. Aqueous shunts for glaucoma. Cochrane Database of Systematic Reviews 2006, Issue 2. Art. No.: CD004918. DOI: 10.1002/14651858.CD004918.pub2.
279. Bluestein EC, Stewart WC. Trabeculectomy with 5-fluorouracil vs single-plate Molteno implantation. Ophthalmic Surg 1993;24:669-73.
280. Tran DH, Souza C, Ang MJ, et al. Comparison of long-term surgical success of Ahmed Valve implant versus trabeculectomy in open-angle glaucoma. Br J Ophthalmol 2009;93:1504-9.
281. Wilson MR, Mendis U, Paliwal A, Haynatzka V. Long-term follow-up of primary glaucoma surgery with Ahmed glaucoma valve implant versus trabeculectomy. Am J Ophthalmol 2003;136:464-70.
282. Gedde SJ, Schiffman JC, Feuer WJ, et al, Tube Versus Trabeculectomy Study Group. Three-year follow-up of the Tube Versus Trabeculectomy Study. Am J Ophthalmol 2009;148:670-84.
283. Jampel HD, Friedman DS, Lubomski LH, et al. Effect of technique on intraocular pressure after combined cataract and glaucoma surgery: An evidence-based review. Ophthalmology 2002;109:2215-24.
284. Buys YM, Chipman ML, Zack B, et al. Prospective randomized comparison of one- versus two-site Phacotrabeculectomy two-year results. Ophthalmology 2008;115:1130-3.
285. Cotran PR, Roh S, McGwin G. Randomized comparison of 1-Site and 2-Site phacotrabeculectomy with 3-year follow-up. Ophthalmology 2008;115:447-54.
286. Gdih GA, Yuen D, Yan P, et al. Meta-analysis of 1- versus 2-site phacotrabeculectomy. Ophthalmology 2010. In press.
287. Chung AN, Aung T, Wang JC, Chew PT. Surgical outcomes of combined phacoemulsification and glaucoma drainage implant surgery for Asian patients with refractory glaucoma with cataract. Am J Ophthalmol 2004;137:294-300.
288. Hoffman KB, Feldman RM, Budenz DL, et al. Combined cataract extraction and Baerveldt glaucoma drainage implant: indications and outcomes. Ophthalmology 2002;109:1916-20.
289. Nassiri N, Sadeghi Yarandi S, Mohammadi B, Rahmani L. Combined phacoemulsification and Ahmed valve glaucoma drainage implant: a retrospective case series. Eur J Ophthalmol 2008;18:191-8.
290. Carassa RG, Bettin P, Fiori M, Brancato R. Viscocanalostomy versus trabeculectomy in white adults affected by open-angle glaucoma: a 2-year randomized, controlled trial. Ophthalmology 2003;110:882-7.
291. Kobayashi H, Kobayashi K, Okinami S. A comparison of the intraocular pressure-lowering effect and safety of viscocanalostomy and trabeculectomy with mitomycin C in bilateral open-angle glaucoma. Graefes Arch Clin Exp Ophthalmol 2003;241:359-66.
292. Luke C, Dietlein TS, Jacobi PC, et al. A prospective randomized trial of viscocanalostomy versus trabeculectomy in open-angle glaucoma: a 1-year follow-up study. J Glaucoma 2002;11:294-9.
293. O'Brart DP, Rowlands E, Islam N, Noury AM. A randomised, prospective study comparing trabeculectomy augmented with antimetabolites with a viscocanalostomy technique for the management of open angle glaucoma uncontrolled by medical therapy. Br J Ophthalmol 2002;86:748-54.
294. Jonescu-Cuypers C, Jacobi P, Konen W, Krieglstein G. Primary viscocanalostomy versus trabeculectomy in white patients with open-angle glaucoma: a randomized clinical trial. Ophthalmology 2001;108:254-8.

295. Gilmour DF, Manners TD, Devonport H, et al. Viscocanalostomy versus trabeculectomy for primary open angle glaucoma: 4-year prospective randomized clinical trial. Eye (Lond) 2009;23:1802-7.
296. Wishart MS, Dagres E. Seven-year follow-up of combined cataract extraction and viscocanalostomy. J Cataract Refract Surg 2006;32:2043-9.
297. Park M, Hayashi K, Takahashi H, et al. Phaco-viscocanalostomy versus phaco-trabeculotomy: a middle-term study. J Glaucoma 2006;15:456-61.
