

## Canadian perspectives in glaucoma management: setting target intraocular pressure range

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It has long been understood that elevated intraocular pressure (IOP) is an important risk factor for glaucoma. As far back as the middle of the 19th century, Von Graefe and Jaeger reported its association with characteristic nerve damage leading to blindness. Population studies have shown that elevated IOP may lead to an increase in the prevalence<sup>1,2</sup> and incidence<sup>3</sup> of glaucoma.

The higher the IOP, the greater the risk of optic nerve damage. The principal goal of current glaucoma treatment is to control IOP, the major modifiable risk factor. Palmberg<sup>4</sup> found, in his review of the Advanced Glaucoma Intervention Study<sup>5</sup> data, that in 95% of cases primary open-angle glaucoma (POAG) is pressure dependent and avoidable.

The objective of glaucoma therapy is the preservation of visual field and vision. The quality of life of the patient should be maintained or enhanced.

Guidelines for setting target IOP have been established through a series of cross-Canada workshops. The participants included many of the glaucoma specialists from across the country. These sessions

included an overview of the most recent randomized clinical trials in the area of glaucoma management and working sessions in which case studies were presented and treatment options discussed. Working guidelines were then developed by consensus over the series of workshops. This article highlights the evidence-based content of the regional workshops and the consensus guidelines developed.

### THE EVIDENCE

Recent evidence supports the concept of aggressively lowering IOP to help prevent glaucoma-related blindness. The Collaborative Normal-Tension Glaucoma Study,<sup>6</sup> the Advanced Glaucoma Intervention Study,<sup>5</sup> the Collaborative Initial Glaucoma Treatment Study<sup>7</sup> and the Ocular Hypertension Treatment Study<sup>8,9</sup> are all "gold standard" prospective randomized clinical trials that support this concept (Table 1).

The purpose of the Collaborative Normal-Tension Glaucoma Study<sup>6</sup> was to determine whether IOP plays a part in the pathogenesis of normal-tension glaucoma by examining whether the rate of progression is changed in eyes in which the IOP is substantially lowered. Of the 230 patients with normal-tension glaucoma initially enrolled in the study, 140 were randomly assigned to receive either restricted medical and surgical treatment (61 eyes) or no treatment (79 eyes). Randomization occurred at enrolment if the original visual field threatened fixation, or if and when documented disc or visual field damage occurred. Target IOP lowering of 30% was arbitrarily chosen for those in the treatment group. Only 12% of the treated eyes progressed to further disc damage or visual field loss, compared with 35% of the control eyes. The time to progression was significantly longer for the treated eyes than for the control eyes. Despite IOP lowering of 30%, some treated patients continued to show progression.

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**Table 1—Randomized controlled clinical trials of glaucoma treatment with published results\***

Study	Design	No. of patients	Length of follow-up, yr	Key findings
Collaborative Normal-Tension Glaucoma Study <sup>6</sup>	POAG in eyes with normal IOP: rate of progression, effect of IOP reduction on progression rate	230	5+	Lowering the IOP slows the rate of progression of visual field loss compared to untreated eyes.
Advanced Glaucoma Intervention Study <sup>5</sup>	POAG after medical treatment failure with no previous surgery: laser trabeculoplasty vs. trabeculectomy	591	4–7	African-American patients did better with laser trabeculoplasty as first surgery, whereas after 4+ years white Americans did better with trabeculectomy as first surgery. Lower IOP during follow-up after surgical intervention protects against further visual field deterioration in patients with moderate to advanced disease.
Collaborative Initial Glaucoma Treatment Study <sup>7</sup>	Newly diagnosed POAG: medicine vs. trabeculectomy	607	5+	Aggressive initial medical IOP management was as effective as early surgical therapy over the follow-up period reported.
Ocular Hypertension Treatment Study <sup>8,9</sup>	Ocular hypertension: medicine vs. no treatment	1636	5+	Topical therapy with ocular hypotensive medication was effective in delaying or preventing the onset of POAG in selected patients with elevated IOP.

\*POAG = primary open-angle glaucoma, IOP = intraocular pressure.

