Managing Traumatic Hyphema, *p. 18* • Recognizing Pseudotumor Cerebri, *p. 79*

REVIEW® OF OPTOMETRY

July 15, 2018

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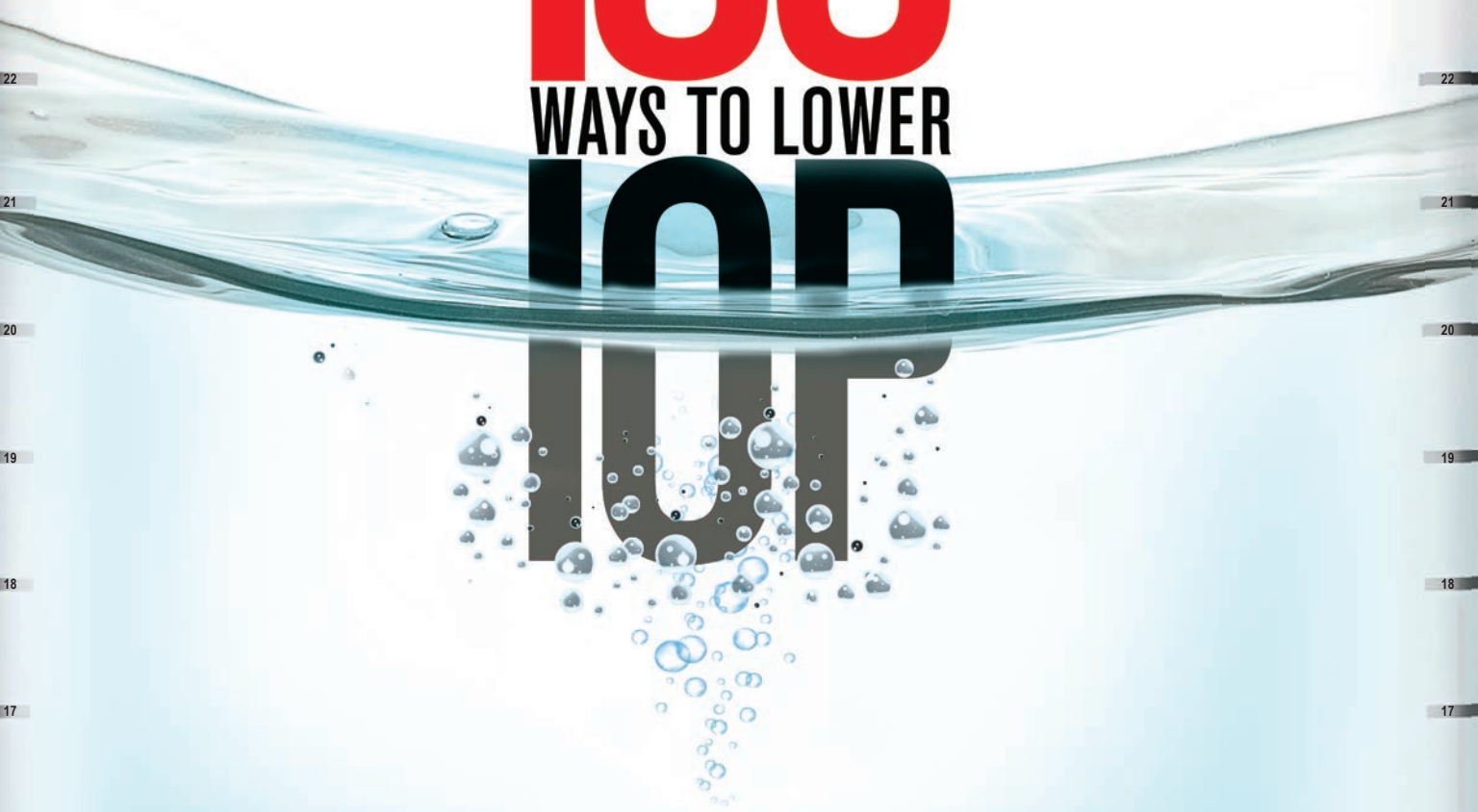
24TH ANNUAL GLAUCOMA REPORT

100 WAYS TO LOWER IOP



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IN THE NEWS

A new study suggests the neural circuit mechanisms underlying **recovery of eye dominance and acuity operate independently**, potentially turning current **amblyopia treatment on its head**. The study evaluated the visual acuity of mice for whom the ngr1 gene had been removed; the results show that, even for mice whose eye dominance remained impaired, visual acuity could still improve.

Stephany C, Ma X, Dorton H, et al. Distinct circuits for recovery of eye dominance and acuity in murine amblyopia. Current Biology. June 7, 2018. [Epub ahead of print].

State Senator **Ed Hernandez, OD, of West Covina, Calif., recently won the Democratic Party's nomination for California's Lieutenant Governor** seat.[1] He's earned the endorsement of several local eye care practices and California optometrists. His website boasts various health care related goals and policies, such as championing lower drug prices and passing clean water and air regulations.[2]

1. Thompson J. California Optometric Association applauds Dr. Ed Hernandez's election night victory. www.coavision.org/files/Ed%20Hernandez%20Primary%20Statement%20FINAL.pdf. June 6, 2018. Accessed June 7, 2018.
2. Dr. Ed Hernandez for Lt. Governor 2018. www.edhernandez4ca.com/media. Accessed June 7, 2018.

Researchers recently used **binocular optical coherence tomography (OCT) to measure the size of strabismus**, with positive results. In evaluating 15 patients with strabismus and 15 controls with both anterior segment OCT and alternating prism cover test (APCT), they found OCT correctly identified the type and direction of the deviation in all 15 patients with strabismus. Imaging was strongly correlated with the APCT measurement as well.

Chopra R, Mulholland PJ, Tailor VK, et al. Use of a binocular optical coherence tomography system to evaluate strabismus in primary position. JAMA Ophthalmol. May 31, 2018. [Epub ahead of print].

New Hypertension Guidelines: Now What?

Research suggests some screening changes that may impact your care.

By Bill Kekevian, Senior Editor

Like diabetes, hypertension rates in the United States and worldwide have exploded. In fact, researchers estimate that 1.3 billion people worldwide suffer from it.[1] For most primary care physicians, this is fairly easily handled by encouraging the patient to make lifestyle changes to control their blood pressure (BP), as well as prescribing BP-lowering medications. But, for eye care clinicians, lowering BP can have a dangerous side effect: elevated risk for glaucoma. Although it is weak, researchers point to a significant positive relationship between systemic blood pressure and intraocular pressure (IOP). "The relationship between glaucoma and systemic hypertension is multifaceted and often puzzling," reads a report, published in the *Journal of Glaucoma*.[2]

Part of that puzzle involves discovering precisely what that relationship is. The authors relied on population-based studies, prospective longitudinal studies and randomized clinical trials to organize "evidence that lower systemic BP and lower ocular perfusion pressure are associated with greater glaucoma prevalence and a higher rate of glaucoma progression." However, they are careful not to imply that treating systemic hypertension increases the risk of glaucoma.

Glaucoma specialist James Fanelli, OD, says this research further supports the idea that optometrists "should consider systemic blood pressure a modifiable risk factor for both glaucoma development and glaucoma progression."

But it isn't a simple see-saw effect in which one goes up, causing the other to go down. One of the major findings of the research shows that "extreme falls in nighttime—but not daytime—systolic and diastolic blood pressure were significantly associated with a higher prevalence of glaucomatous optic neuropathy."

Dr. Fanelli adds that patients should avoid taking BP medications at night, as a nocturnal drop in BP is often when IOP tends to increase.

Due to the nuanced nature of BP and IOP, the authors recommend a close partnership between eye care and primary care for those straddling hypertension and glaucoma.

"ODs don't interact with internal medicine and family practice and the medical community as a whole enough," Dr. Fanelli adds. "We need to comanage and take an active role. It needs to be done all the time."

1. Bloch MJ. Worldwide prevalence of hypertension exceeds 1.3 billion. J American Society of Hypertension: JASH. 2016;10(10):753-4.
2. De Moraes C, Cioffi G, Weinreb R, Liebmann J. Perspective: new recommendations for the treatment of systemic hypertension and their potential implications for glaucoma management. J Glaucoma. May 10, 2018. [Epub ahead of print].

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OCT-A Not Always Better than OCT

Although the diagnostic value of optical coherence tomography angiography (OCT-A) compared with conventional OCT measurement in glaucoma remains inconclusive, many researchers and clinicians fall into the trap of assuming that newer is always better.

However, researchers in Hong Kong took a closer look at both modalities and found OCT-A has its limitations, leading them to believe conventional OCT remains a better option when viewing the macula in some cases.

The study includes 115 patients with glaucoma and 35 healthy individuals. The researchers measured "retinal thickness and retinal vessel density over the 3x3mm^2 macula using swept-source OCT (SS-OCT) and OCT-A, respectively. They found the inner macular vessel density was 4.3% smaller in eyes with glaucoma compared with healthy eyes. Inner macular vessel thickness was also smaller by 21.1µm, compared with healthy eyes.

When comparing the two measurements, they found that "at 90% specificity, the sensitivity of mean inner macular thicknesses for detection of glaucoma was greater than that of mean inner macular vessel densities," the study said.

"OCT measurement of inner macular thickness shows a higher diagnostic performance to detect glaucoma and a stronger structure-function association than the currently used OCT-A measurement of inner macular vessel density," the study authors conclude.

"These findings may suggest that OCT-A of the macula has a limited role in the diagnostic evaluation of glaucoma."

Wan KH, Lam AKN, Leung CK. Optical coherence tomography angiography compared with optical coherence tomography macular measurements for detection of glaucoma. JAMA Ophthalmol. May 31, 2018. [Epub ahead of print].

"Tele-optometry" Offers Remote Exams

A new telemedicine company says it can help optometrists perform virtual eye exams. Touting this concept as "tele-optometry," DigitalOptometrics hopes to provide convenience to patients seeking exams and greater opportunities for ODs—without the acrimony that typically surrounds remote refraction services.

The concept behind DigitalOptometrics is a software system that allows optometrists within the company's network to perform a comprehensive eye exam remotely. Doctors can also interact with patients through live video conferencing during the exam.

Here's how it works: A patient can walk into a participating practice or optical site without an appointment. They would then enter their information and medical history on a computer or tablet. Next, an ophthalmic technician located at the participating site would perform a prescreening exam, including autorefraction, tonometry, fundus photography, a video slit-lamp and auto-lensmeter readings. The information would then be sent to a licensed optometrist who would check the visual findings and discusses them with the patient through a live videoconference. The patient then would receive an eyeglass or contact lens prescription.

DigitalOptometrics says the entire exam takes 30 minutes or less. If an ocular issue shows up, the doctor refers the patient to a specialty provider as needed.

ODs "are not limited to examining patients at a single location but can perform comprehensive exams of patients at multiple locations" remotely, says Howard Fried, OD, company president.

