Who Has Glaucoma? Definitions and Diagnosis

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With many questions still remaining in the field of glaucoma—who has it, who will progress, when to intervene—clear definitions lay the groundwork for effectively navigating the many clinical challenges.

Like cancer, glaucoma is not a single disease; rather, the term refers to a group of degenerative disorders of the optic nerve characterized by a particular constellation of structural and functional changes; key among them are excavation and enlargement of the optic cup, loss of the nerve fiber layer rim, and corresponding visual field deficit.

A concise medical definition or standard dictionary definition of glaucoma typically includes mention of intraocular pressure (IOP). This is understandable in light of the strong clinical association between glaucoma and IOP and the fact that, at present, IOP represents the sole therapeutic target of medical antiglaucoma therapy.

Glaucoma and IOP

That said, elevated IOP (one description of which is IOP greater than the 97.5 percentile for a specific population) is no longer necessary for a definition of glaucoma, nor is it required for the diagnosis, since it is not universally present among glaucoma patients. ("Elevated IOP" can also be defined as IOP over 21 mm Hg or as the IOP at which the optic nerve sustains damage.) In fact, depending on the population studied, between 30% and 80% of eyes with glaucomatous optic nerve damage have “normal” IOPs (ie, IOPs of 21 mm Hg or less).

The converse is also true: elevated IOP may be present, and quite often is present, without evidence of glaucomatous optic nerve damage. It is also true that some degree of ocular hypertension may be present without evidence of optic nerve damage or visual symptoms. Glaucoma risk estimation and when to treat at-risk patients is the subject of significant debate and ongoing research.

PRIMARY VS SECONDARY GLAUCOMAS

Optic nerve damage associated with glaucoma can be thought of as the common endpoint of a variety of pathophysiologic processes. Glaucoma categorization starts with the designation of primary or secondary. Primary glaucoma describes patients with optic nerve damage and visual field loss with no discernable cause; primary open-angle glaucoma (POAG) is a principal subtype of glaucoma in which the angle between the iris and cornea is open.

Among the non-POAG categories of glaucoma, elevated IOP is consistently present at least some times in the course of the disease. Primary angle-closure glaucoma is a subtype of glaucoma in which the cause of irido-corneal angle narrowing is unknown (Figure 1). Secondary glaucoma may
result from a long list of underlying causes, and may affect individuals of any age, from newborns to the elderly. Like primary forms, secondary open-angle glaucoma is associated with an open angle; and secondary angle-closure glaucoma with iridotrabecular contact. Any process that impedes drainage of aqueous fluid via the trabecular meshwork—including preceding ocular trauma, diabetes, pigmentary dispersion syndrome, pseudoxfoliation, and dozens of other causes—can cause secondary glaucoma.

RISK FACTORS

Multiple studies have attempted to identify risk factors for the development of primary glaucoma. Subanalyses of two large multicenter, prospective, randomized trials—the Ocular Hypertension Treatment Study (OHTS) and the European Glaucoma Prevention Study (EGPS)—looked specifically at patient characteristics and disease parameters associated with conversion from ocular hypertension to glaucoma.5,6 Pooled analysis of untreated patients with ocular hypertension enrolled in the two studies and followed for 5 years (N=1341) revealed that poorer outcomes were predicted by: older age, higher IOP, decreased central corneal thickness, increased vertical cup-to-disc ratio, and greater visual field index pattern standard deviation (PSD).6 In addition, low perfusion pressure was also identified to be a risk factor in the Barbados Eye Study.

Family history is also considered a significant risk factor for the development of POAG. Although some controversy exists, there seems to be a propensity to glaucoma among certain ethnic groups. In the US, individuals of African and Hispanic descent are at highest risk for POAG.7 Asian individuals have high rates of glaucoma, and represent 87% of patients with primary angle-closure glaucoma worldwide.8 Other proposed risk factors for POAG include diabetes mellitus, high blood pressure, and myopia.

GLAUCOMA SUSPECTS

A patient or eye considered “glaucoma suspect” has some features suggestive of glaucoma but does not meet the full diagnostic criteria.5 Such patients generally present in one of four ways: 1) elevated IOP and normal appearing optic nerve; 2) large cup-to-disc ratio and normal optic nerve fiber layer; 3) abnormal visual field but normal optic nerve disc; or 4) unusual appearing optic nerve with normal visual field and IOP.