The investigators concluded from these findings that IOP does play a significant role in the pathogenesis of normal-tension glaucoma. These results also support the aggressive lowering of IOP in patients at risk for progression. However, not all untreated patients progressed in this study: 65% showed no further progression at 5 years and longer. This finding strongly suggests that treatment should have minimal side effects so as not to cause harm or decrease the patient's quality of life.

In the Advanced Glaucoma Intervention Study<sup>5</sup> the long-term clinical course and prognosis with two surgical treatment protocols were compared in eyes with moderate to advanced glaucoma. After 7 years of follow-up, the investigators were able to analyse the accrued data in order to draw certain corollary conclusions. In their "predictive analysis" of 738 eyes, they found a statistically significant worsening of the visual field over 6 years in eyes with an early average pressure greater than 17.5 mm Hg compared to eyes

with initial average pressure less than 14 mm Hg. Furthermore, this worsening was found to become more marked over time.

In the "associative analysis," patients with IOP consistently below 18 mm Hg experienced less visual field loss than those with IOP below this value only some of the time, especially with longer follow-up. There was almost no visual field loss on average in the former group. Although the average change in visual field was close to zero, a proportion of these eyes did have visual field loss despite having pressure measurements at this level.

The investigators concluded that a lower IOP is indeed associated with reduced progression of visual field loss.

The main purpose of the Collaborative Initial Glaucoma Treatment Study<sup>7</sup> was to assess the effect of two treatment approaches (initial therapy with topically administered medications versus initial trabeculectomy) in 607 patients with newly diagnosed

open-angle glaucoma. The investigators chose to use a calculation to determine target IOP range. The formula used was as follows:

$$\text{target IOP} = (1 - [\text{reference IOP} + \text{VF score}]/100) \times \text{reference IOP}$$

where reference IOP = baseline IOP, and VF = visual field. Target IOP was set aggressively. For example, in a patient with no visual field damage, the IOP would be lowered by its own value (e.g., an initial IOP of 25 mm Hg would be lowered by 25%). A small amount of field damage resulted in even more aggressive lowering (e.g., VF score of 5). For this patient the target IOP would be  $(1 - [25 + 5]/100) \times 25 = 17.5$  mm Hg.

The study findings showed that surgical treatment produced greater reductions in IOP than medical treatment (46% vs. 35%). Interestingly, there was no difference in visual field protection over the follow-up period of approximately 5 years. This may have been due to the aggressively set targets (with, on average, 35% or greater reduction in IOP) or to the fact that the follow-up period was too short to demonstrate a difference.

The investigators concluded that aggressive initial medical IOP management was as effective as early surgical therapy over the follow-up period reported and that there were no marked differences in quality of life between the groups.

The purpose of the Ocular Hypertension Treatment Study<sup>8,9</sup> was to determine the efficacy and safety of topical therapy with ocular hypotensive medication in delaying or preventing the onset of POAG. A total of 1636 patients with elevated IOP but no evidence of glaucomatous disc or field damage were randomly assigned to receive either observation or treatment. The treatment goal was to achieve a reduction in IOP of 20% or more, and an IOP of less than 25 mm Hg. The primary outcome measure was the development of a reproducible visual field abnormality or reproducible optic disc deterioration attributable to POAG. At 60 months patients in the observed group had a greater cumulative probability of manifesting POAG than the treated patients (9.5% vs. 4.4%,  $p < 0.001$ ).

The investigators concluded that topical therapy with ocular hypotensive medication was effective in delaying or preventing the onset of POAG in certain patients with elevated IOP. They clarified that this does not imply that all patients with borderline or elevated IOP should receive medication but, rather, that

clinicians should consider treatment for patients at moderate or high risk for POAG.

## TARGET INTRAOCULAR PRESSURE RANGE: WHAT IS IT?

In 1989 the American Academy of Ophthalmology defined target IOP range as “the upper limit of a stable range of pressures deemed unlikely to cause further optic nerve damage in a particular eye.”<sup>10</sup> The European Glaucoma Society developed their own definition in 1998: “the mean IOP obtained with treatment that prevents further glaucomatous damage.”<sup>11</sup> Brubaker<sup>12</sup> settled on this working definition of target IOP: “the IOP at which the rate of retinal ganglion cell loss is no greater than the age-related loss.”