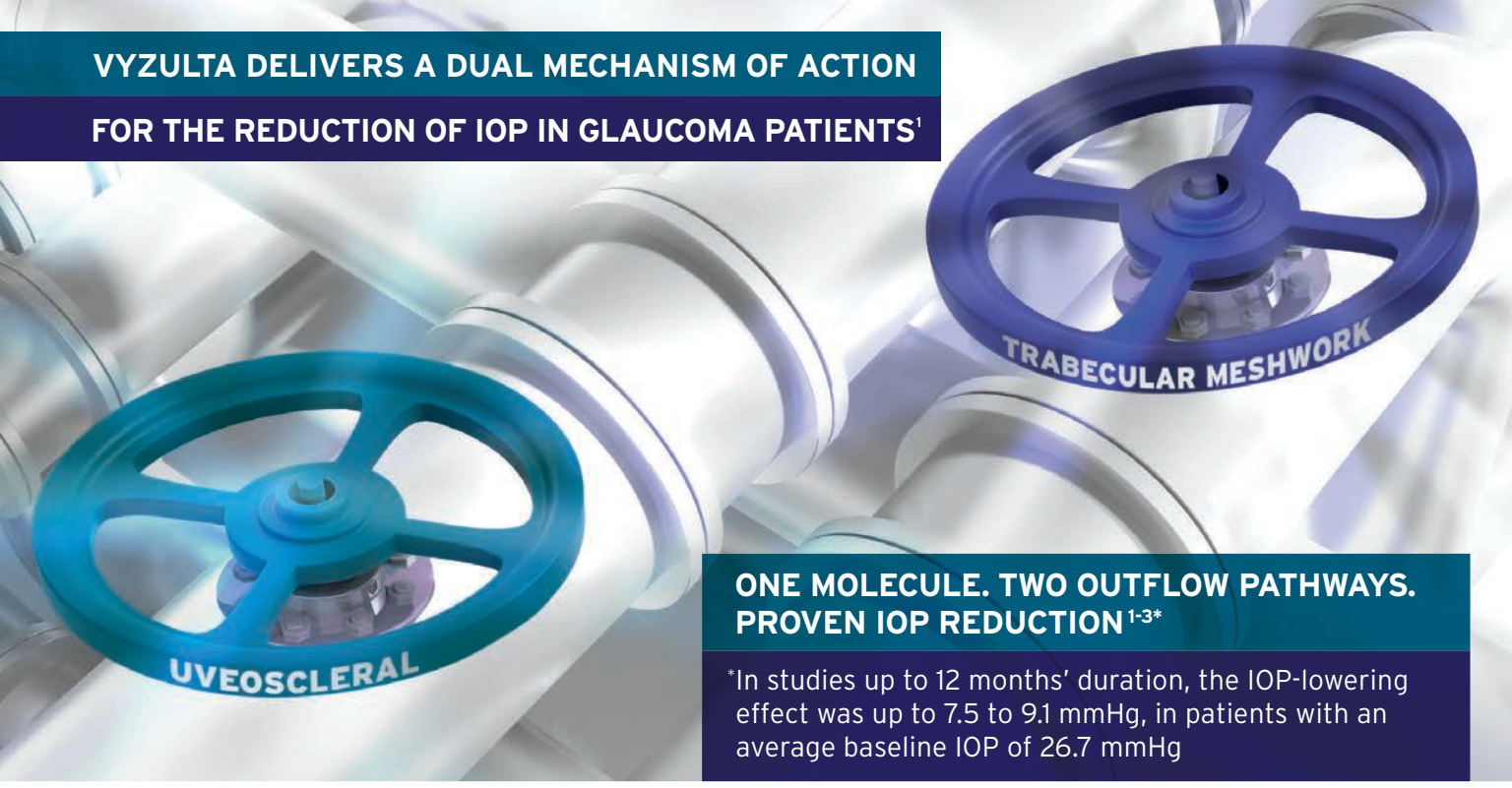
Another plus, according to Dr. Fried, is that optometrists could have greater flexibility and even the opportunity to work in their home offices, which would allow homebound licensed optometrists the chance to return to their profession.

Now optometrists "can expand their practices by opening additional locations and can, in fact, be in two to three places at the same time by seeing some of their patients in person, while seeing other patients remotely," he adds.

Dr. Fried explains a host provider can use their own ODs to serve multiple locations, DigitalOptometrics's optometrists or a combination of both. With a "flip of a switch," an OD can turn off the remote system and use the software manually to do direct office exams, he adds.

Dr. Fried says his system differs from other telehealth optical systems primarily because it consists of a comprehensive eye health and vision analysis that includes a subjective refraction and allows voice and visual communication between the remote doctor and patient. ■

VYZULTA DELIVERS A DUAL MECHANISM OF ACTION FOR THE REDUCTION OF IOP IN GLAUCOMA PATIENTS[1]



ONE MOLECULE. TWO OUTFLOW PATHWAYS. PROVEN IOP REDUCTION[1-3*]

*In studies up to 12 months' duration, the IOP-lowering effect was up to 7.5 to 9.1 mmHg, in patients with an average baseline IOP of 26.7 mmHg

INDICATION

VYZULTA™ (latanoprostene bunod ophthalmic solution), 0.024% is indicated for the reduction of intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension.

IMPORTANT SAFETY INFORMATION

- Increased pigmentation of the iris and periorbital tissue (eyelid) can occur. Iris pigmentation is likely to be permanent
- Gradual changes to eyelashes, including increased length, increased thickness, and number of eyelashes, may occur. These changes are usually reversible upon treatment discontinuation
- Use with caution in patients with a history of intraocular inflammation (iritis/uveitis). VYZULTA should generally not be used in patients with active intraocular inflammation
- Macular edema, including cystoid macular edema, has been reported during treatment with prostaglandin analogs. Use with caution in aphakic patients, in pseudophakic patients with a torn posterior lens capsule, or in patients with known risk factors for macular edema

IMPORTANT SAFETY INFORMATION (CONTINUED)

- There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products that were inadvertently contaminated by patients
- Contact lenses should be removed prior to the administration of VYZULTA and may be reinserted 15 minutes after administration
- Most common ocular adverse reactions with incidence ≥2% are conjunctival hyperemia (6%), eye irritation (4%), eye pain (3%), and instillation site pain (2%)

For more information, please see Brief Summary of Prescribing Information on next page.

References:

1. VYZULTA Prescribing Information. Bausch & Lomb Incorporated. 2017.
2. Weinreb RN, Sforzolini BS, Vittitow J, Liebmann J. Latanoprostene bunod 0.024% versus timolol maleate 0.5% in subjects with open-angle glaucoma or ocular hypertension: the APOLLO study. *Ophthalmology*. 2016;123(5):965-973.
3. Medeiros FA, Martin KR, Peace J, Sforzolini BS, Vittitow JL, Weinreb RN. Comparison of latanoprostene bunod 0.024% and timolol maleate 0.5% in open-angle glaucoma or ocular hypertension: the LUNAR study. *Am J Ophthalmol*. 2016;168:250-259.

For more information about VYZULTA and how it works, visit vyzultanow.com

VYZULTA™
(latanoprostene bunod ophthalmic solution), 0.024%

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BRIEF SUMMARY OF PRESCRIBING INFORMATION

This Brief Summary does not include all the information needed to use VYZULTA safely and effectively. See full Prescribing Information for VYZULTA.

VYZULTA™ (latanoprostene bunod ophthalmic solution), 0.024%, for topical ophthalmic use.

Initial U.S. Approval: 2017

1 INDICATIONS AND USAGE

VYZULTA™ (latanoprostene bunod ophthalmic solution) 0.024% is indicated for the reduction of intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension.

4 CONTRAINDICATIONS

None

5 WARNINGS AND PRECAUTIONS

5.1 Pigmentation

VYZULTA™ (latanoprostene bunod ophthalmic solution), 0.024% may cause changes to pigmented tissues. The most frequently reported changes with prostaglandin analogs have been increased pigmentation of the iris and periorbital tissue (eyelid).

Pigmentation is expected to increase as long as latanoprostene bunod ophthalmic solution is administered. The pigmentation change is due to increased melanin content in the melanocytes rather than to an increase in the number of melanocytes. After discontinuation of VYZULTA, pigmentation of the iris is likely to be permanent, while pigmentation of the periorbital tissue and eyelash changes are likely to be reversible in most patients. Patients who receive prostaglandin analogs, including VYZULTA, should be informed of the possibility of increased pigmentation, including permanent changes. The long-term effects of increased pigmentation are not known.

Iris color change may not be noticeable for several months to years. Typically, the brown pigmentation around the pupil spreads concentrically towards the periphery of the iris and the entire iris or parts of the iris become more brownish. Neither nevi nor freckles of the iris appear to be affected by treatment. While treatment with VYZULTA™ (latanoprostene bunod ophthalmic solution), 0.024% can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly *[see Patient Counseling Information (17) in full Prescribing Information].*

5.2 Eyelash Changes

VYZULTA may gradually change eyelashes and vellus hair in the treated eye. These changes include increased length, thickness, and the number of lashes or hairs. Eyelash changes are usually reversible upon discontinuation of treatment.

5.3 Intraocular Inflammation

VYZULTA should be used with caution in patients with a history of intraocular inflammation (iritis/uveitis) and should generally not be used in patients with active intraocular inflammation as it may exacerbate this condition.

5.4 Macular Edema

Macular edema, including cystoid macular edema, has been reported during treatment with prostaglandin analogs. VYZULTA should be used with caution in aphakic patients, in pseudophakic patients with a torn posterior lens capsule, or in patients with known risk factors for macular edema.

5.5 Bacterial Keratitis

There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products. These containers had been inadvertently contaminated by patients who, in most cases, had a concurrent corneal disease or a disruption of the ocular epithelial surface.

5.6 Use with Contact Lens

Contact lenses should be removed prior to the administration of VYZULTA because this product contains benzalkonium chloride. Lenses may be reinserted 15 minutes after administration.

6 ADVERSE REACTIONS

The following adverse reactions are described in the Warnings and Precautions section: pigmentation (5.1), eyelash changes (5.2), intraocular inflammation (5.3), macular edema (5.4), bacterial keratitis (5.5), use with contact lens (5.6).

6.1 Clinical Trials Experience

Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in practice.

VYZULTA was evaluated in 811 patients in 2 controlled clinical trials of up to 12 months duration. The most common ocular adverse reactions observed in patients treated with latanoprostene bunod were: conjunctival hyperemia (6%), eye irritation (4%), eye pain (3%), and instillation site pain (2%). Approximately 0.6% of patients discontinued therapy due to ocular adverse reactions including ocular hyperemia, conjunctival irritation, eye irritation, eye pain, conjunctival edema, vision blurred, punctate keratitis and foreign body sensation.

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Risk Summary

There are no available human data for the use of VYZULTA during pregnancy to inform any drug associated risks.

Latanoprostene bunod has caused miscarriages, abortion, and fetal harm in rabbits. Latanoprostene bunod was shown to be abortifacient and teratogenic when administered intravenously (IV) to pregnant rabbits at exposures ≥ 0.28 times the clinical dose. Doses ≥ 20 µg/kg/day (23 times the clinical dose) produced 100% embryofetal lethality. Structural abnormalities observed in rabbit fetuses included anomalies of the great vessels and aortic arch vessels, domed head, sternebral and vertebral skeletal anomalies, limb hyperextension and malrotation, abdominal distension and edema. Latanoprostene bunod was not teratogenic in the rat when administered IV at 150 mcg/kg/day (87 times the clinical dose) *[see Data]*.

The background risk of major birth defects and miscarriage for the indicated population is unknown. However, the background risk in the U.S. general population of major birth defects is 2 to 4%, and of miscarriage is 15 to 20%, of clinically recognized pregnancies.

Data

Animal Data

Embryofetal studies were conducted in pregnant rabbits administered latanoprostene bunod daily by intravenous injection on gestation days 7 through 19, to target the period of organogenesis. The doses administered ranged from 0.24 to 80 mcg/kg/day. Abortion occurred at doses ≥ 0.24 mcg/kg/day latanoprostene bunod (0.28 times the clinical dose, on a body surface area basis, assuming 100% absorption). Embryofetal lethality (resorption) was increased in latanoprostene bunod treatment groups, as evidenced by increases in early resorptions at doses ≥ 0.24 mcg/kg/day and late resorptions at doses ≥ 6 mcg/kg/day (approximately 7 times the clinical dose). No fetuses survived in any rabbit pregnancy at doses of 20 mcg/kg/day (23 times the clinical dose) or greater. Latanoprostene bunod produced structural abnormalities at doses ≥ 0.24 mcg/kg/day (0.28 times the clinical dose). Malformations included anomalies of sternum, coarctation of the aorta with pulmonary trunk dilation, retroesophageal subclavian artery with absent brachiocephalic artery, domed head, forepaw hyperextension and hindlimb malrotation, abdominal distention/edema, and missing/fused caudal vertebrae.