This begs the question: does the eye that is suspicious for glaucoma indicate a pre-glaucomatous stage? And, if so, can early intervention prevent the development of glaucoma? These are some of the questions that OHTS and EGPS were designed to evaluate; however, while helpful, they have not put the issue entirely to rest. In the OHTS, development of glaucoma was reduced by 60% with the use of topical hypotensive medication. Patients with ocular hypertension (baseline IOP 24 to 32 mm Hg) treated with IOP-lowering medication developed POAG at a rate of 4.4% over 5 years compared to a rate of 9.5% among untreated patients in the control group.7 EGPS,

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KEY ISSUES IN GLAUCOMA MANAGEMENT — Issue 1

STATEMENT OF NEED
Glaucoma, a group of ocular diseases characterized by progressive damage to the optic nerve, is the second leading cause of blindness worldwide. It affects a significant and growing portion of the US population.1,2 As primary eye care providers, medical optometrists are well positioned to identify patients at risk and to diagnose, monitor, and treat glaucoma. However, given that the expanded scope of practice incorporating glaucoma treatment is relatively new, many optometrists lack confidence in their ability to treat this potentially blinding disease. In order to instill confidence and help optometrists make sound clinical judgments about the care of patients with glaucoma, Key Issues in Glaucoma Management will help optometrists better understand the various aspects and nuances of the disease, including our current understanding of glaucoma patients, help optometrists make sound clinical judgments about the care of patients with glaucoma, and confidence in medical optometrists.

REFERENCES

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a similarly designed trial, did not show statistically significant prevention of POAG with treatment.9

Unfortunately, we lack data regarding early intervention outcomes for the other groups suspicious for glaucoma, and it remains unclear whether treatment is warranted for those groups.

**SHOULD GLAUCOMA SUSPECTS BE TREATED?**

It is important to remember that most patients with identifiable risk factors do not develop glaucoma. This has been shown in prospective studies. As noted, in the OHTS 90% of untreated glaucoma suspect patients with IOP between 24 and 32 mm Hg remained glaucoma-free over the 5 years of follow-up.4 It might seem prudent, then, to limit preventive medical therapy and spare patients the burden of lifelong medication whenever possible.

But the stakes are too high to simply leave it at that. For those who do develop glaucoma, the burden of therapy—inconvenience, side effects, cost—likely pales in comparison to the vision-saving benefits they might derive. Clearly, the ability to predict who is at risk for glaucoma and thus who would benefit from intervention holds enormous value. To that end, based on data from large studies, tools for calculating individual risk have been developed to assist clinicians in recommending or not recommending pre-glaucoma therapy. It is generally recommended that individuals identified as having high risk should be considered for treatment, whereas individuals at low risk should be observed without treatment.

**RISK CALCULATORS**

Like other medical predictive models—such as those used to predict risk for heart disease or osteoporosis—glaucoma risk calculators attempt to enhance objectivity in clinical management by distilling multiple variables down to an actionable metric. A glaucoma risk calculator based upon findings from OHTS uses patient age, baseline IOP, central corneal thickness, PSD, vertical cup-to-disc ratio, and the presence of diabetes to generate an idea of patients’ 5-year risk.6 The algorithm developed at our institution is available online at: https://www.deverseye.org/grc/. Other calculators can be found at http://ohts.wustl.edu/risk/calculator.html and http://oil.wilmer.jhu.edu/risk/.

A separate risk calculator for the progression of visual field loss among patients being treated for glaucoma has also been developed and validated.

**TREATING GLAUCOMA**

For patients with a clear diagnosis of glaucoma, treatment is almost always indicated, assuming that the patient is otherwise capable of maintaining an acceptable quality of life. Aggressiveness of the treatment relates to a number of factors, including stage of glaucoma, age and health status of the patient, and the health and visual acuity of the fellow eye. For example, a young patient with early glaucoma would almost always be treated aggressively since s/he will most likely have to live with glaucoma for a long time.

A glaucoma patient with a shorter life expectancy, eg, a patient in his/her late 80s with mild glaucoma and significant heart disease, after a thorough discussion, might elect to be periodically monitored for progression without treatment. And, as a further example, a patient with very advanced glaucoma in one eye and good vision in their other eye might only elect to receive palliative treatment in the affected eye to keep it comfortable rather than aggressive IOP-lowering drugs.