Regardless of the definition used, the problem remains that there are no accurate methods in place to determine precisely what a safe level of IOP is for any given optic nerve status.

## WHY SET A TARGET INTRAOCULAR PRESSURE RANGE?

The fundamental goal for vision preservation in glaucoma is to slow the rate of retinal ganglion cell loss. By decreasing the IOP, significant therapeutic benefit can be derived (Fig. 1).<sup>12</sup>

Setting a target IOP range provides physicians with a measurable therapeutic goal. This goal is something that can be shared by the patient and the physician alike, helping forge a therapeutic alliance between them.

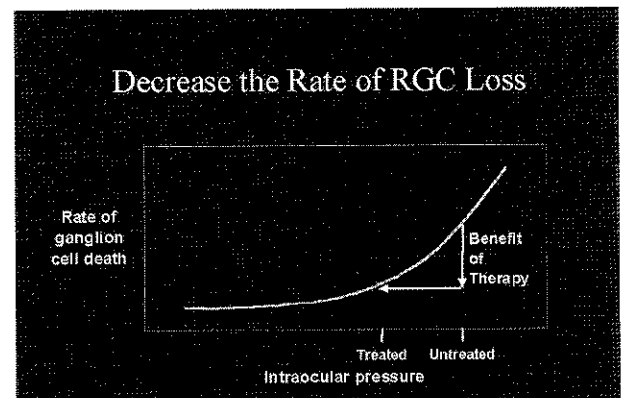


Fig. 1—Plot of retinal ganglion cell (RGC) loss as a function of intraocular pressure (reprinted from reference 12 with permission from Elsevier Science Inc. [*American Journal of Ophthalmology* 1996;121:473–83]).

Setting a target IOP range also assists in standardizing the approach to IOP lowering among ophthalmologists. It allows us to educate patients about their diagnosis and involve them in decisions regarding their treatment. Finally, it permits an individualized approach to each eye of a given patient.

The patient's quality of life is an important consideration when establishing a target IOP range. The benefits of IOP lowering must be weighed against any potential harm or inconvenience. Or, as Hodapp and colleagues<sup>13</sup> stated, "the risk and cost, including side effects of treatment to lower pressure, must be weighed against the risk of pressure itself."

#### **APPROACHES TO SETTING INITIAL TARGET INTRAOCULAR PRESSURE RANGE**

Several approaches have been advocated for setting the initial target IOP range.

##### **Percentage lowering**

The American Academy of Ophthalmology currently recommends lowering of at least 20% from baseline IOP.<sup>14</sup> The European Glaucoma Society currently recommends lowering of at least 30% from baseline IOP.<sup>11</sup>

##### **Absolute cutoff based on evidence**

For glaucoma suspects (based on elevated IOP), the evidence from the Ocular Hypertension Treatment Study<sup>8,9</sup> suggests a target pressure range of less than 25 mm Hg. In the associative analysis in the Advanced Glaucoma Intervention Study,<sup>5</sup> eyes with an IOP consistently lower than 18 mm Hg had almost no visual field deterioration over 5 years. Eyes with an IOP lower than 18 mm Hg only 50% of the time had significant visual field deterioration. In very advanced glaucoma (near-total cupping with split fixation) the evidence suggests that stability can be achieved when the IOP is in the low teens.<sup>15,16</sup>

##### **Formula-based cutoff**

In the Collaborative Initial Glaucoma Treatment Study<sup>7</sup> the following formula was used: target IOP =  $(1 - [\text{reference IOP} + \text{VF score}]/100) \times \text{reference IOP}$ . Jampel<sup>17</sup> proposed the following formula: target range =  $(\text{initial IOP} \times [1 - \text{initial IOP}/100] - Z + Y)$ ,

where Z is an optic nerve damage severity factor and Y is a burden of therapy factor.

#### **CONSIDERATIONS**

Target IOP range is a dynamic concept. The range can vary between patients and between eyes in the same patient in cases such as injury, pseudoexfoliation and advanced damage to one eye. In pseudoexfoliation one may wish to consider setting a lower target IOP range in view of the volatile and unpredictable IOP course (i.e., IOP control can escape between visits) or to maintain more vigilant, frequent follow-up.

