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GLAUCOMA



Rita Dhamankar

Foreword
Sushmita Kaushik

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CHAPTER 3

What Role does Intraocular Pressure Play in Glaucoma?

Rita Dhamankar

INTRODUCTION

Many randomized controlled trials (RCTs) have proven the importance of lowering the intraocular pressure (IOP) to prevent the progression of glaucoma. These are Early Manifest Glaucoma Trial (EMGT), Ocular Hypertension Treatment Study (OHTS), Collaborative Initial Glaucoma Treatment Study (CIGTS), Advanced Glaucoma Intervention Study (AGIS), and Normal-Tension Glaucoma Trial (CNTG). It is the most important risk factor and is measured during all examinations without fail. It is the only modifiable factor. It ranges from 12 to 17 mm Hg ± 2 standard deviations in the normal population, skewed a little to the higher side. 21 mm Hg cannot be taken as a cut off **(Fig. 1)**.

HOW DO WE MEASURE THE INTRAOCULAR PRESSURE?

There are many known ways of measuring the IOP. The oldest and crudest of them was the digital method where on applying digital pressure on the closed lids, you could get an assumption of hard/soft the eyeball is. However, that is just a guesstimate and cannot be documented in terms of figures. It is very subjective and varies from observer to observer. However, there are many IOP measuring devices working on different principles which are known as

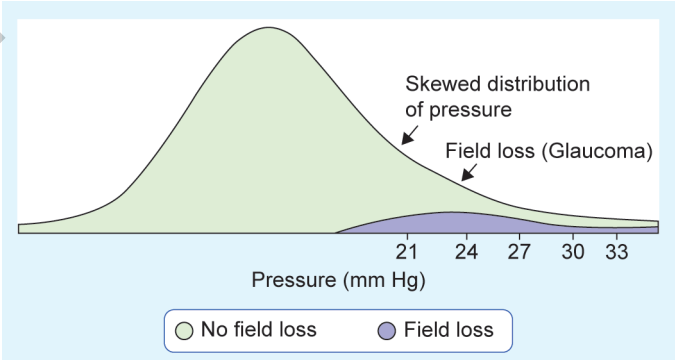

Fig. 1: Skewed distribution of intraocular pressure (IOP) and field loss in a population.

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tonometers and can give you a digital value that can be documented from time to time. The gold standard today is the principle of applanation used by the slit lamp mounted *Goldmann applanation tonometer (GAT)*. Applanation tonometry is also used in the handheld, portable *Perkin's tonometer*. The IOP can be measured in the supine position as well, especially in ill patients, or when measuring the diurnal IOP. A good screening tool is the *noncontact tonometer*. Noncontact tonometry offers the benefit of assessment without anesthesia through an automated process with less risk for contamination and corneal injury. Measurements are obtained in seconds and displayed in a digital format. Studies have demonstrated increased utility of the *Tono-Pen* compared with GAT in patients with corneal irregularities such as scarring or in eyes with a history of keratoplasty due to its smaller area of contact with the cornea (Azuara-Blanco et al., 1998; Rootman et al., 1988). In eyes with corneal edema, the Tono-Pen appears more accurate than GAT with intracameral assessment of pressure (Neuburger et al., 2013).

The *dynamic contour tonometer/ocular response analyzer (ORA)* significantly overestimates IOP compared with GAT, particularly at higher IOP levels (Martinez-de-la-Casa et al., 2006). Both Goldmann IOP (IOPg) and corneal compensated IOP (IOPcc) measure significantly higher than GAT with mean differences of 7.2 and 8.3 mm Hg, respectively, suggesting these values are not interchangeable with GAT. Corneal hysteresis was found to be independent of GAT and IOPg but is related to IOPcc; however, studies have shown very small difference between the IOP values generated. The IOPcc in general, correlates with GAT but is less influenced by corneal properties and remains stable after keratorefractive surgery (Pepose et al., 2007). It gives you an IOP measurement independent of the corneal thickness. Then of course is *the transpalpebral rebound tonometer* which can be used without any topical anesthesia in children or in patients allergic to the topical drugs. Rebound tonometry correlates well with GAT within the normal range of IOPs; however, IOP may be overestimated and underestimated at high and low IOPs, respectively (Subramaniam et al., 2021; Fernandes et al., 2005; Martinez-de-la-Casa et al., 2005; Iliev et al., 2006; Kato et al., 2018). At higher IOP, ≥23 mm Hg, rebound tonometry overestimates IOP compared with GAT (Gao et al., 2017). Additionally, IOP values obtained by rebound tonometry are positively correlated with central corneal thickness (CCT), possibly to a greater degree than GAT (Fernandes et al., 2005; Gao et al., 2017; Brusini et al., 2006; Johannesson et al., 2008). I-Care measurements should be obtained from the central cornea, as peripheral measurements may underestimate IOP (Muttuvelu et al., 2012). Corneal curvature is not correlated with rebound tonometry readings (Johannesson et al., 2008). In eyes with corneal edema, rebound tonometry was found to be more accurate than GAT when compared to intracameral assessment of IOP (Neuburger et al., 2013). Cannot forget to mention the age-old *Schiotz*, which has problems with ocular/scleral rigidity,