**DIAGNOSTIC PROGRESS**

In many of its forms, glaucoma is a sly, often silent disease—by the time patients notice visual change, up to 90% of the optic nerve may be affected. Because glaucoma is progressive and irreversible, timely diagnosis is paramount to controlling it and preventing vision loss. Late diagnosis increases risk for vision loss and raises the overall costs of the disease.10

New means for detecting glaucoma help to prevent late diagnosis and its consequences. In the past, clinicians could only examine the optic nerve with a handheld ophthalmoscope and test the visual fields manually. Currently, laser-based devices allow for glaucomatous optic disc detection without dilation of the pupils. At my practice, we evaluate the optic nerve with ophthalmoscopy at every visit, and we perform stereoscopic disc photography at the initial visit or
when the optic disc has a hemorrhage or we suspect a change. Finally, we use objective structural testing (ocular coherence tomography and Heidelberg Retinal Topography) at least yearly to assess the optic disc and nerve fiber layer for progression. In addition to structural assessment, we use standard automated achromatic visual field testing to assess for visual field loss.

Primary care physicians should advise patients to have regular ophthalmic examinations, including testing for glaucoma, every 2 years for individuals over 40 years old and yearly for those over 60. Patients with glaucoma should be reminded to inform family members so that they can be vigilant for the disease and receive appropriate screening.

CONCLUSION

Glaucoma is a group of diseases that share a common pathophysiologic endpoint: optic nerve degeneration. Early identification and prevention are key to managing glaucoma. Increased physician awareness and more aggressive screening and monitoring with state-of-the-art equipment can be expected to improve outcomes. Overall, the objective is to treat high-risk suspects, monitor low-risk suspects and treat those with glaucoma.

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Aqueous Humor Dynamics: What IOP Means for the Clinician

JOHN J. McSOLEY, OD

Intraocular pressure is a causative risk factor for glaucoma and, at present, the only target of available glaucoma treatments. Although elevated IOP is no longer recognized as necessary for a diagnosis of glaucoma, it is a key element in the evaluation and management of the disease.

Normal intraocular pressure (IOP) is essential to maintaining the structural integrity and visual function of the eye: it ensures inflation of the globe and maintains the eye’s optical components in position. Underlying normal IOP is a delicate balance between aqueous humor inflow and resistance to its outflow. Altered aqueous dynamics, predominantly due to outflow impairment, is the cause of IOP elevation in glaucoma. An understanding of normal aqueous humor dynamics is a cornerstone of understanding both the pathogenesis and treatment of glaucoma.

AQUEOUS DYNAMICS AND IOP

Precursor to aqueous humor passes from the microvasculature of the ciliary body stroma, through the pigmented and nonpigmented epithelia of the ciliary processes, and then as aqueous humor into the posterior chamber. Aqueous humor forms at a typical rate of approximately 2 to 2.5 microliters per minute. Secretion of aqueous humor is a complicated physiological process that is influenced by many factors, including ionic and osmotic forces, enzymatic reactions, activation of various receptors, circadian rhythm, age, neuropeptides, and hormones. Additional information on this complicated process is available. 1-5

After secretion from the ciliary body into the posterior chamber, aqueous humor circulates through the pupillary space to fill the anterior chamber. From there, aqueous humor exits the eye via one of two routes in the anterior chamber angle: the conventional outflow pathway via the trabecular meshwork (which accounts for the bulk of aqueous drainage), or the uveoscleral pathway. In the trabecular pathway, aqueous humor drains through the trabecular meshwork into Schlemm’s canal and then out through collector channels to the blood vessels and lymphatics (Figure 1). In the uveoscleral pathway, aqueous humor exits the eye via the supraciliary and suprachoroidal spaces.

WHAT IS NORMAL?

It is important to remember that the IOP range we call “normal” (IOPs in the 10 to 21 mm Hg range) are derived from measurement of pressures in normal populations, which typically have mean IOPs close to 16 mm Hg (with 2 standard deviations extending about 5 mm Hg). 6 In reality, IOP is dynamic, differing considerably from individual to individual and, for any given individual, rising and falling in a diurnal cycle. A normal individual’s IOP may vary by as much as 4 mm Hg or more over the course of the day as aqueous humor production and outflow fluctuate. 7 In glaucoma patients, the diurnal IOP variation may be even greater, likely a reflection of the pathophysiologic mechanisms modulating aqueous humor dynamics and possible effects of treatment.

The variability of IOP is an important consideration when checking patients’ pressure. Multiple measurements, ideally taken at different times of day, may be necessary for detection of elevated IOP in potential glaucoma patients. Wide ranges of IOP variability may be associated with the clinical course of glaucoma or the result of missed medication. It is prudent to avoid treatment decisions based on a single isolated pressure reading.