Diurnal fluctuation is an important consideration when setting target IOP range. Large diurnal fluctuations in IOP are an independent risk factor for progression in patients with POAG.<sup>18</sup> It has been shown that more diurnal fluctuation correlates with worsening clinical status, even after adjustment for strong risk factors such as age, race and advanced initial field loss.<sup>18</sup> Careful consideration should be given to the time of day that IOP measurements are obtained. It may be best if the IOP is measured at the same time of day when starting medical treatment and at different times when assessing diurnal variation.

There are limitations to setting target IOP range. The concept relies heavily on accurate assessment of IOP, which may depend on the operator, the type of instrument, corneal variables, the cooperation of the patient and the status of the eye.<sup>19</sup> It may also be oversimplifying treatment in that it may fail to consider non-IOP risk factors.

Target IOP range needs to be constantly reevaluated. If the IOP range has been achieved, the IOP and optic nerve should be reevaluated every 3 to 12 months and the visual field every 6 to 24 months, depending on the degree of damage and the IOP control.<sup>20</sup> If there is evidence of progression in the optic nerve (e.g., a change in the neuroretinal rim or a disc hemorrhage) or if there is deterioration in the visual field, despite IOP values within the target range, a new, lower target may be necessary. Conversely, the target IOP range may have to be increased if achieving the target IOP causes adverse ocular or systemic side effects.

#### **TREATMENT OPTIONS**

There are many treatment options available to help achieve a desired target IOP range. Sometimes combinations of these treatments are necessary.

Medical treatment is one option available. There is a variety of medications that can be used to achieve target IOP. Compliance, tachyphylaxis and trough effect can all influence the effectiveness of medical treatment.

Trabeculoplasty (argon laser, selective laser or diode laser) can effectively lower the IOP to the desired target range in appropriately selected patients.

Surgery is another viable treatment option. Trabeculectomy can be very effective in achieving target IOP range. Nonpenetrating filtering surgery can also lower pressure effectively, although long-term data are needed. Surgical implantation of aqueous drainage devices can be effective in difficult cases. Cyclodestruction can be performed as a last-resort option.

## SUMMARY

There is an abundance of evidence from recent randomized clinical trials showing that lowering the IOP is beneficial to the optic nerve and visual field. Setting and achieving a target IOP range is in keeping with evidence-based medicine. The benefits of reaching this target must be weighed against the risks of the treatment itself. Target IOP is a dynamic concept, needing constant reevaluation.

What is lacking are established guidelines for determining the target IOP range that can be used in general ophthalmology practice.

## GUIDELINE DEVELOPMENT WORKSHOP

The Canadian Target IOP Workshop participants reviewed the literature and received commentary on the US perspective from Dr. Steven Simmons, a leading glaucoma specialist from Albany, NY. Current Canadian perspectives were put to practical application through a case study format, which served as a lead-in to the development of the working recommendations. The cases varied widely, from the very complex to the relatively simple, from suspects to patients with early-onset glaucomatous optic neuropathy to patients with advanced glaucomatous optic neuropathy. However, regardless of the complexity of the case, consensus was achieved as to what the target IOP range should be. The process clearly illustrated that it was possible to develop guidelines for setting target IOP range.

## Disclaimer

This disclaimer was developed in consultation with the Canadian Medical Protective Association in order to address specific medicolegal implications, discussed within the workshops, inherent in setting IOP guidelines.

The role of these guidelines is to provide eye care practitioners and their patients with support in the decision-making process for setting target IOP range in patients affected by or suspected of having glaucoma. This work is based on the glaucoma literature, regional target IOP conferences and personal clinical experience. The purpose is to offer a guide, and not a protocol. Clinical care must be individualized to the patient, the treating eye care professional and the socioeconomic milieu.

All contributors and the sponsor disclaim responsibility and liability for any and all adverse medical or legal effects resulting directly or indirectly from the use of the guidelines.

