Elevated IOP After Cataract Surgery

Because patients who have already sustained glaucomatous damage are at greater risk of visual field loss from increased IOP, ophthalmologists must have an understanding of the various options for treating rises in pressure.

BY PARAG A. GOKHALE, MD, AND EMORY PATTERSON, MD

The phenomenon of elevated IOP following cataract surgery has been documented since the 1950s. In 1976, a review of 630 cases of cataract extraction with lens implants concluded that elevated IOP was transient and benign.[1] Nearly all patients' pressures returned to baseline with or without treatment. Some individuals, however, may experience pain, corneal edema, glaucomatous nerve damage, or anterior ischemic optic neuropathy.[2] It is therefore important to continue monitoring the effect of new cataract surgical techniques on postoperative IOP as well as the impact of increased IOP on visual outcomes.

Elevated pressure is the most frequent postoperative complication demanding treatment following phacoemulsification.[3] As many as 18% to 45% of patients may experience an IOP greater than 28 mm Hg following phacoemulsification, but most pressures will return to normal by 24 hours postoperatively.[1] The peaks most commonly occur 8 to 12 hours after surgery, and only 1.3% to 10.0% of cases record an IOP higher than 30 mm Hg 24 hours postoperatively. After uneventful phacoemulsification in eyes without glaucoma, however, IOP spikes may reach 68 mm Hg.[3]

In most patients, postoperative increases in IOP are transient and benign.[1] In individuals without glaucoma, no visual field defects were evident once the IOP returned to normal.[1] Although patients without optic nerve damage seem to tolerate transient increases in IOP without problems, glaucoma patients do not. The latter individuals experience further visual field loss and/or a loss of fixation.[1] These patients are also more likely to experience pressure spikes following cataract extraction.[1] Surgeons therefore must be keenly aware of glaucoma patients' risk for postoperative IOP spikes and understand the various treatment options for elevated pressure when it does occur.

> "Increases in IOP ... can lead to a further loss of retinal ganglion cells in patients whose optic discs have already been compromised."

ETIOLOGY

The causes of the elevated IOP are likely multifactorial. Other contributors include a preexisting compromise of outflow facility and retained ophthalmic viscosurgical devices (OVDs). Surgical trauma, watertight wound closure, retained lenticular debris, the release of iris pigment, hyphema, and inflammation are also thought to contribute to elevations in IOP.[3] The skillfulness of the surgeon has been implicated as well. Increased surgical experience is correlated with a decreased risk for ocular

hypertension following cataract extraction.[3]

A review of 2,727 phacoemulsification procedures over a 2-year period demonstrated that the most frequent complication of posterior capsular rupture (n = 45) was raised IOP. Nine eyes (20%) had an IOP exceeding 30 mm Hg 1 day after surgery despite prophylactic acetazolamide. Five of the nine eyes sustained vitreous loss requiring an anterior vitrectomy.[4]

THE ROLE OF OVDs

In 1983, Berson et al reported that sodium hyaluronate caused a substantial decrease (55% to 60%) in the outflow of aqueous humor when injected into the anterior chamber.[5] Subsequently, it has become well accepted that retained viscoelastic materials inhibit aqueous outflow and result in increased IOP.

Arshinoff has published multiple studies comparing different viscoelastic materials.[6,7] He concluded that, if not completely removed, all OVDs cause postoperative increases in IOP.[7] If no OVD is retained in the anterior chamber, however, then increases in IOP following cataract extraction are of no greater severity or duration than if no OVD had been used at all.

OVDs are generally classified according to their molecular weight and viscosity. Cohesive agents are more viscous than dispersive OVDs, and they have higher molecular weights and longer molecular chains. These properties make cohesive OVDs an excellent choice for maintaining space, stabilizing tissues, and opposing the posterior pressure that occurs during cataract extraction.

The particles of low-viscosity OVDs are considered dispersive, because they do not adhere to one another like they do in high-viscosity OVDs. Dispersive viscoelastics are better able than high-viscosity OVDs to protect individual structures in the anterior chamber such as the corneal endothelium.[6] Because of their dispersive nature, however, low-viscosity OVDs are generally more difficult to remove from the eye completely.