IOP: RELATION TO GLAUCOMA

IOP has been historically linked to glaucoma, and values exceeding the statistically normal range were viewed as leading to glaucoma. Today, glaucoma is defined by the characteristic structural changes in the optic nerve and corresponding impairment of visual function. IOP is not included as a defining feature of glaucoma. Patients...
whose IOP exceeds the normal range but have no glaucomatous damage are considered ocular hypertensives.

Glaucomatous damage can be identified in patients whose IOP remains within the normal range. The development of glaucoma is related to an individual’s susceptibility to various risk factors, including IOP. Indeed, many patients with open-angle glaucoma have IOPs within the statistically normal range (<22 mm Hg at a single measurement).8–13

Although no longer included in the definition of glaucoma, IOP is a key component of glaucoma clinical management. First, IOP is a causative risk factor for the development and progression of glaucomatous damage.14–16 As the level of IOP increases, the prevalence of open-angle glaucoma increases.8,17 On the other hand, IOP reduction in known glaucoma patients decreases the risk of visual field loss.15–17

Most importantly, IOP remains the only glaucoma risk factor that can be effectively modified, even though many other potential factors—such as a patient’s genetic susceptibility to optic nerve injury, compromise of blood flow, alterations of the lamina cribrosa, and other potential contributors, some of which may not yet be identified—may also contribute to glaucomatous damage. In most cases, a target IOP provides a short term benchmark to evaluate treatment efficacy, and IOP measurement allows us to monitor response to treatment in an easy and straightforward manner. That said, the ultimate and most important measure of success is preservation of the patient’s vision.

**GLAUCOMA EVALUATION**

When a patient is identified as having ocular hypertension, further assessment is warranted. Typically, this involves measurement of central corneal thickness, gonioscopy, visual field testing, and examination of the optic nerve head, usually with the help of photography and imaging devices that allow additional qualitative and quantitative measures of the structure of the optic nerve and the retinal nerve fiber layer. These tests serve as diagnostic aids, help in staging, and provide baseline information for future follow-up comparison. In cases where the patient shows no signs of glaucomatous damage, the most important step is to determine the risk of progression to glaucoma. Risk assessment includes how elevated the baseline pressure is and the presence or absence of other factors that add to the risk of glaucoma (eg, age, family history, etc.).

**FIGURE 1** The trabecular meshwork conventional outflow pathway. Aqueous humor is produced by the ciliary body and it flows (dashed line shown with arrowheads) from the posterior chamber through the pupil into the anterior chamber. From there it flows out through the trabecular meshwork into the Schlemm’s canal and is subsequently absorbed into the episcleral veins via the collector channels. (From Goel M, Picciani RG, Lee RK, et al. Aqueous Humor Dynamics: A Review. Open Ophthalmol J. 2010;4:52–9.)

Visualizing the anterior chamber angle, where the trabecular meshwork is located, is an important aspect of the clinical examination for glaucoma. The cause of impaired aqueous outflow leading to IOP elevation varies by the subtype of glaucoma, and gonioscopy is an essential diagnostic tool for determining whether the angle is open or closed and for identification of other pathologies that may cause pressure elevation. The findings can not only provide helpful diagnostic clues but also have important treatment implications.

Based on the risk for progression, the patient should be monitored over time for any structural or functional impairment. If there is no evidence for glaucomatous damage and the pressure is moderate, it may be prudent to just follow up with that patient in 3 or 4 months. If at the next exam there are no changes in IOP or in the condition of the optic nerve, a more detailed examination, including visual field testing, can be scheduled for 6 months later. Once a baseline and track record have been established, and it is clear that the patient is not progressing, it is then reasonable to lengthen follow-up intervals. At the same time, it is important to educate the patient about why the tests are necessary and why ongoing follow-up will be necessary, probably for life.

**MANAGING IOP**

Current glaucoma therapies are aimed almost exclusively at lowering IOP, which can be done by reducing aqueous humor production or enhancing aqueous outflow. Beta blockers, topical carbonic anhydrase inhibitors, and the alpha agonists all lower IOP by reducing aqueous formation; while prostaglandin analogs, the most widely used class of glaucoma management agents, work by increasing aqueous outflow. When a second medication is added from a different class, the effects may be additive.