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but is a much used tonometer in camps. Last but not to be forgotten is the *digital tonometry*, a misnomer, but come in handy when no other form of tonometry works.

There are *errors in measurements* of the IOP due to many reasons. IOP is dynamic and varies according to the circadian rhythm. It also depends on the CCT as the GAT is calibrated for a CCT of 520 μm. There is no algorithm for correction of the corneal thickness that can be used as a correction factor. It is known from the OHTS study that thinner corneas are known to progress much faster than thicker corneas. Thinner corneas, normally show false lower readings, which could be misleading when treating a patient. In practice due to the diurnal variations in IOP, in real time practice, we are still not sure as to which IOP should we be looking at, the mean IOP, the peak, or the trough. Also, what about short-term and long-term fluctuations? Other factors like breath holding must be avoided when recording a GAT reading. Astigmatism can give wrong readings, 1 mm Hg for every 4 D of astigmatism. The rebound or the transpalpebral tonometer determines the IOP by applanating the bouncing a small plastic-tipped metal corneal contour probe against the attached to a pressure sensor in the tip. This creates an induction current from ocular pulse from which IOP is measured amplitude.

Mires in the applanation tonometry are seen **(Figs. 2A to C and Flowchart 1)**.

ERRORS IN TONOMETRY

Intraocular pressure (IOP) is dynamic. It follows the circadian rhythm, hence, varies according to that. It is very important to note the time that the IOP has been recorded. There can be *measurement errors*, as the mires may be too thick/thin. They may not be matched exactly. We know that the *corneal thickness influences the IOP*. The GAT has been calibrated for a corneal thickness of 520 μm. Thicker corneas record false high readings and vice versa. However, *there is no standardized algorithm* for the same, so cannot be used in real time practice. From the OHTS study we know that patients with thinner corneas are known to progress faster, also patients with thinner corneas give us a false low reading and hence we might be unaware of the severity looking at the IOP in isolation. Herein comes the importance of *measuring the CCT*. IOP as has been documented early follows a circadian rhythm, and hence has *diurnal variations***.** The IOP in the supine position is also higher than in the erect position, so these factors must be noted, e.g., in a patient with hypertension, if he takes a BP lowering medication at night and goes to bed, we are enhancing progression, as he has a low BP and a high IOP when sleeping, which results in low ocular perfusion pressure. This leads to a vascular dysregulation and ischemia to the optic nerve head, leading to progression of glaucoma. To prevent this, the timing of the antihypertensive