298. Yalvac IS, Sahin M, Eksioglu U, et al. Primary viscocanalostomy versus trabeculectomy for primary open-angle glaucoma: three-year prospective randomized clinical trial. J Cataract Refract Surg 2004;30:2050-7.
299. Chiselita D. Non-penetrating deep sclerectomy versus trabeculectomy in primary open-angle glaucoma surgery. Eye (Lond) 2001;15:197-201.
300. El Sayyad F, Helal M, El-Kholify H, et al. Nonpenetrating deep sclerectomy versus trabeculectomy in bilateral primary open-angle glaucoma. Ophthalmology 2000;107:1671-4.
301. Ambresin A, Shaarawy T, Mermoud A. Deep sclerectomy with collagen implant in one eye compared with trabeculectomy in the other eye of the same patient. J Glaucoma 2002;11:214-20.
302. Mermoud A, Schnyder CC, Sickenberg M, et al. Comparison of deep sclerectomy with collagen implant and trabeculectomy in open-angle glaucoma. J Cataract Refract Surg 1999;25:323-31.
303. Cillino S, Di Pace F, Casuccio A, et al. Deep sclerectomy versus punch trabeculectomy with or without phacoemulsification: a randomized clinical trial. J Glaucoma 2004;13:500-6.
304. Francis BA, Lin SC, Singh K, et al. Novel glaucoma procedures: a report by the American Academy of Ophthalmology. Ophthalmology. In press.
305. Lewis RA, von Wolff K, Tetz M, et al. Canaloplasty: circumferential viscodilation and tensioning of Schlemm canal using a flexible microcatheter for the treatment of open-angle glaucoma in adults: two-year interim clinical study results. J Cataract Refract Surg 2009;35:814-24.
306. Minckler D, Mosaed S, Dustin L, Francis BF, Trabectome Study Group. Trabectome (trabeculectomy-internal approach): additional experience and extended follow-up. Trans Am Ophthalmol Soc 2008;106:149-59; discussion 159-60.
307. Minckler D, Baerveldt G, Ramirez MA, et al. Clinical results with the Trabectome, a novel surgical device for treatment of open-angle glaucoma. Trans Am Ophthalmol Soc 2006;104:40-50.
308. Spiegel D, Wetzel W, Neuhann T, et al. Coexistent primary open-angle glaucoma and cataract: interim analysis of a trabecular micro-bypass stent and concurrent cataract surgery. Eur J Ophthalmol 2009;19:393-9.
309. Jonsen A, Siegler M, Winslade W. Clinical Ethics: a practical approach to ethical decisions in clinical medicine. 3rd ed. Summit, PA: McGraw-Hill, Inc., Health Professions Division, 1992; 40-3.
310. Roth SM, Spaeth GL, Starita RJ, et al. The effects of postoperative corticosteroids on trabeculectomy and the clinical course of glaucoma: five-year follow-up study. Ophthalmic Surg 1991;22:724-9.
311. Starita RJ, Fellman RL, Spaeth GL, et al. Short- and long-term effects of postoperative corticosteroids on trabeculectomy. Ophthalmology 1985;92:938-46.
312. Stewart WC, Shields MB. Management of anterior chamber depth after trabeculectomy. Am J Ophthalmol 1988;106:41-4.
313. Fiore PM, Richter CU, Arzeno G, et al. The effect of anterior chamber depth on endothelial cell count after filtration surgery. Arch Ophthalmol 1989;107:1609-11.
314. Phillips CI, Clark CV, Levy AM. Posterior synechiae after glaucoma operations: aggravation by shallow anterior chamber and pilocarpine. Br J Ophthalmol 1987;71:428-32.
315. Brubaker RF, Pederson JE. Ciliochoroidal detachment. Surv Ophthalmol 1983;27:281-9.
316. Gressel MG, Parrish RK II, Heuer DK. Delayed nonexpulsive suprachoroidal hemorrhage. Arch Ophthalmol 1984;102:1757-60.
317. Ruderman JM, Harbin TS Jr, Campbell DG. Postoperative suprachoroidal hemorrhage following filtration procedures. Arch Ophthalmol 1986;104:201-5.

318. Radhakrishnan S, Quigley HA, Jampel HD, et al. Outcomes of surgical bleb revision for complications of trabeculectomy. Ophthalmology 2009;116:1713-8.