A critical aspect of glaucoma management is selecting the most appropriate IOP-lowering agent. In selecting an
agent (or agents), the best strategy is the one that is safest, most effective, and creates the least burden—including economic burden—to the patient. This explains how prostaglandin analogs became the most popular first-line therapy for glaucoma: they provide very effective IOP lowering, are generally well tolerated, and are taken only once each day. Contrast that with an older agent like pilocarpine, which although effective, requires frequent dosing and has multiple side effects. Today pilocarpine is rarely used to treat glaucoma.

For patients who cannot tolerate prostaglandins, the next option is typically a beta blocker. Topical beta blockers are well tolerated by most patients, but there are some caveats. Topical beta blockers can greatly aggravate some ailments, such as asthma and certain cardiovascular conditions, and should be avoided in patients with a history of those disorders. Additionally, a topical beta blocker to reduce IOP may be less effective than expected when administered to a patient who is already on treatment. If progression in patients with more advanced damage usually requires a more aggressive target—eg, up to 50% of their baseline IOP or to the low-normal range of IOP. Even when patients reach their short-term goals for IOP reduction, they still require ongoing, careful follow-up.

The term “maximally tolerated medical therapy” well encapsulates the notion that IOP-lowering therapies should be effective, safe, and well tolerated. This may vary from patient to patient or may change for an individual patient during the follow-up period. Laser trabeculectomy may be a good option at any point during the course of follow-up care. If despite having optimized medical and laser therapy, there is progression (or a high likelihood of progression) more aggressive measures should be sought. We are fortunate to practice at a time when there are several good surgical options available to our patients. Referral to surgeons skilled in these methods has an important role in visual preservation.

**TREATMENT GOALS**

The goal in treating glaucoma is to halt or slow disease progression and, to the degree possible, preserve the current level of vision. How much IOP lowering is required depends on several factors: the severity of disease, the rate of progression, and the baseline IOP. The extent of damage at diagnosis and the rapidity of progression are good indicators of a patient’s susceptibility to IOP-caused damage and, thus, indicate how low the target IOP should be set. In general, a patient with minimal damage over an extended period of time would be at less risk for further vision loss than someone who has extensive damage or one whose damage has evolved quickly.

An initial treatment goal of 25% to 30% reduction in IOP makes a reasonable target for initial therapy in patients with mild damage. Prevention of progression in patients with more advanced damage usually requires a more aggressive target—eg, up to 50% of their baseline IOP or to the low-normal range of IOP. Even when patients reach their short-term goals for IOP reduction, they still require ongoing, careful follow-up.

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1. Which of the following is true of glaucoma risk calculators?
   A. Their difficulty makes them of value only to glaucoma specialists
   B. None is currently available for clinical use
   C. They help apply results from large studies to individual patients
   D. They are meaningful to research study participants only

2. Which of the following classes of glaucoma agents reduces IOP by increasing aqueous outflow?
   A. Beta blockers
   B. Carbonic anhydrase inhibitors
   C. Alpha agonists
   D. Prostaglandin analogs

3. Which of the following is NOT a risk factor for the development of glaucoma?
   A. Ocular trauma
   B. Family history of glaucoma
   C. Thick central corneal pachymetry
   D. Large vertical cup-to-disc ratio

4. How much pressure reduction is typically recommended when initiating therapy in glaucoma patients with mild damage?
   A. 5% or more
   B. 10% or more
   C. 25% or more
   D. 65% or more

5. Early detection of glaucoma is important because:
   A. Glaucoma is reversible with treatment
   B. Newly available neuroprotective agents can halt disease progression
   C. Both A and B are true
   D. None of the above is true

6. Primary open angle glaucoma:
   A. Is glaucoma with no known cause
   B. Remains asymptomatic at every stage
   C. Does not affect Asians
   D. Is caused by angle closure

7. Which of the following is true about the relationship between IOP and glaucoma?
   A. A single measurement can give a true and complete picture of a patient’s IOP status
   B. Glaucmatous damage can occur in patients whose pressure is consistently found in the normal range
   C. The prevalence of open-angle glaucoma decreases as IOP increases
   D. IOP lowering is one of many therapeutic options for glaucoma

8. Which of the following changes in aqueous flow is primarily responsible for increasing IOP in open-angle glaucoma?
   A. Excessive aqueous production
   B. Increased outflow through the trabecular meshwork
   C. Decreased uveoscleral outflow
   D. Both A and C are correct

9. Which of the following can influence aqueous humor production?
   A. Age
   B. Circadian rhythm
   C. Hormones
   D. All of the above