According to Arshinoff et al,[6] high-viscosity OVDs are associated with higher postoperative IOPs (although not necessarily above 21 mm Hg) compared with lower-viscosity OVDs. He asserted that retained viscoelastic and patients' predispositions (eg, trabecular insult or undiagnosed glaucoma) are the main causes of postoperative rises in IOP above 21 mm Hg.

ELEVATED IOP IN GLAUCOMA

Although increases in IOP after cataract surgery are usually benign, they can lead to a further loss of retinal ganglion cells in patients whose optic discs have already been compromised. In addition to individuals with glaucoma, those prone to anterior ischemic optic neuropathy are at increased risk for further visual field loss. They include persons with diabetes mellitus, nocturnal hypotension, anemia, and systemic arterial hypertension.[1] A patient's susceptibility as well as the duration and degree of ocular hypertension are all likely to factor into the permanent effects of increases in IOP following cataract extraction.[3]

Studies comparing patients with and without glaucoma have routinely revealed a difference in their postoperative rises in IOP. Shingleton et al reported a maximum increase in IOP to 44 versus 32 mm Hg in patients with and without glaucoma, respectively, 24 hours after cataract surgery.[8] Another study found a mean IOP of 29.9 mm Hg 8 hours after cataract surgery in patients with glaucoma compared with a mean IOP of 22.2 mm Hg 12 hours postoperatively in patients without glaucoma. Seven of the 13 eyes in the glaucoma group had peak IOPs that were greater than 35 mm Hg.[1] In their study, Arshinoff et al discovered that eight of 40 patients with elevated IOP had glaucoma, were glaucoma suspects, or were steroid responders. Subsequently, the investigators realized that higher IOP spikes correlated directly with glaucoma risk. Eight of 40 patients with a pressure greater than 21 mm Hg in either eye and two of four patients with a pressure higher than 30 mm Hg were found to fit into one of the glaucoma groups.[6]

MEDICAL TREATMENT

Pharmaceuticals

Although several drugs lower IOP after cataract surgery, none of them consistently prevents increases in pressure from occurring. The classes of drugs used to treat postoperative increases in IOP include carbonic anhydrase inhibitors (acetazolamide, dorzolamide, and brinzolamide), alpha agonists (apraclonidine and brimonidine), prostaglandin analogs (latanoprost and travoprost), beta-blockers (timolol and levobunolol), and miotics (intracameral carbachol, pilocarpine, and intracameral acetylcholine).

Acetazolamide has been used for many years to treat IOP increases following cataract extraction and has proven moderately successful. This carbonic anhydrase inhibitor was more effective than topical apraclonidine, an alpha agonist, in a head-to-head trial.[9] Another comparative study showed that subjects' mean IOP in the first 24 hours following cataract extraction was greater than 21 mm Hg in the acetazolamide group and less than 21 mm Hg in the dorzolamide group. Both groups, however, had an equal number of patients with an IOP greater than 30 mm Hg 4 hours following surgery.[10] Brinzolamide has been shown to be as effective as dorzolamide in controlling IOP postopera-

tively, but it is associated with less ocular discomfort following administration.[10] A study comparing acetazolamide and brinzolamide found that the drugs were equally effective at 4 to 6 hours after cataract surgery but that only brinzolamide produced a statistically significant decrease in IOP at 24 hours.[10]

Rainer et al compared dorzolamide and latanoprost, a prostaglandin analog. Both drugs produced a clinically significant reduction in IOP 6 hours after cataract surgery, but only dorzolamide was effective at 24 hours.[11] Neither drug prevented elevations in IOP greater than 30 mm Hg from occurring. A comparison of travoprost and brinzolamide showed that both produced a clinically significant decrease in IOP 6 and 24 hours postoperatively. Neither, however, was always able to prevent a spike greater than 30 mm Hg.[2]

Tests of apraclonidine to prevent postoperative increases in IOP have been inconsistent in their results. The explanation may be differences in surgical technique, surgeons' experience, the OVD used, or the administration of the IOP-lowering agents. Most recently, Kasetti et al found no benefit with apraclonidine versus placebo to reduce postoperative IOP and prevent pressure spikes.[9]