319. Kapasi MS, Birt CM. The efficacy of 5-fluorouracil bleb needling performed 1 year or more posttrabeculectomy: a retrospective study. J Glaucoma 2009;18:144-8.
320. Anand N, Khan A. Long-term outcomes of needle revision of trabeculectomy blebs with mitomycin C and 5-fluorouracil: a comparative safety and efficacy report. J Glaucoma 2009;18:513-20.
321. Sharan S, Trope GE, Chipman M, Buys YM. Late-onset bleb infections: prevalence and risk factors. Can J Ophthalmol 2009;44:279-83.
322. Pastor SA, Singh K, Lee DA, et al. Cyclophotocoagulation: a report by the American Academy of Ophthalmology. Ophthalmology 2001;108:2130-8.
323. Lin SC. Endoscopic and transscleral cyclophotocoagulation for the treatment of refractory glaucoma. J Glaucoma 2008;17:238-47.
324. Kosoko O, Gaasterland DE, Pollack IP, Enger CL, Diode Laser Ciliary Ablation Study Group. Long-term outcome of initial ciliary ablation with contact diode laser transscleral cyclophotocoagulation for severe glaucoma. . Ophthalmology 1996;103:1294-302.
325. Youn J, Cox TA, Allingham RR, Shields MB. Factors associated with visual acuity loss after noncontact transscleral Nd:YAG cyclophotocoagulation. J Glaucoma 1996;5:390-4.
326. Fankhauser F, Kwasniewska S, Van der Zypen E. Cyclodestructive procedures. I. Clinical and morphological aspects: a review. Ophthalmologica 2004;218:77-95.
327. Bechrakis NE, Muller-Stolzenburg NW, Helbig H, Foerster MH. Sympathetic ophthalmia following laser cyclocoagulation. Arch Ophthalmol 1994;112:80-4.
328. Bloom PA, Tsai JC, Sharma K, et al. "Cyclodiode". Trans-scleral diode laser cyclophotocoagulation in the treatment of advanced refractory glaucoma. Ophthalmology 1997;104:1508-19; discussion 1519-20.
329. Reynolds AC, Skuta GL. Current Trends and Challenges in Glaucoma Care. Focal Points: Clinical Modules for Ophthalmologists. Module 6. San Francisco, CA: American Academy of Ophthalmology; 2008:4.
330. Chen J, Cohn RA, Lin SC, et al. Endoscopic photocoagulation of the ciliary body for treatment of refractory glaucomas. Am J Ophthalmol 1997;124:787-96.
331. Kahook MY, Lathrop KL, Noecker RJ. One-site versus two-site endoscopic cyclophotocoagulation. J Glaucoma 2007;16:527-30.
332. Murthy GJ, Murthy PR, Murthy KR, Kulkarni VV. A study of the efficacy of endoscopic cyclophotocoagulation for the treatment of refractory glaucomas. Indian J Ophthalmol 2009;57:127-32.
333. Ahmad S, Wallace DJ, Herndon LW. Phthisis after endoscopic cyclophotocoagulation. Ophthalmic Surg Lasers Imaging 2008;39:407-8.
334. Uram M. Ophthalmic laser microendoscope ciliary process ablation in the management of neovascular glaucoma. Ophthalmology 1992;99:1823-8.
335. Gayton JL, Van Der Karr M, Sanders V. Combined cataract and glaucoma surgery: trabeculectomy versus endoscopic laser cycloablation. J Cataract Refract Surg 1999;25:1214-9.
336. Lima FE, Magacho L, Carvalho DM, et al. A prospective, comparative study between endoscopic cyclophotocoagulation and the Ahmed drainage implant in refractory glaucoma. J Glaucoma 2004;13:233-7.
337. West AL, Oren GA, Moroi SE. Evidence for the use of nutritional supplements and herbal medicines in common eye diseases. Am J Ophthalmol 2006;141:157-66.
338. Gunasekera V, Ernst E, Ezra DG. Systematic internet-based review of complementary and alternative medicine for glaucoma. Ophthalmology 2008;115:435-9.
339. Coleman AL, Stone KL, Kodjebacheva G, et al. Glaucoma risk and the consumption of fruits and vegetables among older women in the study of osteoporotic fractures. Am J Ophthalmol 2008;145:1081-9.

340. American Academy of Ophthalmology Complementary Therapy Task Force. Complementary Therapy Assessment. Marijuana in the Treatment of Glaucoma. San Francisco, CA: American Academy of Ophthalmology; 2003. Available at: http://one.aao.org/CE/PracticeGuidelines/Therapy.aspx.