An embryofetal study was conducted in pregnant rats administered latanoprostene bunod daily by intravenous injection on gestation days 7 through 17, to target the period of organogenesis. The doses administered ranged from 150 to 1500 mcg/kg/day. Maternal toxicity was produced at 1500 mcg/kg/day (870 times the clinical dose, on a body surface area basis, assuming 100% absorption), as evidenced by reduced maternal weight gain. Embryofetal lethality (resorption and fetal death) and structural anomalies were produced at doses ≥ 300 mcg/kg/day (174 times the clinical dose). Malformations included anomalies of the sternum, domed head, forepaw hyperextension and hindlimb malrotation, vertebral anomalies and delayed ossification of distal limb bones. A no observed adverse effect level (NOAEL) was established at 150 mcg/kg/day (87 times the clinical dose) in this study.

8.2 Lactation

Risk Summary

There are no data on the presence of VYZULTA in human milk, the effects on the breastfed infant, or the effects on milk production. The developmental and health benefits of breastfeeding should be considered, along with the mother's clinical need for VYZULTA, and any potential adverse effects on the breastfed infant from VYZULTA.

8.4 Pediatric Use

Use in pediatric patients aged 16 years and younger is not recommended because of potential safety concerns related to increased pigmentation following long-term chronic use.

8.5 Geriatric Use

No overall clinical differences in safety or effectiveness have been observed between elderly and other adult patients.

13 NONCLINICAL TOXICOLOGY

13.1 Carcinogenesis, Mutagenesis, Impairment of Fertility

Latanoprostene bunod was not mutagenic in bacteria and did not induce micronuclei formation in the *in vivo* rat bone marrow micronucleus assay. Chromosomal aberrations were observed *in vitro* with human lymphocytes in the absence of metabolic activation.

Latanoprostene bunod has not been tested for carcinogenic activity in long-term animal studies. Latanoprost acid is a main metabolite of latanoprostene bunod. Exposure of rats and mice to latanoprost acid, resulting from oral dosing with latanoprost in lifetime rodent bioassays, was not carcinogenic.

Fertility studies have not been conducted with latanoprostene bunod. The potential to impact fertility can be partially characterized by exposure to latanoprost acid, a common metabolite of both latanoprostene bunod and latanoprost. Latanoprost acid has not been found to have any effect on male or female fertility in animal studies.

13.2 Animal Toxicology and/or Pharmacology

A 9-month toxicology study administered topical ocular doses of latanoprostene bunod to one eye of cynomolgus monkeys: control (vehicle only), one drop of 0.024% bid, one drop of 0.04% bid and two drops of 0.04% per dose, bid. The systemic exposures are equivalent to 4.2-fold, 7.9-fold, and 13.5-fold the clinical dose, respectively, on a body surface area basis (assuming 100% absorption). Microscopic evaluation of the lungs after 9 months observed pleural/subpleural chronic fibrosis/inflammation in the 0.04% dose male groups, with increasing incidence and severity compared to controls. Lung toxicity was not observed at the 0.024% dose.

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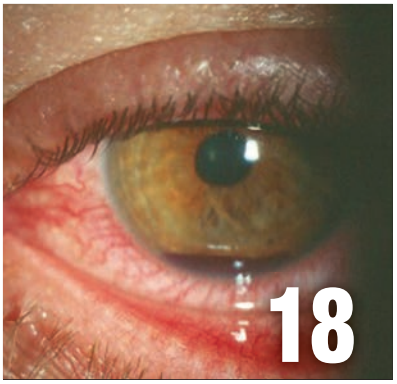

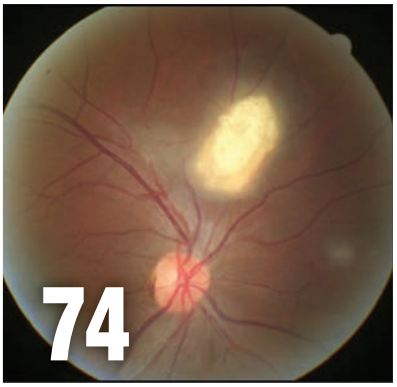

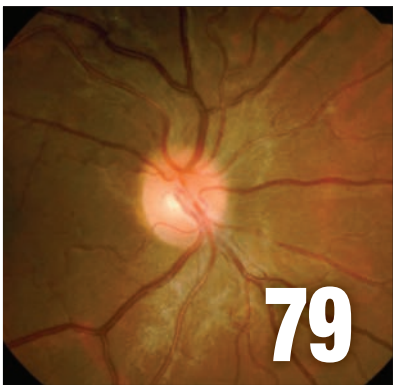

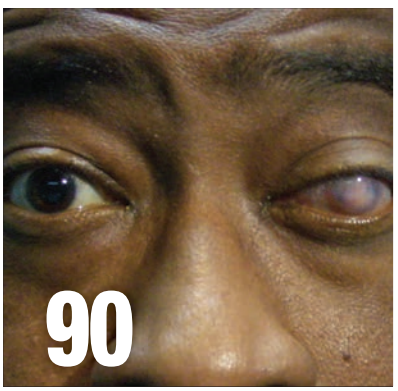

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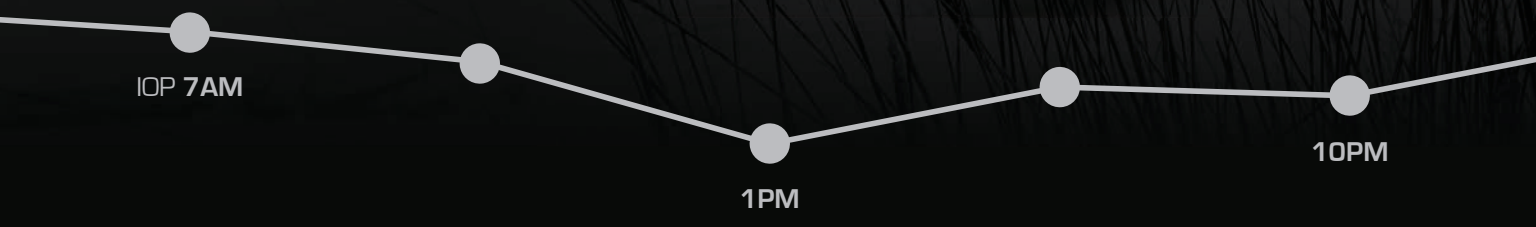

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Outlook

By Jack Persico, Editor-in-Chief

Push the Button

Glaucoma requires a leap of faith, in your clinical abilities and in the efficacy of intervention. Accept it and move on.

Before it went off the rails in its final season, ABC's *Lost* was a peerless example of great storytelling, full of puzzle-box mysteries and philosophical musings, but ultimately a human drama of survival about people from different walks of life forced to cooperate. Early in season two, "man of science" Jack Shephard and "man of faith" John Locke square off against each other when faced with the prospect of having to enter a series of numbers into a computer every 108 minutes. Once they make that fateful first push of the button, they'll be signing up for a tedious, unending series of events—or else risk their lives by abandoning the task.

Locke couldn't do it alone. He needed the man of science to do it, to validate it in the eyes of the others.

Sometimes I think about that scene as I wonder why more optometrists don't manage glaucoma. Diagnose the disease and you instantly put a lifelong burden on your patient—to commit to therapy and frequent office visits, which they might not even need. You'll put in motion a series of events from which there's really no good escape.

You and your patients will be pushing the button, pretty much forever.

With stakes that high, reluctance to manage glaucoma is understandable. But that doesn't make it excusable. The stark reality is that more people will experience vision loss if optometrists don't embrace glaucoma care more fully. There simply aren't enough ophthalmologists to do it. And because ODs do the bulk of primary care, you're the ones on the front lines seeing at-risk patients at the stage when early intervention can give them the best possible prognosis.

Our current four-part series, *Take Charge of Glaucoma*, addresses the obstacles many ODs encounter. In the May issue, Part 1 tackled the mindset needed to add glaucoma. June's installment covered diagnostic technology. This month we explain the principles of medical therapy (still the first line of defense) as part of a bigger theme detailing nearly every IOP-lowering effort used in practice or on the drawing board. Next month, the series will conclude with advice on surgical comanagement.

We hope the series leaves you with a roadmap forward. But ultimately, of course, it comes down to you. Start by acknowledging that uncertainty lies at the core of glaucoma care, even among experts. A new study in the *Journal of Glaucoma* finds only middling agreement among doctors, at the same institution using the same generally accepted protocols, in how they decide if glaucoma progression is present. In a report on a two-OD, two-MD team, the optometrists agreed 74.2% of the time; the ophthalmologists, at 78.7%, didn't do much better. And all four providers only agreed 54.4% of the time.

Bottom line: if you wait until you're dead certain you're seeing glaucoma before pulling the trigger, odds are you'll have missed your best chance at mitigating its effects.

"It's never been easy" to believe, an emotional John Locke confesses to Jack in that scene. But sometimes you have to take on a burden for the greater good. ■

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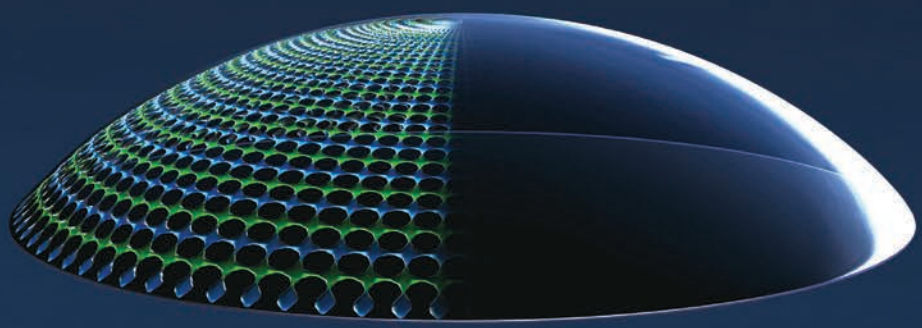

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Step Up to Glaucoma

With new therapies at our disposal and a boom in MIGS procedures, it's time to take the reins for our glaucoma patient's sake. **By Paul M. Karpecki, OD, Chief Clinical Editor**

After a 20-year drought in glaucoma drug development, we recently got two new ones—and a third is on the way. Late 2017 gave us Vyzulta (latanoprostene bunod, Bausch + Lomb) and Rhopressa (netarsudil, Aerie), and soon we expect to see Roclatan (Aerie). These agents go where no drug has gone before: the trabecular meshwork (TM). Vyzulta, dosed QHS, combines a prostaglandin analog with butanediol mononitrate, which releases nitric oxide (NO) to relax the TM and increase outflow.[1] Patients with glaucoma often have a lower concentration of NO.[2]

Rhopressa is an entirely new class of drug, a rho-kinase inhibitor, that targets the TM and is said to alter the cytoskeleton. Research shows it can lower IOP up to 5mm Hg, regardless of entering IOP. It would make an ideal second medication but may also be a great option as a primary treatment in normal tension glaucoma or early primary open-angle glaucoma.

Later this year or in early 2019, Roclatan, a combination of latanoprost and Rhopressa, may hit the market. Recent clinical FDA trial results look remarkable.

Good Things, Small Packages

Minimally invasive glaucoma surgery (MIGS) has caused a significant shift in care. Studies show these procedures have a far lower risk profile than conventional surgery and can get most patients down to one or no medications after implantation.[3,4] With four million cataract procedures performed each year and a 20% rate of concurrent glaucoma in that population, we should be seeing 800,000 MIGS procedures a year. But we have yet to get close to this number, in part because optometry is not up to speed. A recent patient referred to us for cataract surgery said her OD recommend she get a "stent for glaucoma," without elaboration. Though she has done quite well, I could have missed that brief suggestion in my pre-op assessment; ODs can and should provide more robust recommendations.

Our word goes far with patients, and it's a pivotal opportunity to save their vision on fewer medications.

Compounding Interest

I recently had a family friend's grandfather present with 0.9 cup-to-disc ratios, visual field loss and pressures of 27mm Hg and 28mm Hg—while on three different drops. He was on track for complete vision loss. Practicing in Kentucky, I performed a selective laser trabeculoplasty, which initially lowered his pressures to 19mm Hg OU. While I was pleased with this outcome, his pressures were 22mm Hg and 23mm Hg just one month later. He also needed cataract surgery, so I recommended MIGS at the same time, which lowered his pressures to 18mm Hg in each eye.