## Working recommendations

### *Establishing baseline intraocular pressure*

To establish a target IOP range, an accurate baseline IOP is needed. Practically, when establishing baseline IOP, a single applanation reading may be sufficient. The time of day the pressure is measured should be noted, especially if treatment is to be started at the first visit. However, it is helpful to have more than one IOP reading, and this can include previously documented IOP values from the referral source or a diurnal tension curve.

A thorough assessment should also be performed to make sure all relevant risk factors and treatment considerations are taken into account when setting the initial target IOP range. The initial assessment should include the following components:

- History-taking: ocular, systemic and family
- Ocular examination: IOP measurement, gonioscopy, documentation of baseline optic nerve appearance
- Testing of the visual field(s): it is important to obtain at least one reliable baseline visual field consistent with the optic nerve appearance
- Additional investigations as appropriate: imaging of the optic nerve and nerve fibre layer, measurement of the central corneal thickness.

**Table 2—Staging an eye at risk of or with glaucoma**

**Suspect (at least one of the following)**

- IOP above 22 mm Hg (adjusted for pachymetry if available)
- Suspicious disc or cup-to-disc asymmetry greater than 0.2
- Suspicious visual field defect on Humphrey 24-2 (or similar) testing

**Early disease**

- Early glaucomatous disc features (e.g., cup:disc ratio 0.65 or less) or mild visual field defect not within 10° of fixation, or both

**Moderate disease**

- Moderate glaucomatous disc features (e.g., cup:disc ratio 0.7–0.85) or moderate visual field defect not within 10° of fixation, or both

**Advanced disease**

- Advanced glaucomatous disc features (e.g., cup:disc ratio 0.9 or greater) or visual field defect within 10° of fixation, or both
- Also consider baseline Humphrey 10-2 visual field (or similar)

*Glaucomatous disc features*

The working recommendations use the following features to help define a glaucomatous disc:

- Increased cup-to-disc ratio (relative to disc size<sup>21</sup>), significant disc asymmetry
- Decreased or documented change in neuroretinal rim area
- Notch of neuroretinal rim
- “Saucerization” of neuroretinal rim
- Flame-shaped disc hemorrhage
- Nerve fibre layer loss
- 360° peripapillary atrophy

*Target intraocular pressure range*

Based on the staging of the glaucomatous eye, the Target IOP Workshops developed suggested upper limits of *initial* target IOP range for each eye (Table 3). The lower limit of target IOP is episcleral venous pressure.

It is important to note that these target IOP ranges may need to be modified based on the presence or development of risk factors, quality-of-life issues or the length of time the pressure has been maintained.

*Risk factors affecting initial target pressure range*

There are many risk factors for progression that should be considered when establishing an initial target IOP range (Table 4).

*Staging criteria*

The Target IOP Workshops developed recommendations for staging an eye at risk of or with glaucoma (Table 2). This staging can then be used to help determine an appropriate target IOP range.

**Table 3—Suggested upper limits of initial target IOP range for each eye\***

**Suspect (if decision is made to treat)†**

< 25 mm Hg with reduction of at least 20% from baseline IOP

**Early disease**

< 21 mm Hg with reduction of at least 20% from baseline IOP

**Moderate disease**

< 18 mm Hg with reduction of at least 30% from baseline IOP

**Advanced disease**

< 15 mm Hg with reduction of at least 30% from baseline IOP

\*Modify as needed based on longevity, quality of life and risk factors for progression.

†See the Ocular Hypertension Treatment Study.<sup>8,9</sup>

**Table 4—Risk factors for progression that may modify the initial target IOP range**

- Presence and severity of damage to involved or fellow eye<sup>22,23</sup>
- Rapid rate of progression of damage to involved or fellow eye<sup>24</sup>
- Family history of or genetic mutation predisposing to early-onset disease or severe disease, or both<sup>25</sup>
- African ancestry<sup>5</sup>
- Age<sup>26</sup> and life expectancy
- Vascular risk factors: disc hemorrhage,<sup>27</sup> nocturnal hypotension,<sup>28</sup> migraine,<sup>27</sup> Raynaud's disease, diabetes mellitus,<sup>7,29</sup> previous vein occlusion
- Large fluctuation or instability in IOP<sup>30</sup> (e.g., IOP spikes, pseudoexfoliation syndrome)
- Poor follow-up<sup>22</sup>
- Axial myopia (speculative)