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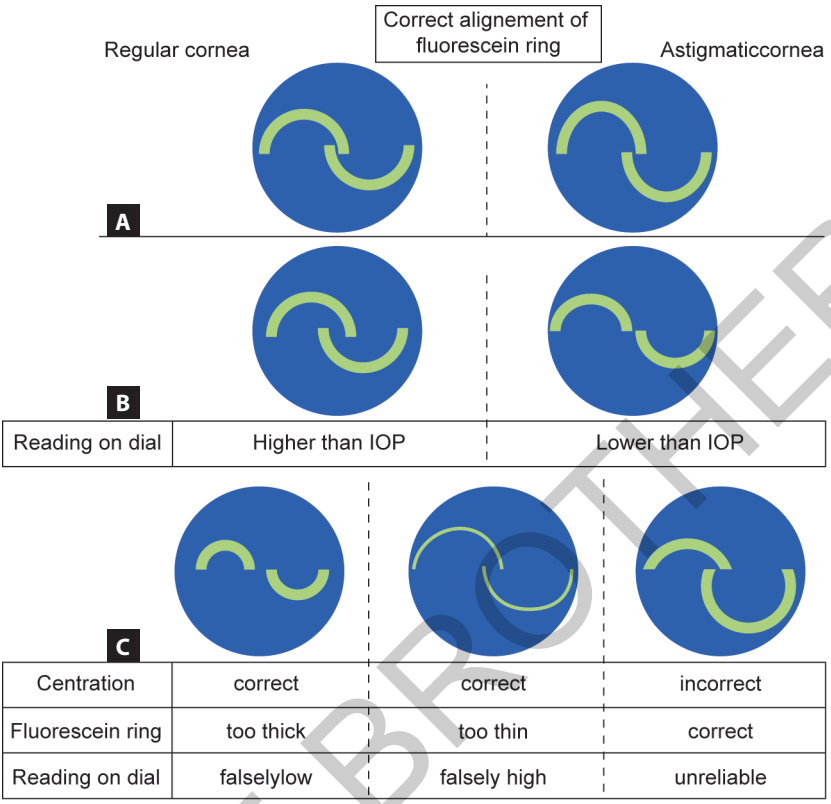
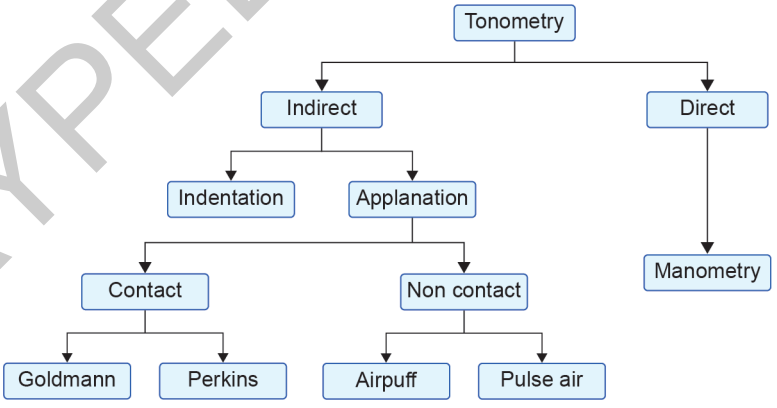


Figs. 2A to C: Various GAT mires interpretation.

Flowchart 1: Classification of tonometry.



medication can be altered in consultation of the treating physician. As the IOP varies throughout the day, which IOP should we consider in real time? *Should it be the mean IOP/peak IOP/IOP fluctuations? Breath holding* can cause a rise in IOP, and this should be avoided. *Astigmatism* induces a change

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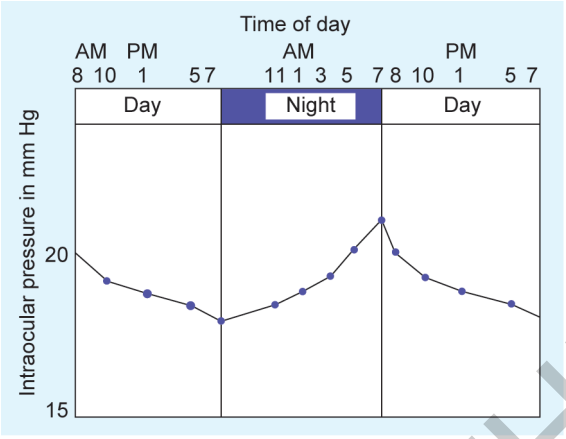

Fig. 3: Diurnal variation in intraocular pressure (IOP).

in IOP measurement. There is a rise of 1 mm Hg for every 4 D of astigmatism. All these factors have a place of concern in practice and must be thought of. We are all aware that we measure only snapshots of IOP which could be changing continuously, there are both short-term fluctuations and long-term fluctuations **(Fig. 3)**.