Given his fixation loss potential and his difficulty with various drops, I opted for a compounded quad drop QHS (timolol, brimonidine, dorzolamide and latanoprost) and a triple drop (timolol, brimonidine and dorzolamide) in the morning. It's been over a year now and his most recent visit is showing great stability with consistent pressures of 13mm Hg and 14mm Hg. Compounded drops give us even more flexibility to customize the regimen to the patient's needs. They should be considered sooner in patients who are having difficulty with multiple drops or who need a preservative-free formulation.

Making Contact

The Triggerfish contact lens, placed by an eye doctor and left in for 24 hours, records shape changes that correlate with IOP and visual field progression.[5] This technology can tell us the overnight IOP level and may help assess risk of future functional loss, even in situations when insufficient historical visual field information is available.[5] And as a contact lens, it's bread-and-butter optometry.

The incidence of glaucoma is increasing, and optometry must play a proactive role to manage it effectively. Knowing about new drop options, surgical advances and new diagnostics is key to elevating your care and improving patients' lives. ■

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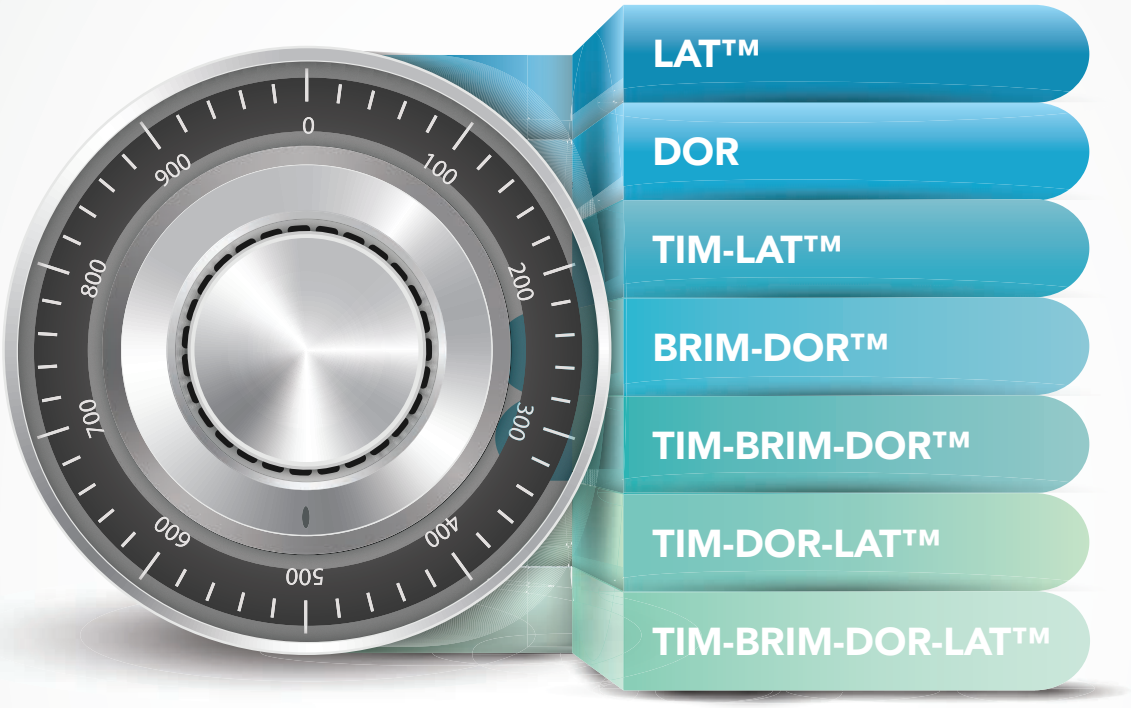

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Everyone's a Comedian

Guess I won't be quitting my day job with these wise-crackers waiting in the wings to take over. **By Montgomery Vickers, OD**

When I aimlessly wander the conference halls at CE meetings—mostly to find breakfast muffins and Diet Coke (they cancel each other out)—I am invariably recognized by a few colleagues. This is a great compliment, except in cases when they think I am Jerry Lewis.

The conversation often goes a little like this:

"You look really familiar. Are you that guy?" And I answer, "Yes, you got it. I invented meibomitis!"

When they realize the comedic brilliance of my response a few hours later, they want to tell me something funny I should use in my column. I love it. I also love colonoscopies, so… Nonetheless, I want to honor those courageous enough to pipe up by presenting to you a few of my favorite comments given to me by my fellow docs:

Mom's glasses. An OD told me his mom never wants to wear her glasses because, as she constantly reminds him, they make her eyes "look baggy." So, when he delivers her new glasses, he always brings them in a baggie. She never gets the joke, but we do.

Who's your doctor? All of us have been asked this question somewhere along the line. This doctor always answers, "My dog's vet."

Then don't do that! The patient said, "When I turn my eyes to the left I always see things."

Time to relocate. "When we moved across the country, your office was further away."

New patient wishes. "I saw this woman in Florida, and I want those same exact glasses."

Meow. "Do you have those cat contact lenses?"

Implants. "If they implant a magnet in my head, will my glasses stay up better?"

Insurance. "I just want what my insurance will cover." The doc says, "I hand them a cleaning cloth and two temples."

Macular aversion. "My dad has macular degeneration. What can I do to avoid him?"

Cool car. "We just found out our dog has cadillacs."

A thorough exam indeed. "Doctor, can you check and see if I am pregnant?"

LASIK eye. "Does LASIK have any effect on eyes?"

Phantom glasses. A patient who never got glasses tells you, "I could never wear those glasses."

Our hours. "Are you ever open when you are closed?"

The independent type. "I don't need glasses. I only wear them when I want to see something."

Drops. "Have you heard of those eye drops that help your eyes burn?"

Up to you. Doctor: "Which is better? Number one or number two?" Patient: "You tell me, you're the doctor."

Round two. Doctor: "Which is better? Number one or number two? "Patient: "Hard to beat a good number two."

It's always my fault. "I never had any eye problems until I came here."

The best no-show excuse. "I'm sorry I missed my appointment, but I had pink-eye."

Family matters. "How come my sister can see?"

Doctors, keep them coming. Feel free to come up a say, "Howdy!" anytime you want.

Anyone who knows me knows that I am very, very grateful that you take a moment to read "Chairside" and *Review of Optometry*. Have a really hilarious summer!

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A Bloody Mess

A game of racquetball goes wrong when a player winds up with a case of traumatic hyphema. **By Morgan Schuiteman, OD, and Richard Mangan, OD**

Hyphema is a common sequelae associated with blunt ocular trauma.[1,2] It is a condition in which blood is present in the anterior chamber, including when it is suspended and no clot forms (microhyphema).[1,3,4] Major complications of a traumatic hyphema include increased intraocular pressure (IOP), secondary hemorrhages, secondary glaucoma and blood staining of the cornea with decreased visual function.[1,2,4] Management of these patients is directed at accelerating absorption of the blood to prevent complications; however, there are no current guidelines for treatment.

Examination

A 23-year-old Caucasian male presented from the emergency room after being hit in the right eye with a racquetball. He complained of mild pain, blurred vision and photophobia. A computed tomography (CT) scan was performed at the hospital and no orbital fractures were noted.

He was taking Flonase (fluticasone, GlaxoSmithKline) for seasonal allergies, but no other medications. The patient did have a history of strabismus surgery as a child with no lasting visual impact.

The patient had an entering uncorrected visual acuity of counting fingers at three feet with pinhole improving vision to 20/100 OD. A dilated and unreactive right pupil was detected with pupillary testing, and extraocular muscles showed a full range of motion with the patient reporting mild pain in right gaze.

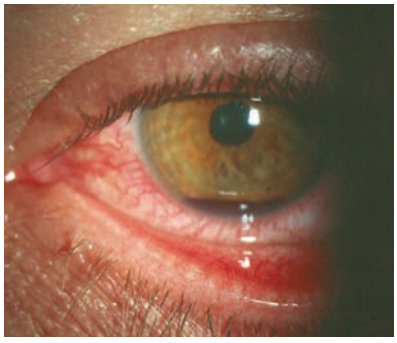

Photo: Alan Kabat, OD

Hyphema, seen here, is common after athletic injury or other trauma.

His IOPs were 14mm Hg OU. Anterior segment examination revealed 3+ diffuse conjunctival injection with two small subconjunctival hemorrhages. The cornea had 2+ diffuse microcystic edema and scattered punctate epithelial erosions. His anterior chamber was deep with 4+ cells and flare with a 0.5mm high hyphema settling at the bottom of the angle. The eye was dilated with one drop of atropine 1% in office. Posterior segment views were difficult due to the severe anterior chamber reaction, so a B-scan was performed to confirm the retina was attached.

Diagnosis

The patient was diagnosed with a traumatic hyphema and secondary iridocyclitis. He was placed on atropine 1% BID OD and Pred Forte (prednisolone acetate, Allergan) in his right eye every two hours. He was instructed to limit physical activity, sleep with his head elevated and avoid aspirin products. He returned the next day with improved vision of 20/30 but had a persistent hyphema and anterior chamber reaction. A posterior view was obtained and a three disc diameter area of *commotio retinae* was noted in the superior nasal retina. Treatment was continued as directed and the patient was followed closely over the next nine days until the hyphema and anterior chamber reaction resolved. At that time, the steroid was tapered appropriately with no rebound inflammation. At the one-month mark, gonioscopy showed no angle recession was noted. Vision returned to 20/20 and he had no lasting ocular damage.

Discussion

The estimated annual incidence of hyphema is 17 per 100,000, with males being impacted more often than females 3:1.[1,4-7] Athletic injuries have become a common source for hyphemas, with a study showing that 39.2% of hyphemas occur from athletic injuries whereas work-related injuries only accounted for 9.9%.[8] Rupturing of the iris and ciliary body vessels leading to a hyphema is thought to be caused either by compressive forces creating damage to the angle or rapidly rising pressure within the vasculature.[1,9]

Hyphemas are graded clinically by the amount of blood in the anterior chamber. If the blood cells are still suspended in the chamber, it is referred to as a microhyphema. Once the blood starts to settle in the anterior chamber, the hyphema is graded 1 to 4. A grade 1 hyphema is defined as blood filling less than

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References: **1.** Alcon sales data on file. **2.** Wirtitsch MG, Findl O, Menapace R, et al. Effect of haptic design on change in axial position after cataract surgery. *J Cataract Refract Surg.* 2004;30(1):45-51. **3.** Visser N, Bauer NJ, Nuijts RM. Toric intraocular lenses: Historical overview, patient selection, IOL calculation, surgical techniques, clinical outcomes, and complications. *J Cataract Refract Surg.* 2013;39(4):624-637. **4.** Potvin R, Kramer BA, Hardten DR, Berdahl JP. Toric intraocular lens orientation and residual refractive astigmatism: An analysis. *Clin Ophthalmol.* 2016;10:1829-1836.

one third of the chamber; one third to one half is a grade 2; and greater than one half is a grade 3 hyphema. When the blood completely fills the anterior chamber, it is considered a grade 4 hyphema. When a grade 4 hyphema turns dark it is considered an "eight-ball hyphema" and carries a much worse prognosis than a bright red grade 4 hyphema.[1]

Investigators estimate that about 30% of all traumatic hyphemas will present with an increase in IOP, which can be a significant risk factor for vision loss.[10] Increased IOP is caused by anterior synechiae formation, increased outflow resistance and fibrosis of the trabecular meshwork. Secondary glaucoma may develop weeks to years after a hyphema and occurs in up to 20% of cases.[1,4] Patients with larger hyphemas have an increased risk of secondary glaucoma; however, any amount of blood in the anterior chamber can be associated with elevations in IOP.[4]

Secondary hemorrhages usually occur two to seven days after the initial trauma and are a result of lysis and retraction of the blood clot and fibrin at the ruptured blood vessel. Patients have a 35% chance of having a rebleed during this time frame, and it can be noted clinically by an increase in the size of the hemorrhage or by fresh red blood overlying the darker clot.[4,9,11] Secondary hyphemas can increase the risk of IOP spikes and corneal blood staining. The incidence of corneal blood-staining varies between 2% and 11% with an increased risk in patients with larger hypehmas, rebleeding, prolonged clot duration, sustained increased IOP and previous corneal endothelial cell dysfunction.[1,4,11] Research shows that patients with an IOP equal to or greater than 25mm Hg for six or more days are at a significantly higher risk of having corneal blood staining and therefore reduced visual function.[1,4,11]

Treatment

Numerous controversies in management of hyphemas exist, and standardized guidelines for treatment have yet to be determined. However, the most important factor in treating traumatic hyphemas is to stabilize the eye and accelerate the absorption of the blood to prevent complications. Patients should be advised of quiet ambulation and resting at a 30-degree angle to promote settling of the hyphema. Patients should avoid any substances that will delay clotting, including aspirin.