Ongoing evaluation of the target IOP range is imperative. At subsequent visits the practitioner should reevaluate the patient, examining the following areas: medication (side effects, compliance), any relevant general health problems, the IOP (both the method of measurement and the time of day should be recorded), the optic nerve (for any change in glaucomatous features) and, depending on the clinical picture, the visual field. It is important to distinguish between "ideal," "borderline" and "acceptable" IOP. For example, in a patient with a normal disc and no detectable visual field loss whose target IOP range is

set at less than 21 mm Hg, although 20 mm Hg may be "ideal" and 21 mm Hg or 22 mm Hg may be "borderline," one IOP reading in the mid 20s may be "acceptable."

Based on the reevaluation, the target IOP range may have to be adjusted. If there is evidence of progression in the optic nerve or a reproducible change in the visual field despite achieving an IOP within the target range, the target IOP range should be lowered. If the findings suggest that achieving the target IOP range caused unacceptable ocular or systemic side effects, a higher target IOP range may have to be accepted.

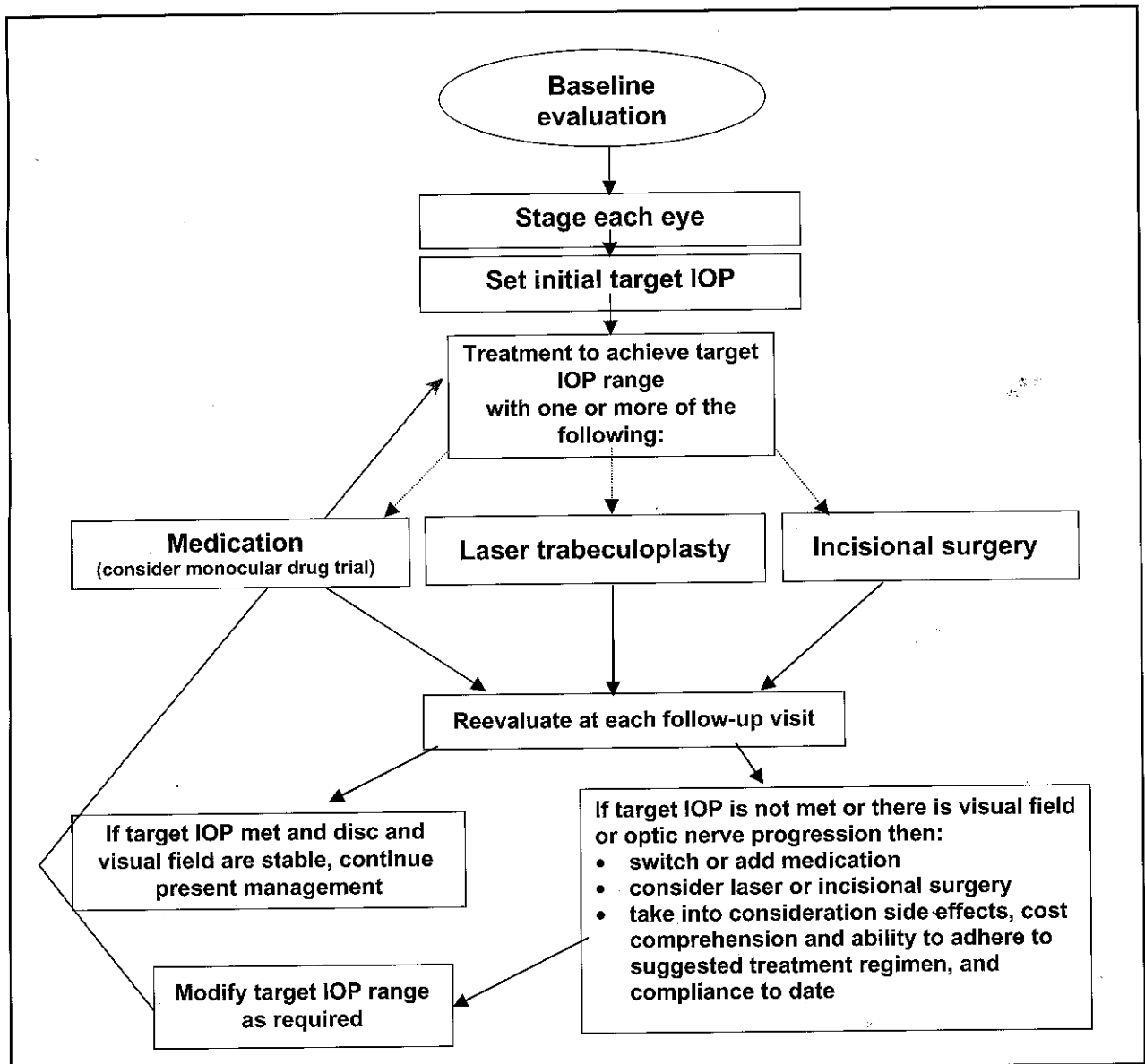



Fig. 2—Summary approach to managing patients with glaucoma. IOP = intraocular pressure.

## Summary approach to managing patients with glaucoma

The management of patients with glaucoma is summarized in Fig. 2. 

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## REFERENCES

- Sommer A, Tielsch JM, Katz J, Quigley HA, Gottsch JD, Javitt 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.
- Leske CM, Connell AM, Wu SY, Hyman LG, Schacht AP. Risk factors for open-angle glaucoma. The Barbados Eye Study. *Arch Ophthalmol* 1995;113:918-24.
- Armaly MF, Krueger DE, Maunder L, Becker B, Hetherington J Jr, Kolker AE, et al. Biostatistical analysis of the collaborative glaucoma study: 1. Summary report of the risk factors for glaucomatous visual-field defects. *Arch Ophthalmol* 1980;98:2163-71.
- Palmberg P. Target IOP: How low? American Academy of Ophthalmology Subspecialty Day glaucoma meeting; 2001 Nov 10; New Orleans.
- 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(4):429-40.
- Collaborative Normal-Tension Glaucoma Study Group (CNTGS). The effectiveness of intraocular pressure reduction in the treatment of normal-tension glaucoma. *Am J Ophthalmol* 1998;126:498-505.
- Janz NK, Wren PA, Lichter PR, Musch DC, Gillespie BW, Guire KE, et al. The Collaborative Initial Glaucoma Treatment Study: interim quality of life findings after initial medical or surgical treatment of glaucoma. *Ophthalmology* 2001;108:1954-65.
- Kass MA, Heuer DK, Higginbotham EJ, Johnson CA, Keltner JL, Miller JP, 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(6):701-13.