341. Herndon LW, Weizer JS, Stinnett SS. Central corneal thickness as a risk factor for advanced glaucoma damage. Arch Ophthalmol 2004;122:17-21.
342. Hjortdal JO, Moller-Pedersen T, Ivarsen A, Ehlers N. Corneal power, thickness, and stiffness: results of a prospective randomized controlled trial of PRK and LASIK for myopia. J Cataract Refract Surg 2005;31:21-9.
343. Caprioli J, Prum B, Zeyen T. Comparison of methods to evaluate the optic nerve head and nerve fiber layer for glaucomatous change. Am J Ophthalmol 1996;121:659-67.
344. Airaksinen PJ, Tuulonen A, Alanko HI. Rate and pattern of neuroretinal rim area decrease in ocular hypertension and glaucoma. Arch Ophthalmol 1992;110:206-10.
345. Smith SD, Katz J, Quigley HA. Analysis of progressive change in automated visual fields in glaucoma. Invest Ophthalmol Vis Sci 1996;37:1419-28.
346. Katz J, Tielsch JM, Quigley HA, Sommer A. Automated perimetry detects visual field loss before manual Goldmann perimetry. Ophthalmology 1995;102:21-6.
347. Heijl A, Asman P. A clinical study of perimetric probability maps. Arch Ophthalmol 1989;107:199-203.
348. Jay JL, Murdoch JR. The rate of visual field loss in untreated primary open angle glaucoma. Br J Ophthalmol 1993;77:176-8.
349. Chauhan BC, Mikelberg FS, Balaszi AG, et al. Canadian Glaucoma Study: 2. risk factors for the progression of open-angle glaucoma. Arch Ophthalmol 2008;126:1030-6.
350. Kwon YH, Kim YI, Pereira ML, et al. Rate of optic disc cup progression in treated primary open-angle glaucoma. J Glaucoma 2003;12:409-16.
351. Daugeliene L, Yamamoto T, Kitazawa Y. Risk factors for visual field damage progression in normal-tension glaucoma eyes. Graefes Arch Clin Exp Ophthalmol 1999;237:105-8.
352. Tezel G, Siegmund KD, Trinkaus K, et al. Clinical factors associated with progression of glaucomatous optic disc damage in treated patients. Arch Ophthalmol 2001;119:813-8.
353. Martus P, Stroux A, Budde WM, et al. Predictive factors for progressive optic nerve damage in various types of chronic open-angle glaucoma. Am J Ophthalmol 2005;139:999-1009.
354. Nouri-Mahdavi K, Hoffman D, Gaasterland D, Caprioli J. Prediction of visual field progression in glaucoma. Invest Ophthalmol Vis Sci 2004;45:4346-51.
355. Stewart WC, Kolker AE, Sharpe ED, et al. Factors associated with long-term progression or stability in primary open-angle glaucoma. Am J Ophthalmol 2000;130:274-9.
356. Brandt JD. Corneal thickness in glaucoma screening, diagnosis, and management. Curr Opin Ophthalmol 2004;15:85-9.
357. Jonas JB, Stroux A, Velten I, et al. Central corneal thickness correlated with glaucoma damage and rate of progression. Invest Ophthalmol Vis Sci 2005;46:1269-74.
358. Stewart WC, Day DG, Jenkins JN, et al. Mean intraocular pressure and progression based on corneal thickness in primary open-angle glaucoma. J Ocul Pharmacol Ther 2006;22:26-33.
359. Papadia M, Sofianos C, Iester M, et al. Corneal thickness and visual field damage in glaucoma patients. Eye (Lond) 2007;21:943-7.
360. Chauhan BC, Hutchison DM, LeBlanc RP, et al. Central corneal thickness and progression of the visual field and optic disc in glaucoma. Br J Ophthalmol 2005;89:1008-12.
361. Kim JW, Chen PP. Central corneal pachymetry and visual field progression in patients with open-angle glaucoma. Ophthalmology 2004;111:2126-32.
362. Kass MA, Kolker AE, Becker B. Prognostic factors in glaucomatous visual field loss. Arch Ophthalmol 1976;94:1274-6.
363. Chen PP. Correlation of visual field progression between eyes in patients with open-angle glaucoma. Ophthalmology 2002;109:2093-9.