Cycloplegics are often used in treatment of a traumatic hyphema (atropine 1% BID). They help improve comfort in patients with a secondary iridocyclitis, decrease the risk of posterior synechiae and theoretically reduce secondary hemorrhages by immobilizing the iris and ciliary body and stabilize the blood-aqueous barrier.[1] Studies show, however, that cycloplegics do not improve visual outcomes or reduce the risk of rebleeding.[4]

Corticosteroids, such as prednisolone acetate 1% QID to Q2H, are also important tools when managing hyphemas. Steroids work by stabilizing the blood-aqueous barrier and decreasing the amount of plasminogen, a mediator of fibrinolysis, into the anterior chamber. It can also prevent posterior synechiae formation and reduce concurrent inflammation.[1,4] One study shows patients treated with topical steroids had a 5% rebleeding rate whereas the non-treated group had a 12% incidence of rebleeding.[12]

Ocular hypotensive agents are an indicated treatment in patients who have an increase in IOP.1 Topical beta-blockers (timolol 0.5%),

alpha agnoists (brimonidine 0.1%) and carbonic anhydrase inhibitors (brinzolamide 1%) can be used to control elevated IOPs. Prostaglandins are avoided due to the amount of inflammation already associated with the traumatic event. Oral acetazolamide is also avoided as it promotes sickling of erythrocytes in patients with the sickle-cell trait.[1]

The last and most controversial treatment option is antifibrinolytic agents such as aminocaproic acid. These systemic medications can significantly lower the rate of rebleeding by stabilizing the interface between the clot and vessel wall by inactivating plasmin and preventing clot lysis.[1,4] Studies show a decrease in rebleeding rates from 4% to 33%; however, aminocaproic acid can cause nausea and vomiting and also prolongs clot duration, which could put patients at an increased risk for prolonged IOP elevation or blood staining.[1,13] ■

Dr. Schuiteman finished her primary care residency last July at Indiana Optometry. She currently practices in South Bend, IN.

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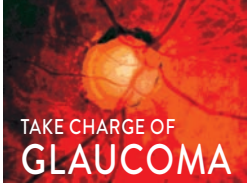
100 WAYS TO LOWER IOP

INDEX TO TREATMENTS BY CATEGORY AND PAGE NUMBER

DE Device, implanted or worn
IS Incisional surgery
ID Injectable drug, sustained release
LP Laser procedure
LM Lifestyle modification
MI Minimally invasive glaucoma surgery
TD Topical drug, conventional
TS Topical drug, sustained release
ST Systemic therapy



Part 3: Medications

24TH ANNUAL GLAUCOMA REPORT

100 WAYS TO LOWER IOP

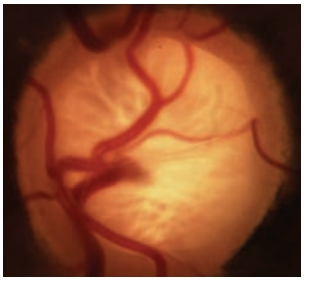
PEER • REVIEWED • PRA • ARTICLE

A Guide to Applying IOP-lowering Drugs

Several classes of pharmaceutical agents can address glaucoma patients' needs. Here's a primer. **By Michael Dorkowski, OD, Jim Williamson, OD, and Andrew Rixon, OD**

Experts have yet to reach consensus on a universally accepted etiology of glaucoma. However, they do agree on this: lowering intraocular pressure (IOP) is the only modifiable risk factor for slowing its progression. Topical pharmacotherapy is the traditional first-line approach, and the options are more abundant than ever.

This article explores the many meds ODs can employ, with advice on when to use which therapy for which patients.



Glaucomatous cupping with characteristic loss of neuroretinal rim, lamina and alteration of the vasculature.

Early Autonomics

Medications that act on the autonomic nervous system (i.e., cholinergics and adrenergics) have been a basis of glaucoma therapy since the 1800s.[1,2] Cholinergics induce miosis, which stretches and stimulates the trabecular meshwork (TM) to increase aqueous outflow there and to Schlemm's canal.[3] Members of this class include pilocarpine, carbachol, physostigmine, neostigmine and ecothiophate. Of those, only pilo is still in routine use. Treatment exhibits a dose-related response with a decrease in IOP of about 20% when prescribed QID.[4]

Though effective, cholinergic use is limited by its ocular and systemic side effects. These include ciliary muscle spasm (with associated headache and induced myopia), miosis, corneal toxicity, redness, uveitis, possible cataract formation, respiratory depression and gastrointestinal distress. Further, newer medications have greater IOP-lowering efficacy, which leaves cholinergics reserved for specific cases where the miotic effect may have an added benefit, such as acute angle closure.

Adrenergic agonists, on the other hand, impact the alpha or beta adrenergic receptors (or both if non-selective). Epinephrine, the primary non-selective agent, reduces IOP by first decreasing aqueous production and then increasing outflow through the TM.[5] Unfortunately, it has limited application due to significant systemic side effects. Dipivefrin, a prodrug of epinephrine developed in the 1970s, allows use of much lower concentrations of the parent compound, with fewer systemic effects.[6] Nonselective adrenergics see minimal use today except in cases where other drugs may be contraindicated.

Beta Blockers

Adrenergic antagonists, or beta (β)-blockers, inhibit aqueous production and represent the standard by which new medications are compared. Specifically, β-blockers reduce ultrafiltration, which limits the availability of aqueous humor substrate available for transmission into the posterior chamber.[1,7,8] These medications can be nonselective, meaning they inhibit both isoforms of the β-adrenergic receptors (β1 and β2), or cardioselective, which have much greater affinity for the β1 receptor. β2 is the eye's predominant adrenoreceptor, so nonselective agents will have a greater impact on IOP control.[9,10]

Tables: Bruce Onofrey, OD, RPh

Table 1. Cholinergic Agents

Brand Name	Generic & Concentration	Mechanism of Action	Dosing	Typical IOP Reduction	Size	Side effects	Warnings
Isopto Carpine (Novartis)	Pilocarpine 1%, 2%, 4%	Increased trabecular outflow; direct acting.	BID-QID	15% to 25%	15mL	Headache, blurred vision, myopia, retinal detachment, bronchiole constriction, narrowing of angle.	RD symptoms, angle closure, shortness of breath.
Pilopine HS Rx (Novartis)	Pilocarpine HCl 4%; oph gel		Qhs	20% to 30%	4g	Lacrimation, discomfort, headache, ciliary spasm, conjunctival vascular congestion, keratitis, myopia, corneal granularity, reduced nighttime acuity; retinal detachment, lens opacity (rare).	
Phospholine Iodide (Wyeth)	Echothiophate Iodide 0.125%	Increased trabecular outflow; indirect acting.	QD-BID	15% to 25%	5ml, 10ml	Same as above, plus cataractogenic iris cysts in children, pupillary block, increased paralysis with succinylcholine.	Same, plus avoid prior to general anesthesia.

Table 2. Beta Blockers

Brand Name	Generic & Concentration	Mechanism of Action	Dosing	Typical IOP Reduction	Size	Side effects	Warnings
Betagan (Allergan)	levobunolol hydrochloride 0.25%, 0.50%	Decrease aqueous production	QD-BID	20% to 30%	5mL, 10mL, 15mL	Bronchospasm, bradycardia, hypotension, elevated triglycerides and decreased HDL= increased CV risk, CNS confusion, lethargy, depression, impotence, masked hypoglycemia, exacerbates myasthenia gravis.	Monitor for SOB, hypoglycemia, altered blood lipids, angina, dizziness.
Betimol (Akorn)	timolol hemihydrate 0.25%, 0.5%		QD-BID	20% to 30%	5mL, 10mL, 15mL		
Betoptic-S (Novartis)	betaxolol hydrochloride 0.25%	Decrease aqueous production; beta 1 selective blocker	BID	15% to 20%	5mL, 10mL, 15mL	Fewer pulmonary, side effects, otherwise same as timolol. Less effective than timolol.	
Istalol (Bausch + Lomb)	timolol maleate ophthalmic solution 0.5%	Decrease aqueous production	QD	20% to 30%	5mL	Bronchospasm, bradycardia, hypotension, elevated triglycerides and decreased HDL= increased CV risk, CNS confusion, lethargy, depression, impotence, masked hypoglycemia, exacerbates myasthenia gravis.	
Optipranolol (Bausch Health)	metipranolol 0.3%		BID	20% to 30%	5mL, 10mL		
Timoptic (Merck) Timoptic in Ocudose (Bausch + Lomb)	timolol maleate 0.25%, 0.50%		QD-BID	20% to 30%	2.5mL, 5mL, 10mL, 15mL		
Timoptic XE (Merck)	timolol maleate gel-forming solution		QD	20% to 30%	5mL		

Table 3. Carbonic Anhydrase Inhibitors

Brand Name (Manufacturer)	Generic & Concentration	Mechanism of Action	Dosing	Typical IOP Reduction	Size	Side effects	Warnings
Azopt (Novartis)	brinzolamide 1%	Decrease aqueous production	BID	15% to 20%	10mL, 15m	Slight risk of parasthesia, metallic taste, nausea, malaise, depression, loss of libido, hypokalemia, aplastic anemia, metabolic acidosis, kidney stones, sulfonamide sensitivity.	Avoid in sulfonamide allergies, sickle cell and renal disease. Side effects more common with the oral agents.
Diamox (Teva) tablets	acetazolamide		BID-QID	15% to 20%	125mg, 250mg		
Neptazane (Fera, Perrigo) tablets	methazolamide		BID-TID	15% to 20%	25mg, 50mg		
Trusopt (Merck)	dorzolamide 2%		BID-TID	15% to 20%	10mL		

Table: Bruce Onofrey, OD, RPh

Topical β-blockers include timolol, levobunolol, metipranolol, carteolol and betaxolol. Only betaxolol is cardioselective—which makes it helpful in certain contraindications, but it may be less effective at reducing IOP.[11] β-blockers reduce IOP by 20% to 30% and may be dosed twice daily. They can also be dosed once daily, particularly when using gel-forming solutions due to their increased ocular contact time.[12,13] Adrenergic antagonists may have less impact during sleep, so care should be taken when β-blockers are dosed close to bedtime, especially if prescribed as a once daily regimen.[14,15] If patients are taking systemic β-blockers, the ocular hypotensive effect of topical β-blockers is reduced, and other classes of topical medications could be considered.[16]

Although this class of medication should mostly be avoided in pulmonary or cardiac conditions, in select cases it may be reasonable to consider beta-blocker therapy. However, this should be done with the consent of the appropriate specialist (cardiology or pulmonary).