- Gordon MO, Beiser JA, Brandt JD, Heuer DK, Higginbotham EJ, Johnson CA, et al. The Ocular Hypertension Treatment Study: baseline factors that predict the onset of primary open-angle glaucoma. *Arch Ophthalmol* 2002;120:714-20.
- American Academy of Ophthalmology. *Preferred Practice Pattern: primary open-angle glaucoma*. San Francisco: The Academy; 1989.
- European Glaucoma Society. *Terminology and guidelines for glaucoma*. Savona (Italy): The Society; 1998.
- Brubaker RF. Delayed functional loss in glaucoma. LII Edward Jackson Memorial Lecture. *Am J Ophthalmol* 1996;121(5):473-83.
- Hodapp E, Parrish RP, Anderson D. *Clinical decisions in glaucoma*. St Louis: CV Mosby Company; 1993. p. 65.
- American Academy of Ophthalmology. *Preferred Practice Pattern: primary open-angle glaucoma*. San Francisco: The Academy; 2000.
- Shirakashi M, Iwata K, Sawaguchi S, Abe H, Nanba K. Intraocular pressure-dependent progression of visual field loss in advanced primary open-angle glaucoma: a 15-year follow-up. *Ophthalmologica* 1993;207:1-5.
- Odberg T. Visual field prognosis in advanced glaucoma. *Acta Ophthalmol* 1987;182(Suppl 65):27-9.
- Jampel HD. Target pressure in glaucoma therapy. *J Glaucoma* 1997;6:133-8.
- Asrani S, Zeimer R, Wilensky J, Gieser D, Vitale S, Lindenmuth K. Large diurnal fluctuations in intraocular pressure are an independent risk factor in patients with glaucoma. *J Glaucoma* 2000;9:134-42.
- Whitacre MM, Stein R. Sources of error with use of Goldmann-type tonometers. *Surv Ophthalmol* 1993;38: 1-30.
- American Academy of Ophthalmology. *Summary benchmarks for Preferred Practice Patterns*. San Francisco: The Academy; 2000. Available: [www.aaof.org/aaof/education/library/benchmarks.cfm](http://www.aaof.org/aaof/education/library/benchmarks.cfm) (accessed 2002 July 31).
- Quigley HA, Brown AE, Morrison JD, Drance SM. The size and shape of the optic disc in normal human eyes. *Arch Ophthalmol* 1990;108:51-7.
- Grant WM, Burke JF. Why do some people go blind from glaucoma? *Ophthalmology* 1982;89:991-8.
- Suzuki Y, Shirato S, Adachi M, Hamada C. Risk factors for the progression of treated primary open-angle glaucoma: a multivariate life-table analysis. *Graefes Arch Clin Exp Ophthalmol* 1999;237:463-7.
- 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.
- Wilson R, Walker AM, Dueker DK, Crick RP. Risk factors for rate of progression of glaucomatous visual field loss. *Arch Ophthalmol* 1982;100:737-41.
- Stewart WC, Kolker AE, Sharpe ED, Day DG, Holmes KT, Leech JN, et al. Factors associated with long-term progression or stability in primary open-angle glaucoma. *Am J Ophthalmol* 2000;130:274-9.
- Drance S, Anderson DR, Schulzer M, for the Collaborative Normal-Tension Glaucoma Study Group. Risk factors for progression of visual field abnormalities in normal-tension glaucoma. *Am J Ophthalmol* 2001;131: 699-708.
- Graham SL, Drance SM. Nocturnal hypotension: role in glaucoma progression. *Surv Ophthalmol* 1999;43(Suppl 1):S10-6.
- 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.