The alpha-2 agonist brimonidine 0.2% dosed b.i.d. on the day before and the day after cataract surgery was more effective than placebo at reducing postoperative IOP. The mean IOP in the brimonidine group was significantly lower than in the placebo group at most time points. At 6 hours postoperatively, one patient in the brimonidine group and six in the placebo group experienced an IOP spike greater than 10 mm Hg. No patients treated with brimonidine had a peak IOP exceeding 30 mm Hg.[12] In other studies in which subjects received one drop of brimonidine 0.2% 1 hour before surgery or just after cataract extraction, the drug did not produce a significant decrease in IOP compared to placebo.[13,14]

In another study, timolol but not latanoprost was effective in reducing postoperative IOP. In fact, patients receiving one drop of timolol at the end of surgery had a mean decrease in IOP of 4.77 mm Hg and 2.99 mm Hg at 4 and 24 hours, respectively.[15]

Rainer et al compared a fixed dorzolamide-timolol combination with latanoprost. The fixed combination reduced postoperative IOP more effectively, and it prevented any increase in IOP to greater than 30 mm Hg.[16] Another study comparing a dorzolamide-timolol combination to placebo found the fixed combination to produce a clinically significant reduction in postoperative IOP. The agent, however, did not completely prevent pressure spikes greater than 30 mm Hg.[17]

A 1992 report concluded that intracameral carbachol was the most effective medication to control IOP following extracapsular cataract extraction. Timolol, acetazolamide, pilocarpine, and levobunolol also produced a clinically significant reduction in IOP but were less effective.[18] In addition, carbachol was more effective than acetylcholine when both drugs were administered intracamerally.[19]

Decompression of the Anterior Chamber

Another proposed method of controlling IOP after cataract surgery is decompressing the anterior chamber. In 2003, Hildebrand et al found that decompression effectively corrected 11 consecutive cases of severely increased IOP.[3] Pressure decreased from a range of 40 to 68 mm Hg to a mean of 4.73 ±3.00 mm Hg immediately after decompression. The IOP, however, rapidly rose to greater than 30 mm Hg at 30 minutes and 38.5 mm Hg at 60 minutes after decompression. Hildebrand et al therefore concluded that this measure provides only a transient benefit and that additional treatment is necessary in high-risk eyes.[3] Arshinoff recommended multiple attempts at sideport drainage. He proposed decompressing the anterior chamber hourly for 3 hours in combination with the administration of one drop each of pilocarpine 2% and latanoprost q.i.d. for 2 days postoperatively.[7]

PROPOSED GUIDELINES

Ophthalmologists must recognize the potential for postoperative increases in IOP spikes following uncomplicated phacoemulsification, know the risk factors for this complication, and be comfortable with a variety of treatment options. In patients with known outflow obstruction or optic nerve damage or in those who are already being treated for increased IOP, we recommend prophylactic treatment both before and after surgery. Surgeons should be aggressive in their removal of OVDs from the eye. They should inject carbachol intracamerally at the end of surgery. If a posterior capsular rupture occurs intraoperatively, we recommend aggressive treatment to lower the IOP postoperatively. The medications used will depend on the patient's tolerance (eg, due to allergy and systemic conditions).

We recommend the following approach for patients with high-risk eyes if they tolerate the medications. Surgeons should administer a fixed combination of timolol and dorzolamide along with brimonidine at the end of the case, and patients should instill these drugs at their usual scheduled time. Depending on the patient's level of risk for postoperative IOP spikes and the status of his optic nerve at the time of surgery, it may be prudent to see him later on the operative day and to perform serial paracenteses if his IOP is elevated. If paracenteses are required, surgeons can consider prescribing prostaglandins and/or cholinergics

up to q.i.d. for 2 days after surgery. If the patient is at high risk, then performing combined cataract and glaucoma surgery or glaucoma surgery alone before cataract surgery may be in his best interest. ■

Reprinted with permission from Glaucoma Today*'s May/June 2007 edition.*

Parag A. Gokhale, MD, is a fellowship-trained glaucoma specialist at the Virginia Mason Medical Center in Seattle. Dr. Gokhale may be reached at (206) 583-2300; ophpag@vmmc.org.