Diurnal Variation in Intraocular Pressure

Factors affecting IOP: Central corneal thickness, the circadian rhythm where the IOP was lowest between 2 and 4 AM, with a subsequent rise taking place during the latter third of the night's sleep period (Ciulla et al., 2020). Stress changes in body position during exercise and normal daily activities also result in transient IOP changes. Gravity inversion, positioning in the head-down vertical position, can more than double the IOP to pathologic levels, and may even cause glaucoma in circumstances of repeated prolonged activity (Friberg and Weinreb, 1985). In addition to physical activity, use of swimming goggles has been shown to cause IOP elevation by a mean of 4.5 mm Hg, which varied depending on the style of goggle used with one style causing a mean elevation of up to 13.4 mm Hg (Morgan et al., 2008). When changing from the sitting to supine position, IOP increased by around 4 mm Hg in eyes with ocular hypertension (OHT), low tension glaucoma, and controls with no glaucoma risk factors (Yamabayashi et al., 1991). Eyelid squeezing and rubbing can produce large excursions in IOP. Telemetric monitoring of IOP with an IOP implant sensor enabled van den Bosch et al. (van den Bosch et al., 2023) to examine the effects of eyelid muscle action on IOP in 11 patients with primary open-angle glaucoma (POAG). Eyelid rubbing induced an average peak IOP change of 59 mm Hg from baseline, squeezing induced a peak IOP increase of 42.2 mm Hg, relaxed lid closure induced a peak change of 3.8 mm Hg, and voluntary blinking induced a peak change of 11.6 mm Hg.

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HOW DOES INTRAOCULAR PRESSURE CAUSE GLAUCOMA?

Glaucoma is characterized by the progressive loss of retinal ganglion cells (RGCs) and their axons with corresponding functional loss of the visual field. While the precise mechanism(s) underlying, the pathogenesis of glaucoma has yet to be fully determined, it is clear that IOP plays an important role in triggering and propagating injury to RGCs **(Fig. 4)**.

When to Treat?

Higher the IOP greater the risk of progression. With IOP of 22–29 mm Hg, there is a 13-fold increase in the risk of converting to glaucoma. This increases to 40-fold once it reaches 30 mm Hg. Dr Palmberg analyzed the AGIS study and proposed the concept of target IOP

WHAT IS TARGET INTRAOCULAR PRESSURE?

The European Glaucoma Society guidelines define target IOP as "an estimate of the mean IOP obtained with treatment that is expected to prevent further glaucomatous damage". The American Academy of Ophthalmology defines target IOP as "a range of IOP adequate to stop progressive pressure-induced injury". The World Glaucoma Association defines it as "an estimate of the

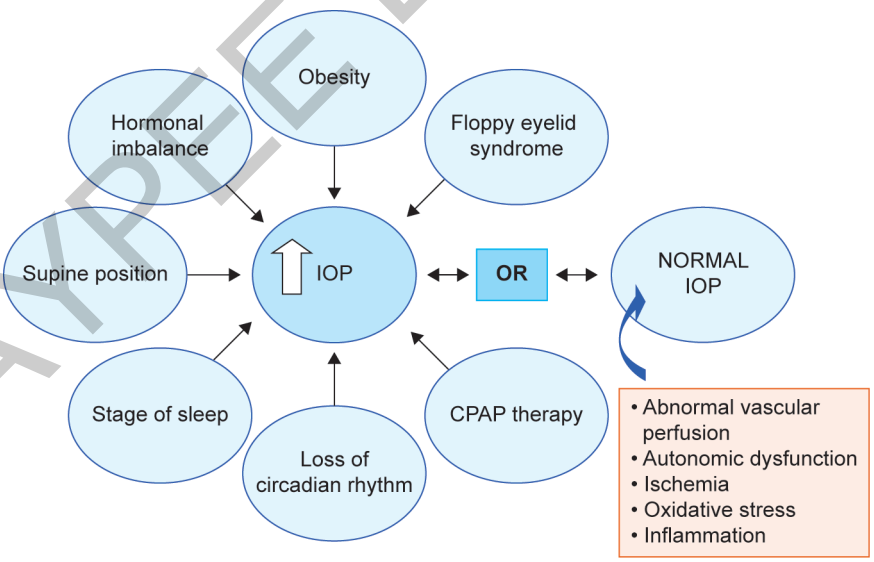

Fig. 4: Factors affecting IOP.
Source: van Gestel A, Webers CA, Severens JL, Beckers HJ, Jansonius NM, Hendrikse F, et al. The long-term outcomes of four alternative treatment strategies for primary open-angle glaucoma. Acta Ophthalmol. 2012;90(1):20-31.