Generally, however, β-blocker use should be avoided in those with atrioventricular block, sinus bradycardia, and obstructive pulmonary disease.[17]

Carbonic Anhydrase Inhibitors

Researchers have acknowledged the ability of oral carbonic anhydrase inhibitors (CAIs) to decrease IOP since the 1950s.[18] They accomplish this by suppressing aqueous production.[18] However, systemic side effects (such as fatigue, gastrointestinal disturbances and paresthesia) limit their chronic use in glaucoma.[18] Oral CAIs are still used in cases when topical CAIs cause hypersensitivity or when the use of drops is precluded, as well as in cases of acute angle closure.[18] Attempts to formulate a topical variety succeeded with the introduction of dorzolamide in the mid-1990s and, soon after, brinzolamide.[19]

There are at least seven different isoenzymes of carbonic anhydrase (CA), with CA-II in the ciliary processes being predominately involved with aqueous production.[18] Both dorzolamide and brinzolamide are potent inhibitors of this isoenzyme, but they have several clinically relevant differences. For example, dorzolamide has a 5.6 pH while brinzolamide has a 7.5 pH. Additionally, brinzolamide is available as a suspension. These are just some of the properties that can account for the products' individual side effects, which can include stinging in the case of dorzolamide and blurred vision in the case of brinzolamide.[19]

Table 4. Alpha Agonist Clonidine Derivatives

Brand Name (Manufacturer)	Generic & Concentration	Mechanism of Action	Dosing	Typical IOP Reduction	Size	Side effects	Warnings
Alphagan P (Allergan)	brimonidine tartrate w/Purite preservative 0.1%, 0.15%	Decrease aqueous production and increase uveoscleral outflow	BID-TID	up to 26%	5mL, 10mL, 15mL	Dry mouth, hypotension, bradycardia, follicular conjunctivitis, ocular irritation, pruritis, dermatitis, conjunctival blanching, eyelid retraction, mydriasis, drug allergy (lopidine > Alphagan P).	Monitor for shortness of breath, dizziness, ocular redness and itching, fatigue.
Iopidine (Novartis)	apraclonidine 0.5%	Decreases aqueous production	BID-TID	up to 25%	5mL, 10mL		

Table: Bruce Onofrey, OD, RPh

The Straight Dope

Medical marijuana has been making big news of late, thanks to a number of legislative changes in the United States. Although it's been researched as a method to reduce IOP since the 1970s, investigators have only found it capable of lowering IOP for brief periods of time. That, coupled with its rather hefty side-effect profile, makes it a poor candidate for treatment. However, research says it may have a role to play for "end-stage glaucoma patients who have failed maximal medical therapy and surgery or who are poor surgical candidates."

Sun X, Xu C, Chadha N,et al. Marijuana for glaucoma: a recipe for disaster or treatment? Yale J Biol Med. 2015 Sep; 88(3): 265-9.

Though the drugs FDA-labeled for TID dosing, some practitioners opt for BID administration. For brinzolamide, phase III trials report clinically equivalent IOP reductions with either BID or TID regimens.[20] Others report no statistically significant differences between BID or TID dosing with dorzolamide.[18] Alternatively, others advocate TID dosing for monotherapy and BID dosing when used as adjunctive therapy.[21]

Alpha Agonists

Clonidine represented the earliest alpha agonist (AA) effective for lowering IOP. However, even in topical form it produced profound systemic side effects of bradycardia, sedation and hypotension.[22] Apraclonidine's amide group substitution decreased blood-brain barrier penetration, and thus side effects. Unfortunately, its diminished efficacy over time and an increased incidence of ocular allergy limits it to short-term use.

Introduced in 1997, brimonidine is a potent AA with 32 times more selectivity for β2 adrenoreceptors than apraclonidine.[22] It lowers IOP through a dual mechanism of decreasing aqueous production and increasing uveoscleral outflow.[23] Like its predecessor, it has a notable allergy rate (up to 20%), which may occur up to eight months after initiation of therapy.[22] Three formulations have been developed, each with similar efficacy: 0.2% with benzalkonium chloride (BAK); and 0.15% and 0.1% with the preservative Purite.[24] Allergan has since discontinued the BAK-preserved formulation.

Though side effects mostly correlate with prostaglandin use, the literature shows brimonidine-induced anterior uveitis.[25] Duration ranged from seven days to five years with a mean of nearly 20 months.

Prostaglandin Analogs

These provide the most robust IOP reduction of all drops by enhancing uveoscleral aqueous outflow. Prostaglandin analogs (PGAs) bind to receptors in the

Table 5. Prostaglandin Analogs

Brand	Generic & Concentration	Mechanism of Action	Dosing	Typical IOP Reduction	Size	Side effects	Warnings
Lumigan (Allergan)	bimatoprost 0.03%	Increased uveoscleral outflow	Qhs	27% to 33%	5mL	Hyperemia, iris pigment, CME, Hypertrichosis, conjunctival injection, keratitis, uveitis, ocular pain	May darken eye color
Travatan Z (Novartis)	travoprost 0.004%		Qhs	Same	5mL		
Xalatan (Pfizer)	latanoprost 0.005%		Qhs	25% to 32%	2.5mL		
Zioptan (Akorn)	tafluprost 0.0015%	Same	QD	225 to 29%	0.3mL	Conjunctival hyperemia, stinging, pruritus, cataract, dry eye, increased ocular pigmentation, blurred vision, macular edema, headache, cold, cough, urinary tract infection.	Risk of macular edema
Prostaglandin/Nitric oxide producer							
Vyzulta (Bausch + Lomb)	latanoprostene bunod 0.024%	Increase uveoscleral and trabecular outflow	QD	34.6%	5mL	Hyperemia, eye pain, hypertrichosis	No systemic effects

Table: Bruce Onofrey, OD, RPh

ciliary body and induce smooth muscle relaxation, and alter the extracellular matrix within the ciliary muscle to increase aqueous outflow through uveoscleral routes.[26] Dosed once per day, commonly at bedtime, PGAs yield a 30% to 35% IOP reduction; options include latanoprost, bimatoprost, travoprost and tafluprost (supplied in a single dose, preservative-free option).[26] The most common side effect of PGAs is conjunctival hyperemia, often in the first few weeks after initiation of therapy.[26] Ocular irritation, exacerbation of existing inflammatory conditions (macular edema, iritis), atrophy of the periorbital fat pad, pigmentation of periocular skin, eyelashes and iris and hypertrichosis may also be seen.[26]

A new variant in the prostaglandin analog category is latanoprostene bunod 0.024%. This compound has a dual mechanism: increasing uveoscleral outflow and enhancing trabecular meshwork outflow through the impact of nitric oxide.[27] The eye breaks down latanoprostene bunod twice to yield the active components latanoprost acid and nitric oxide.[28] The latter impacts a signaling pathway that relaxes contractile components in the TM, which increases outflow.[28] The additional impact drops IOP >1mm Hg vs. latanoprost alone across multiple time points, with total IOP reduction ranging from 7.5mm Hg to 9.1mm Hg. Side effects with latanoprostene bunod were comparable to those

Table 6. Rho-kinase Inhibitors

Brand Name (Manufacturer)	Generic & Concentration	Mechanism of Action	Dosing	Typical IOP Reduction	Size	Side effects	Warnings
Rhopressa (Aerie)	netarsudil 0.02%	Increase trabecular outflow/decrease episcleral venous pressure and decrease aqueous production	QD	25% to 30%	2.5mL	Hyperemia, conjunctival hemorrhage, corneal verticillata	Significant red eye, no systemic effects

Tables: Bruce Onofrey, OD, RPh

Table 7. Hyperosmotic Agents

Brand Name (Manufacturer)	Generic	Mechanism of Action	Warnings
Glycerin (numerous mfgs.)	glycerol	osmotic diuretic	May be associated with neurotoxicity and reduced phenylbutyrate absorption in some patients.
Osmitrol (Baxter)	mannitol	osmotic diuretic	May impair renal function, further aggravate pre-existing hemoconcentration

of latanoprost alone, with possibly less periocular pigmentation and hypertrichosis, according to investigators.[28,29]

ROCK Inhibitors

In a normal eye, the main drainage pathway for aqueous humor is the TM. Resistance to aqueous humor through this structure is increased in patients with glaucoma, raising IOP.[30] Until recently, glaucoma medications failed to target this structure. That changed in 2017 with the introduction of Rhopressa (netarsudil 0.02%, Aerie), a rho-kinase (ROCK) inhibitor.

Rho-kinase is widely expressed in many tissues, including the TM, where it promotes the assembly of actin stress fibers and regulates cell contraction.[31] ROCK inhibitors increase aqueous outflow by decreasing actin and myosin-driven cellular contraction and reducing extracellular matrix protein production.[32] Rhopressa also has inhibitory action against norepinephrine transporter (NET), making it a ROCK/NET inhibitor. The NET mechanism may be the result of reduced blood flow to the ciliary body through norepinephrine-induced vasoconstriction, which results in decreased aqueous production.[33] In addition, netarsudil also decreases episcleral venous pressure (EVP), thus providing multiple avenues for IOP reduction.[32] The most common ocular side effect is ocular hyperemia (about half of treated patients), which for the most part is mild, transient and self-resolving.[32]

Of note, rho itself plays an important role in axon growth and guidance, as well as the regulation of neuronal survival and death.[34] Researchers found that, following optic nerve injury, topical application of netarsudil reduces retinal ganglion cell death and promotes axonal regeneration.[34]

Table 8. Combination Agents

Brand Name (Mfg.)	Generic
Available in US	
Combigan (Allergan)	timolol 0.5%, brimonidine 0.2%
Cosopt/Cosopt PF (Akorn)	timolol 0.5%, dorzolamide 2%
Roclatan (Aerie) *FDA approval anticipated late 2018/early 2019*	netarsudil 0.02%, latanoprost 0.005%
Simbrinza (Alcon)	brimonidine 1%, brinzolamide 0.2%
Available outside US	
Xalcom (Pfizer)	latanoprost, timolol
Ganfort (Allergan)	bimatoprost, timolol
DuoTrav (Novartis)	travoprost, timolol
Azarga (Novartis)	brinzolamide, timolol

Table 9. Investigational Sustained-release Drug Delivery Methods and Devices

Modality	Product	Mechanism of Action	Discussion
Punctal plugs	OTX-TP (Ocular Therapeutix)	Intracanalicular punctal plug embedded with travoprost.	Sustained release up to 90 days, marked with fluorescein to enhance visualization. Exhibited IOP reduction (less than timolol BID) with some reduced effect over time.
	Evolute (Mati) Therapeutics)	Silicone plug fit at punctal opening containing drug-eluting core loaded with latanoprost.	Unidirectional emission of medication reduces systemic effects. Achieved 5mm Hg to 6mm Hg in IOP reduction over 12 weeks and >90% retention rate.
Contact lenses	N/A	Silicone hydrogel contact lens embedded with latanoprost.	In animal studies, IOP control was greater than that of topical latanoprost over one month of therapy.
	N/A	Nanoparticle polymeric ring embedded in lens with hyaluronidase for comfort and release of timolol.	Effective IOP reduction in animal studies but some altered lens modulus and oxygen permeability of contact lens.
Fornix-based ocular surface ring	BIM-ring (Allergan)	Fit under eyelids and placed in fornices by physician.	Sustained IOP reduction for up to six months with >90% retention. All patients were aware when device dislodged.
Ocular surface depot device	TODDD (Amorphex Therapeutics)	Elastomer material resting on ocular surface under superior lid. Achieves movement and tear exchange like a contact lens and can be placed and removed by patient.	Sustained drug release over several months, possibly up to 180 days. Multiple medications being studies individually and in combination including timolol, prostaglandins, pilocarpine, brimonidine and non-ocular hypotensives.
Gel formulations	SoliDrop (Selkie Therapeutics, Otero Therapeutics)	Drug in polymer microspheres with a hydrogel carrier altered by body temperature. Forms pliable, glue-like state in inferior fornix; gradually breaks down to emit medication.	Single treatment with gel formulation of brimonidine in animal studies showed comparable IOP-lowering effect as compared with topical brimonidine dosed BID over 28 days. Also, may reduce systemic absorption of medication.
	Durasite ISV-215 (InSite Vision)	Bimatoprost mixed in Durasite copolymer.	Single dose of Durasite/bimatoprost compound gives effective intraocular concentrations at various time points. Could allow for decreased dosing with lower concentrations for better compliance and fewer side effects.
Intracameral implants	Bimatoprost SR (Allergan)	Intracameral injection of biodegradable implant containing bimatoprost.	Human studies exhibit a 7mm Hg to 9.5mm Hg reduction in IOP over four months with active but reduced effects at six months.
	iDose (Glaukos)	Non-degradable, reusable titanium implant filled with travoprost.	Early results show ~30% IOP reduction over 12 months. Reduced surface and adnexa side effects vs. topicals.
	ENV-515 (Envisia/ Aerie)	Biodegradable nanoparticle technology for intracameral injection of travoprost.	Achieved 11-month sustained IOP reduction with results comparable to topical latanoprost or timolol. Hyperemia side effects were associated with initial administration.
Subconjunctival devices and injections	Durasert (EyePoint Pharma)	Bioerodable insert loaded with latanoprost administered with subconjunctival injection.	Evaluating 12-month efficacy. Current focus on use of anti-inflammatories on the platform, but possibility of neuroprotective agent being considered.
	Eye-D (BioLight)	Controlled-release latanoprost insert, injected subconjunctivally.	Achieved 24% IOP reduction at 12 weeks post-implantation.
	Peregrine Ophthalmics	Nano-liposomal based latanoprost subconjunctival injection.	Small pilot study on human subjects showed effective IOP response for three months with single injection.
	IBI-60089 (EyePoint)	Sustained-release latanoprost injection in Verisome delivery vehicle.	May be able to achieve therapeutic levels for a duration of six months from a single injection.
	GB-201, GB-202, GB-203 (GrayBug)	Drug-encapsulated microparticle; ability to be injected into multiple intraocular sites.	Company is evaluating prodrug delivery of single agent (GB-201), dual mechanism agents (GB-202) and multiple agents (GB-203) for IOP reduction and neuroprotection.
Suprachoroidal injection	Microneedle (Clearside Biomedical and Santen)	33-gauge needle used to deliver sustained-release drug to the supraciliary space.	Rabbit study of a single injection of brimonidine using a microsphere vehicle showed a 6mm Hg drop in IOP that was sustained over one month.
Trans pars plana delivery	Replenish MicroPump (Replenish)	Surgically implanted refillable reservoir with cannula to anterior chamber (like a glaucoma drainage device).	Effective safety profile in animal studies. Refilled with 31-gauge needle. Also has applications for treatment of retinal disease.