Emory Patterson, MD, graduated from the Medical College of Georgia in Augusta and will begin his ophthalmology residency at that institution this year. Dr. Patterson may be reached at epattersonmd@students.mcg.edu

1. Tranos P, Bhar G, Little B. Postoperative intraocular pressure spikes: the need to treat. *Eye*. 2004;18:673-679.
2. Ermis SS, Ozturk F, Inan UU. Comparing the effects of travoprost and brinzolamide on intraocular pressure after phacoemulsification. *Eye*. 2005;19:303-307.
3. Hildebrand GD, Wickremasinghe SS, Tranos PG, et al. Efficacy of anterior chamber decompression in controlling early intraocular pressure spikes after uneventful phacoemulsification. *J Cataract Refract Surg*. 2003;29:1087-1092.
4. Ang GS, Whyte IF. Effect and outcomes of posterior capsule rupture in a district general hospital setting. *J Cataract Refract Surg*. 2006;32:623-627.
5. Berson FG, Patterson MM, Epstein DL. Obstruction of aqueous outflow by sodium hyaluronate in enucleated human eyes. *Am J Ophthalmol*. 1983;96:668-672.
6. Arshinoff S, Albiani DA, Taylor-Laporte J. Intraocular pressure after bilateral cataract surgery using Healon, Healon 5, and Healon GV. *J Cataract Refract Surg*. 2002;28:617-625.
7. Arshinoff S. Postoperative intraocular pressure spikes. *J Cataract Refract Surg*. 2004;30:733-734.
8. Shingleton BJ, Gamell LS, O'Donoghue MW, et al. Long-term changes in intraocular pressure after clear corneal phacoemulsification: normal patients versus glaucoma suspect and glaucoma patients. *J Cataract Refract Surg*. 1999;25:885-890.
9. Kasetti SR, Desai SP, Sivakumar S, Sunderraj P. Preventing intraocular pressure increase after phacoemulsification and the role of perioperative apraclonidine. *J Cataract Refract Surg*. 2002;28:2177-2180.
10. Dayanir V, Ozcura F, Kir E, et al. Medical control of intraocular pressure after phacoemulsification. *J Cataract Refract Surg*. 2005;31:484-488.
11. Rainer G, Menapace R, Schmetterer K, et al. Effect of dorzolamide and latanoprost on intraocular pressure after small incision cataract surgery. *J Cataract Refract Surg*. 1999;25:1624-1629.
12. Katsimpris JM, Dimitrios S, Konstas AGP, et al. Efficacy of brimonidine 0.2% in controlling acute postoperative intraocular pressure elevation after phacoemulsification. *J Cataract Refract Surg*. 2003;29:2288-2294.
13. Cetinkaya A, Akman A, Akova YA. Effect of topical brinzolamide 1% and brimonidine 0.2% on intraocular pressure after phacoemulsification. *J Cataract Refract Surg*. 2004;30:1736-1741.
14. Rainer G, Menapace R, Findl O, et al. Effect of topical brimonidine on intraocular pressure after small incision cataract surgery. *J Cataract Refract Surg*. 2001;27:1227-1231.
15. Lai JSM, Chua, JKH, Leung ATS, et al. Latanoprost versus timolol gel to prevent ocular hypertension after phacoemulsification and intraocular lens implantation. *J Cataract Refract Surg*. 2000;26:386-391.
16. Rainer G, Menapace R, Findl O, et al. Intraindividual comparison of the effects of a fixed dorzolamide-timolol combination and latanoprost on intraocular pressure after small incision cataract surgery. *J Cataract Refract Surg*. 2001;27:706-710.
17. Rainer G, Menapace R, Findl O, et al. Effect of a fixed dorzolamide-timolol combination on intraocular pressure after small-incision cataract surgery with Viscoat. *J Cataract Refract Surg*. 2003;29:1748-1752.
18. Fry LL. Comparison of the postoperative intraocular pressure with Betagan, Betoptic, Timoptic, Iopidine, Diamox, Pilopine Gel, and Miostat. *J Cataract Refract Surg*. 1992;18:14-19.
19. Ruiz RS, Rhem MN, Prager TC. Effects of carbachol and acetylcholine on intraocular pressure after cataract extraction. *Am J Ophthalmol*. 1989;107:7-10.