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TABLE 1: Recommended intraocular pressure (IOP) range in various stages of glaucoma.

Stage of disease	*Recommend IOP range (mm Hg)*
Early glaucoma	15–17
Moderate glaucoma	12–15
Advanced glaucoma	10–12

Initial recommended target pressure range.

mean IOP at which the risk of decreased vision-related quality of life due to glaucoma exceeds the risk of the treatment".

Notes:
European Glaucoma Society. Terminology and Guidelines for Glaucoma, 3rd edition. Savona, Italy. DOGMA: European Glaucoma Society; 2008.

American Academy of Ophthalmology. Primary Open-Angle Glaucoma Preferred Practice Pattern. San Francisco, CA: American Academy of Ophthalmology; 2010.

World Glaucoma Association Consensus Statement: Intraocular Pressure. The Netherlands: Kluger; 2007.

Setting a "target" IOP range provide an algorithm for management **(Table 1)**. Quality of life may be affected by the medications used. Contrast sensitivity, mobility, night vision, and driving are significantly affected. Attaining a balance is the way to go lowering an IOP to such levels may need medications, lasers, and even surgery in some patients. Should one take a graded approach to get there? A study done by van Gestel et al. proved to the contrary.

WHAT ARE THE MEANS OF LOWERING INTRAOCULAR PRESSURE?

You can lower the IOP by *medications* acting by different mechanisms, reducing the aqueous production, increasing the outflow by both pathways—the conventional and the uveoscleral. You could use *lasers* in angle closure for doing an iridotomy or an iridoplasty, in POAG by doing a selective laser trabeculoplasty (SLT) and, of course, there is always surgery to resort to trabeculectomy, glaucoma valves, or the newly introduced minimally invasive glaucoma surgery (MIGS) **(Fig. 5)**.

Medications

The choice of medical therapy is guided by the baseline intraocular pressure and the desired target pressure, with escalation or combination therapy based on the initial response to treatment **(Flowchart 2)**.

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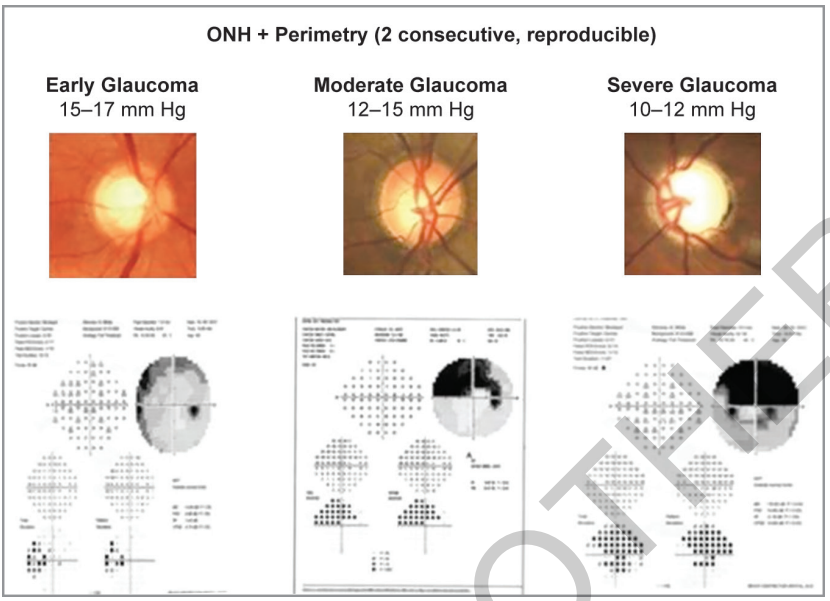
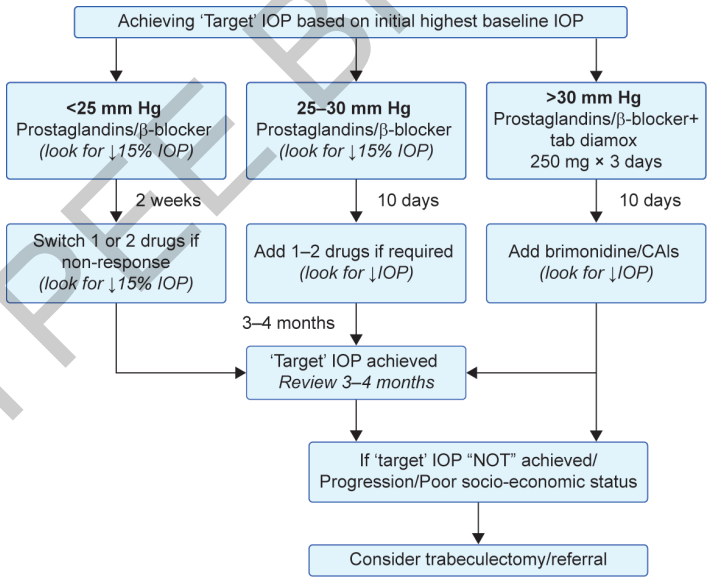