Getting in the Mix

Sometimes patients need more than what off-the-shelf products offer. Patient sensitivity or ocular surface toxicity from chronic use of preservatives may lead to a search for alternatives. Some compounded drugs are available preservative free. If compliance is an issue, these mixtures can contain up to four medications in a single bottle. Further, many fixed combinations have been used effectively outside of the United States, but are not currently approved by the FDA; compounding gives US doctors access to those regimens. If cost is a limitation, compounding could be an added advantage. While the compounded product is often not covered by the patient's insurance plan, the price of one or two combination products may be more cost effective than that of multiple single-medication options, even generic formulations. Two compounding pharmacies offer multiple pre-set variations: Simple Drops from Imprimis Pharmaceuticals and the Omni prodcut line from Ocular Science. Additional customization is possible from each, too.

Although the individual agents are FDA approved, particular combinations may not be. It is up to the practitioner to weigh the risks and benefits of compliance.

COMPOUNDED GLAUCOMA PRODUCTS:

Simple Drops (Imprimis Pharmaceuticals)

All available preservative free.

- Tim-Lat PF (timolol/latanoprost)
- Brim-Dor PF (brimonidine/dorzolamide)
- Tim-Brim-Dor PF (timolol/brimonidine/dorzolamide)
- Tim-Dor-Lat PF (timolol/dorzolamide/latanoprost)
- Tim-Brim-Dor-Lat PF (timolol/brinzolamide/dorzolamide/latanoprost)
- Dor-Tim (dorzolamide/timolol)
- Dorzolamide PF
- Latanoprost PF

Omni Drops (Ocular Science)

Contain benzalkonium chloride, but in lower concentrations (0.001%) than common generic formulations (0.002%).

- Timolol/latanoprost
- Timolol/brimonidine/dorzolamide (AM formula)*
- Timolol/brimonidine/dorzolamide/latanoprost (PM formula)*

** Intended to be used in unison, with the AM version lacking the prostaglandin, which is commonly dosed only once per day.*

Fixed Combination Products

Despite the efficacy of these individual products, it is common for many glaucoma patients to require multiple therapies to control their condition.[35] When dual therapy is necessary it may be best to offer the same dispenser, promoting increased compliance and reducing impact on the ocular surface from toxicity.[36] Timolol pairs with brimonidine, (as Combigan, Allergan) and dorzolamide (as Cosopt, Akorn), both dosed twice per day, with a preservative-free option available for

the timolol-dorzolamide product. Another twice daily combination—Simbrinza—couples brimonidine and dorzolamide. The fixed combinations show good efficacy both as primary therapy and in addition to PGAs. Side effects are comparable to the individual components.

Adherence/Patient Perception

Although the decision to recommend treatment can be complex and is contingent on many factors, once the doctor and patient agree to initiate therapy, certain background facts must be acknowledged and basic tenets must be employed.

Visual field progression and disease severity is linked to poor adherence.[37] Patients with chronic medical conditions use, on average, 30% to 70% of their prescribed medication doses, and 50% discontinue their medications within the first few months of therapy, according to one study.[38] Adherence to glaucoma medications is similarly poor to that of other chronic conditions.[38] Accordingly, there may exist an efficacy-effectiveness gap where, although it has been proven that topical medications are efficacious in large clinical studies, in practice they can be ineffective due to patient noncompliance.[39]

Major factors that contribute to poor adherence in glaucoma are, among others; side effect profile of medications, cost of therapy, patient education and the doctor-patient relationship.[40] Additionally, the disease may be asymptomatic until late in its course, with lack of awareness of visual field loss.[41]

An additional barrier to adherence is difficulty with drop instillation and dosing schedule.[42] In fact, up to 80% of patients contaminate their drops by touching their face, up to 61% do not instill exactly one drop and, most critically, up to 37% miss the eye with the drop.[43] The practitioner should never assume the patient is proficient with drop instillation. Prior to initiating drop therapy, teach the patient how to appropriately instill drops and have them successfully demonstrate instillation prior to leaving the office. This can be efficiently delegated to an optometric technician and reinforced with an educational handout on technique (printable PDF at www.glaucoma.org/treatment/eyedrop-tips.php) as well as the use of videos such as one produced by the Glaucoma Research Foundation (www.glaucoma.org/treatment/putting-in-eye-drops.php).

If the patient has significant difficulty instilling eye drops, a mechanical dosing aid may improve the likelihood of success. Adherence to the correct dosing schedule improves with the use of automated telecommunication-based reminders, smartphone and tablet based reminder apps.[44,45] A combination of in-office education, goal setting, simplified drop regimen and technology should be embraced to improve adherence.[46] Addressing known barriers to medication adherence is a necessary first step toward success.

And Now For Something Completely Different

Medications have been the backbone of glaucoma management for well over a century, and development of new drugs continues unabated. But some researchers are looking for alternatives beyond the bottle. Glasses embedded with an electromagnetic coil, coupled with a contact lens containing a trace of gold, may one day help to lower IOP. Developed by a company called Bionode, the combo is designed to generate an electric current that flows through the ciliary muscles to stimulate the natural drainage pathway and decrease IOP.

American Academy of Ophthalmology. Eyeglasses may one day treat glaucoma. www.aao.org/eye-health/news/eyeglasses-for-glaucoma-treatment. Accessed July 3, 2018.

Dropping the Pressure

IOP is the only known modifiable glaucomatous risk factor and lowering it has undeniably proven to reduce the risk of disease progression.[47,48] Lowering IOP effectively thus becomes the goal when managing patients with glaucoma. After the disease is accurately classified, a therapeutic goal is typically set. Preferred practice patterns in the United States suggest the use of a target IOP range that the clinician believes will prospectively reduce the patients' lifetime risk of blindness while concurrently minimizing treatment-related burden.[49–52]

Although multiple methodologies can help determine an initial target IOP (threshold IOP, calculated, one size fits all) the simplest and most evidence-based method is to reduce the IOP by a percentage from the baseline peak diurnal IOP at which damage to the optic nerve is occurring. IOP is dynamic and exhibits short-term and long-term fluctuations, which makes currently available tonometry not perfectly reproducible. Therefore, it is recommended that the clinician obtain multiple IOP readings at different times of day prior to initiating treatment to attempt establishing a diurnal peak IOP from which to base the target range.[53-55] However, short of obtaining multiple 24-hour IOP curves, it is unlikely a true peak will ever be captured. The clinician will ultimately need to gauge successful treatment by reducing the rate of progression on structural and functional testing.[48,56,57]

Recommended target IOP percentage reductions range from 20% to 50% depending on the condition (i.e., glaucoma suspect, ocular hypertension, normal tension glaucoma, high tension glaucoma, angle closure glaucoma) baseline disease severity, and life expectancy.[58-61]

Getting Started

PGAs are approved for first-line treatment in the United States and are more effective in lowering IOP, have a relatively limited side effect profile and are dosed less frequently (once daily) than other classes of medication.[62,63]

Once a specific agent is selected, the patient should be seen again in no more than four weeks to gauge the effectiveness of the drop, to head off any adverse events that may arise, ensure patient adherence and reinforce the value of treatment.

Ultimately, proper education and vigilant follow-up with structural and functional testing will help minimize vision loss. ■

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PEER-REVIEWED PRA ARTICLE

Glaucoma Care with Laser Precision

Trabeculoplasty, performed early in the disease course, may prevent more invasive surgical intervention for certain glaucoma patients. **By Anthony DeWilde, OD**

With today's advances, glaucoma patients have any number of therapy options, ranging from medical management to highly invasive surgery. But for many patients with open-angle glaucoma (OAG), a simpler option such as laser trabeculoplasty (LTP) can often be the best management strategy. The procedure lowers intraocular pressure (IOP) by treating the trabecular meshwork (TM).

While the mechanism of action is not well understood, its benefit comes from one of three TM changes: mechanical, cellular or biochemical. It is also possible that these effects are synergistic and work together to improve aqueous outflow.[1,2] The mechanical theory suggests that the TM absorbs heat from the laser, which alters its structure and stretches its pillars to increase outflow. The cellular theory, on the other hand, postulates that laser therapy alters cellular division to improve the health of the TM cells. Lastly, the biochemical theory proposes that LTP stimulates macrophage-like activity to improve aqueous outflow.

Two different lasers can be used for the procedure, each with slight differences. Here, we take a look at these two options and how you can prepare your patients—and yourself—for the pre- and post-op care.

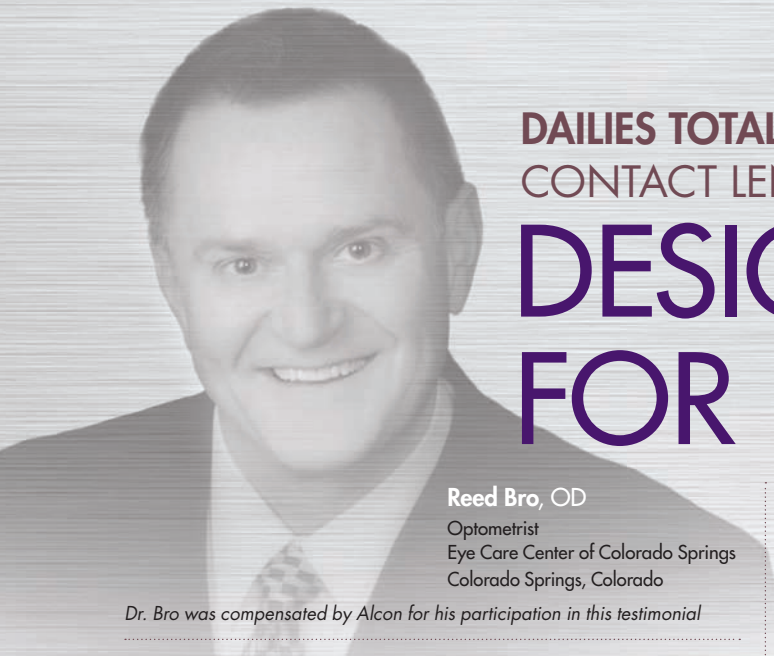
Photo: Derek Cunningham, OD

SLT, using a frequency-doubled short-pulsed Nd:YAG laser, selectively targets pigmented TM cells.