30. Ishida K, Yamamoto T, Kitazawa Y. Clinical factors associated with progression of normal-tension glaucoma. *J Glaucoma* 1998;7:372-7.

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### Self-test /Auto-évaluation\*

1. Which of these randomized controlled trials demonstrates that intraocular pressure (IOP) does play a significant role in the pathogenesis of normal-tension glaucoma?
  - a. Collaborative Initial Glaucoma Treatment Study
  - b. Ocular Hypertension Treatment Study
  - c. Advanced Glaucoma Intervention Study
  - d. Collaborative Normal-Tension Glaucoma Study
2. Which of the following is not an acceptable definition of target IOP range:
  - a. The upper limit of a stable range of pressure values deemed unlikely to cause further optic nerve damage
  - b. The mean IOP obtained with treatment that prevents further glaucomatous damage
  - c. The specific level of pressure that, if achieved, will definitely prevent further nerve damage
  - d. The IOP at which the rate of retinal ganglion cell loss is no greater than the age-related loss
3. Factors that may modify initial target IOP range include all of the following except:
  - a. African ancestry
  - b. Axial hyperopia
  - c. Disc hemorrhage
  - d. Presence and severity of damage to the involved eye
4. Initial treatment options to lower IOP in patients with newly diagnosed primary open-angle glaucoma generally include each of the following except:
  - a. Aqueous drainage device
  - b. IOP-lowering medication
  - c. Laser trabeculoplasty
  - d. Trabeculectomy
5. In the Ocular Hypertension Treatment Study, IOP was lowered to:
  - a. < 28 mm Hg with at least 20% lowering
  - b. < 25 mm Hg with at least 20% lowering from baseline
  - c. 30% lowering from baseline
  - d. < 21 mm Hg with at least 30% lowering from baseline

\*These questions can be answered and scored, and a certificate obtained, on the COS Web site ([www.eyesite.ca](http://www.eyesite.ca))./On peut soumettre ses réponses et recevoir une note globale ainsi qu'un certificat dans le site Web de la SCO ([www.eyesite.ca](http://www.eyesite.ca)).