364. Chen PP, Bhandari A. Fellow eye prognosis in patients with severe visual field loss in 1 eye from chronic open-angle glaucoma. Arch Ophthalmol 2000;118:473-8.
365. Fontana L, Armas R, Garway-Heath DF, et al. Clinical factors influencing the visual prognosis of the fellow eyes of normal tension glaucoma patients with unilateral field loss. Br J Ophthalmol 1999;83:1002-5.
366. Vogel R, Crick RP, Mills KB, et al. Effect of timolol versus pilocarpine on visual field progression in patients with primary open-angle glaucoma. Ophthalmology 1992;99:1505-11.
367. Stewart WC, Chorak RP, Hunt HH, Sethuraman G. Factors associated with visual loss in patients with advanced glaucomatous changes in the optic nerve head. Am J Ophthalmol 1993;116:176-81.
368. Coleman AL, Caprioli J. The logic behind target intraocular pressure. Am J Ophthalmol 2009;147:379-80.
369. Damji KF, Behki R, Wang L. Canadian perspectives in glaucoma management: setting target intraocular pressure range. Can J Ophthalmol 2003;38:189-97.
370. European Glaucoma Society. Terminology and Guidelines for Glaucoma. 3rd ed. Savona, Italy:Editrice Dogma S.r.l.; 2008:127. Available at: www.eugs.org/eng/EGS_guidelines.asp. Accessed May 28, 2010.
371. Centers for Medicare and Medicaid Services. Physician Quality Reporting Initiative. Available at: www.cms.hhs.gov/pqri/. Accessed September 16, 2010.
372. Lim MC, Shiba DR, Clark IJ, et al. Personality type of the glaucoma patient. J Glaucoma 2007;16:649-54.
373. Odberg T, Jakobsen JE, Hultgren SJ, Halseide R. The impact of glaucoma on the quality of life of patients in Norway. II. Patient response correlated to objective data. Acta Ophthalmol Scand 2001;79:121-4.
374. Lundmark PO, Trope GE, Shapiro CM, Flanagan JG. Depressive symptomatology in tertiary-care glaucoma patients. Can J Ophthalmol 2009;44:198-204.
375. Mabuchi F, Yoshimura K, Kashiwagi K, et al. High prevalence of anxiety and depression in patients with primary open-angle glaucoma. J Glaucoma 2008;17:552-7.
376. Skalicky S, Goldberg I. Depression and quality of life in patients with glaucoma: a cross-sectional analysis using the Geriatric Depression Scale-15, assessment of function related to vision, and the Glaucoma Quality of Life-15. J Glaucoma 2008;17:546-51.
377. Demailly P, Zoute C, Castro D. Personalities and chronic glaucoma [in French]. J Fr Ophtalmol 1989;12:595-601.
378. Cumurcu T, Cumurcu BE, Celikel FC, Etikan I. Depression and anxiety in patients with pseudoexfoliative glaucoma. Gen Hosp Psychiatry 2006;28:509-15.
379. Erb C, Batra A, Lietz A, et al. Psychological characteristics of patients with normal-tension glaucoma. Graefes Arch Clin Exp Ophthalmol 1999;237:753-7.
380. Sherwood MB, Garcia-Siekavizza A, Meltzer MI, et al. Glaucoma's impact on quality of life and its relation to clinical indicators. A pilot study. Ophthalmology 1998;105:561-6.
381. Cameron BD, Saffra NA, Strominger MB. Laser in situ keratomileusis-induced optic neuropathy. Ophthalmology 2001;108:660-5.
382. Lyle WA, Jin GJ, Jin Y. Interface fluid after laser in situ keratomileusis. J Refract Surg 2003;19:455-9.
383. Galal A, Artola A, Belda J, et al. Interface corneal edema secondary to steroid-induced elevation of intraocular pressure simulating diffuse lamellar keratitis. J Refract Surg 2006;22:441-7.
384. Hamilton DR, Manche EE, Rich LF, Maloney RK. Steroid-induced glaucoma after laser in situ keratomileusis associated with interface fluid. Ophthalmology 2002;109:659-65.
385. Teichman JC, Ahmed, II. Intraocular lens choices for patients with glaucoma. Curr Opin Ophthalmol 2010;21:135-43.
386. American Academy of Ophthalmology Vision Rehabilitation Committee. Preferred Practice Pattern® Guidelines. Vision Rehabilitation for Adults. San Francisco, CA: American Academy of Ophthalmology; 2007. Available at: www.aao.org/ppp.

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