Fig. 5: Clinically applicable target intraocular pressure (IOP) range.

Flowchart 2: Use of medications based on baseline intraocular pressure (IOP).



Lasers

Like depicted earlier, a laser iridotomy or a laser iridoplasty is very useful in angle-closure disease as a primary treatment. In the open-angle glaucoma (OAG), an SLT works very well primarily, especially in treatment-naïve

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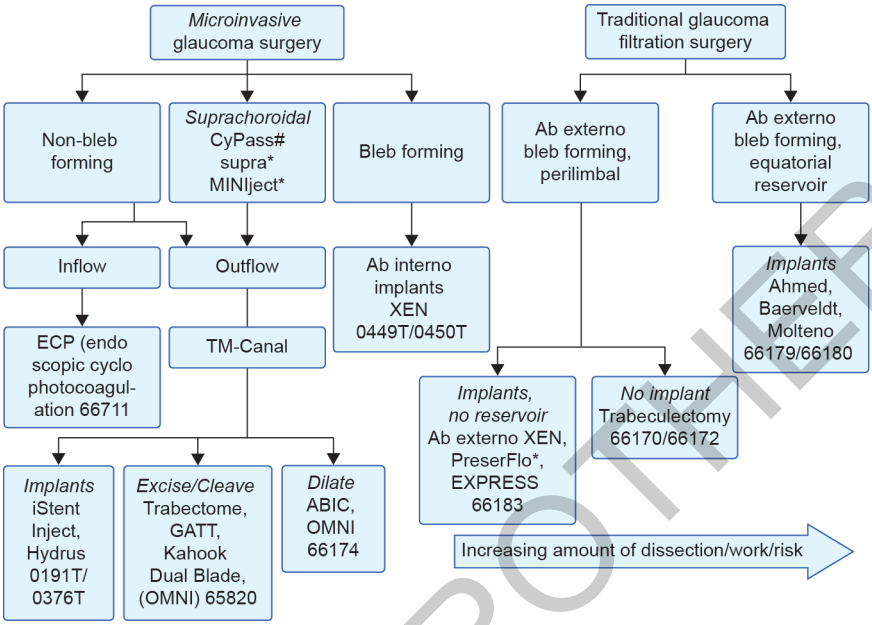
Flowchart 3: Surgical options.



patients. Patients already on prostaglandins need to be kept off prostaglandins at least for a month before SLT to get maximum action. It is a safe and repeatable procedure. The LiGHT trial has shown SLT is a safe treatment for OAG and OHT, providing better long-term disease control than initial drop therapy, with reduced need for incisional glaucoma and cataract surgery over 6 years **(Flowchart 3)**.

What are the Disadvantages of Target Intraocular Pressure?

Once a "target" IOP is set, patients could be stressed if it is not achieved at every visit. There may be possible medicolegal consequences if "target" IOP is considered the standard of care and progression continues. There is no certainty of no progression once a target IOP is reached.

Target IOP is very dynamic. It is possible that the target IOP may be set too low at the beginning. If at the end of aggressive treatment there is no change seen, one may reduce the treatment and let the IOP rise a little to see if the disease stays stable. Vice versa, if the target set is low and more risk factors set in such as hypertension and diabetes need for a systemic steroid for another illness, the target has to be reduced further.

SUGGESTED READING

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Rita Dhamankar MS DOMS is a practicing Ophthalmologist for the last 44 years. She has been working as the Head, Department of Glaucoma, Laxmi Eye Institute, Mumbai, Maharashtra, India, for the last 30 years. She has been a DNB Teacher for the last 20 years.

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