ALT vs. SLT

LTP is most commonly administered as either argon laser trabeculoplasty (ALT) or selective laser trabeculoplasty (SLT). While both procedures lower IOP by applying a laser to the TM to increase aqueous outflow, they also have their differences. ALT uses an argon laser to create larger burns and structural alterations to the TM pillars. Although *argon laser trabeculoplasty* is still the commonly understood term, argon lasers have been replaced with green lasers. SLT uses a frequency-doubled short-pulsed neodymium-doped yttrium aluminum garnet (Nd:YAG) laser that selectively targets pigmented TM cells and minimizes destruction of the surrounding tissue. Efficacy is comparable for both treatments.[1,3,4] It is important to note that while efficacy is comparable, ALT is rarely used in clinical practice.

One advantage SLT holds over ALT is repeatability. By minimizing both the amount of energy absorbed and the potential for collateral damage, SLT could theoretically be repeated over time. Although evidence suggests that repeat SLTs are fairly close in effectiveness to the original SLT, current clinical trials are hampered by small sample size and short, 12-month follow-up periods.[2,3,5-8] Such short follow-up provides poor evidence for the long-term clinical utility of repeat SLT.



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Reed Bro, OD
Optometrist
Eye Care Center of Colorado Springs
Colorado Springs, Colorado

Dr. Bro was compensated by Alcon for his participation in this testimonial

AT THE EYE CARE CENTER OF COLORADO SPRINGS, our reputation for helping presbyopes become successful contact lens wearers differentiates us from other practices in the community. One driver of this success is the ease of fitting presbyopic patients with the Alcon portfolio of multifocal contact lenses, which includes DAILIES TOTAL1® Multifocal, DAILIES® AquaComfort Plus® Multifocal, and AIR OPTIX® AQUA Multifocal contact lenses. With these impressive lenses—which share the same optical design[1] and an effective, easy-to-use fitting guide across multiple lens materials and replacement schedules—I'm able to satisfy the unique needs of almost any presbyopic patient.

To help our practice achieve multifocal success, we implemented several "best practices" to identify presbyopic patients who are candidates for contact lens wear and introduce them to multifocal lenses. First, we identify potential presbyopes as early as possible. This includes emerging presbyopes who are currently wearing spherical contact lenses, because it's easier to introduce multifocal lenses while the patient's ADD requirement is low. Once candidates are identified, my staff proactively begins the multifocal conversation prior to the exam, asking about any visual difficulties the patients are experiencing and about any recent changes in their spectacle use—including the need to use readers. We also ask existing contact lens wearers if they'd like to hear about options that can keep them in lenses, and non-wearers whether they'd be interested in learning about options to help them maintain a spectacle-free appearance. Proactively beginning these discussions prior to the exam gives patients time to consider exactly what their issues are, and whether they'd be interested in trying multifocal lenses.

During our consultations, I ask my patients how they feel about readers and bifocals, and whether they want more freedom from their spectacles. If they are interested in contact lenses, I'm careful to manage their expectations around the continued need for spectacles in some situations. DAILIES TOTAL1® Multifocal contact lenses are usually my go-to product—in my opinion, they are a tremendous leap forward in lens material and multifocal optics, with extraordinary comfort.[2,3] Patient response is usually very positive, with patients expressing surprise at the level of comfort they experience with DAILIES TOTAL1® Multifocal contact lenses (They "feel like nothing is on my eye"; "I didn't know lenses could be this comfortable").[2,3]

> With these impressive lenses I'm able to satisfy the unique needs of almost any presbyopic patient

The versatility of the Alcon multifocal portfolio—including our first-choice, DAILIES TOTAL1® Multifocal—allows me to set my practice apart by meeting the unique needs of my many presbyopic patients. The satisfaction expressed by my patients has been very rewarding, and the subsequent increase in presbyopic referrals has helped my practice thrive. Set your practice apart by fitting your presbyopic patients with the Alcon family of multifocal contact lenses.

References 1. Alcon data on file, 2016. **2.** Kern J, Kannarr S, Miller JD. Clinical outcomes for Dailies Total1 Multifocal lens in symptomatic patients. Presented at the British Contact Lens Association Clinical Conference & Exhibition, June 9-11, 2017. Liverpool, UK. **3.** Based on a survey of 544 presbyopic contact lens wearers. Alcon data on file, 2017.

Important information for AIR OPTIX® AQUA Multifocal (lotrafilcon B) contact lenses: For daily wear or extended wear up to 6 nights for near / far-sightedness and/or presbyopia. Risk of serious eye problems (i.e., corneal ulcer) is greater for extended wear. In rare cases, loss of vision may result. Side effects like discomfort, mild burning or stinging may occur.

See product instructions for complete wear, care and safety information. Rx only

Our passion is to help your patients see, look and feel their best. SEE LOOK FEEL

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Under the Laser

Before the operation, patients are given pilocarpine 1% and Iopidine (apraclonidine 0.5%, Novartis) or Alphagan P (brimonidine 0.1%, Allergan). Pilocarpine, an optional drop some choose to include in the pre-op regimen, helps to contract the ciliary body to open the pillars of the TM and induce miosis to enhance the view of the angle during the procedure. Iopidine or Alphagan P help mitigate possible IOP spikes that may occur during or after the procedure.

Practitioners can then use a gonio lens to visualize the angle before using a fixed 400µm laser spot size for a three nanosecond pulse. Many practitioners start with a lower energy, such as 0.7mJ, and titrate up from there. On average, 45 to 60 laser spots are applied to the TM per 180 degrees. While most physicians treat 360 degrees of the angle one eye at a time, some may prefer to treat less of the angle at a time—even as little as 90 degrees. This is often the case with heavily pigmented TM due to the potential higher energy absorbed by the more pigmented meshwork, especially with ALT. This is less of a concern in SLT, which minimizes structural damage to the TM.[3]

Post-laser, practitioners should immediately instill Iopidine or Alphagan P and check IOP to ensure no spike exists. Many practitioners send patients home with topical nonsteroidal anti-inflammatory drugs (NSAIDs) to control pain and inflammation. Using topical corticosteroids in these cases is controversial, as many believe that mild inflammation from the laser causes the treatment effect on the TM; however, practitioners may prescribe steroids for these patients to control postoperative inflammation. Recent evidence demonstrates that postoperative anti-inflammatory medications are not necessary after SLT, but using them does not diminish the effectiveness of the procedure.[9] Patients should return at a week post-procedure to check for complications such as IOP spike, inflammation, pain and redness. Beyond that, the two-month visit is particularly important for analyzing LTP efficacy, as it may take six to eight weeks for IOP lowering to occur.

All patients undergoing LTP for glaucoma should understand going into treatment that the procedure will not cure their chronic disease or reduce the need for careful follow-up. While many patients are familiar with laser treatments such as laser in situ keratomileusis (LASIK) and photorefractive keratectomy (PRK), few know about LTP, and managing patient expectations regarding visual outcomes with LTP is crucial.

Patient Selection: A Case Example

A 75-year-old patient diagnosed with pigmentary glaucoma presented to the office with a 0.65 cup-to-disc ratio, symmetric optic nerves and no focal neuroretinal rim thinning OU. Visual fields show mild visual field loss, and he was pseudophakic following an uncomplicated cataract extraction five years earlier. His untreated IOP has been as high as 26mm Hg OU, with average pachymetry readings. The patient discontinued Cosopt (dorzolamide/timolol, Akorn) due to breathing difficulties and was unable to use Xalatan (latanoprost, Pfizer) due to burning upon instillation. His inability to take topical glaucoma medications was exacerbated by his moderate dry eye syndrome and ocular rosacea. Based on his difficulties with topical medication and the fact that he is pseudophakic, his options are limited to Alphagan P, oral medication, LTP and surgical interventions such as trabeculectomy or tube shunt. Of those options, the patient agreed that LTP has the best risk/benefit profile and chose to pursue SLT.

Who's a Candidate, Who's Not

Ideal LTP patients include those with mild or moderate OAG, especially those with compliance issues such as patients with physical or cognitive disabilities that limit topical medication administration, patients with allergies to topical medication or patients with a general history of noncompliance with topical medication.[3,10,11] Other good candidates include patients with pseudoexfoliative glaucoma, pigmentary glaucoma, low-tension glaucoma and ocular hypertension.

Poor candidates for LTP, on the other hand, include patients with narrow angles (due to the need for TM visualization), those with secondary glaucomas (e.g., neovascular, uveitic, iridocorneal endothelial syndrome) and individuals with increased episcleral venous pressure. Those with advanced glaucoma are often not good candidates due to their need for low IOP over long periods of time. Patients with secondary glaucomas tend to need more aggressive forms of treatment such as trabeculectomy or tube shunt.

Young patients are best handled on a case-by-case basis. While they may be good candidates due to the decreased medication burden LTP provides, research shows only 31% maintain treatment effect in five years, suggesting LTP may not be ideal for those who will need long-term treatment efficacy.[3]

Lasers Go Micro

Micropulse laser trabeculoplasty (Iridex), a variation on SLT, targets the TM with pulsed laser treatments to minimize thermal damage. One study of 48 patients found IOP fell by an average of 2.5mm Hg for the SLT group and 3.0mm Hg for the MLT group.

Chadha N, Belyea D, Lamba T, Abramowitz B. A randomized, prospective comparison of 360 degree selective laser trabeculoplasty (SLT) vs. 577 nm micropulse laser trabeculoplasty (MLT) in eyes with open-angle glaucoma. Poster presented at the American Glaucoma Society 25th Annual Meeting, Feb. 26-March 1, 2015; Coronado, CA.

The *FIRST* and *ONLY* NSAID indicated to prevent ocular pain in cataract surgery patients[1]

A DROP OF PREVENTION

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Defend against ocular pain and combat postoperative inflammation with the penetrating power of BromSite® formulated with DuraSite®[1]

- DuraSite® increases ocular surface retention time, resulting in increased bromfenac absorption[2-5]
- Provides 24-hour coverage with BID dosing[1]
- Available in 5 mL bottle

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BromSite®
(bromfenac ophthalmic solution) 0.075%
Formulated with DURASITE® DELIVERY SYSTEM

Indications and Usage

BromSite® (bromfenac ophthalmic solution) 0.075% is a nonsteroidal anti-inflammatory drug (NSAID) indicated for the treatment of postoperative inflammation and prevention of ocular pain in patients undergoing cataract surgery.

Recommended Dosing

One drop of BromSite® should be applied to the affected eye twice daily (morning and evening) 1 day prior to surgery, the day of surgery, and 14 days postsurgery.

Important Safety Information

- **Slow or Delayed Healing:** All topical nonsteroidal anti-inflammatory drugs (NSAIDs), including BromSite®, may slow or delay healing. Topical corticosteroids are also known to slow or delay healing. Concomitant use of topical NSAIDs and topical steroids may increase the potential for healing problems.
- **Potential for Cross-Sensitivity:** There is the potential for cross-sensitivity to acetylsalicylic acid, phenylacetic acid derivatives, and other NSAIDs, including BromSite®. Therefore, caution should be used when treating individuals who have previously exhibited sensitivities to these drugs.
- **Increased Bleeding Time of Ocular Tissue:** With some NSAIDs, including BromSite®, there exists the potential for increased bleeding time due to interference with platelet aggregation. There have been reports that ocularly applied NSAIDs may cause increased bleeding of ocular tissues (including hyphemas) in conjunction with ocular surgery. It is recommended that BromSite® be used with caution in patients with known bleeding tendencies or who are receiving other medications which may prolong bleeding time.
- **Keratitis and Corneal Effects:** Use of topical NSAIDs may result in keratitis. In some susceptible patients, continued use of topical NSAIDs may result in epithelial breakdown, corneal thinning, corneal erosion, corneal ulceration or corneal perforation. Patients with evidence of corneal epithelial breakdown should immediately discontinue use of topical NSAIDs, including BromSite®, and should be closely monitored for corneal health. Patients with complicated ocular surgeries, corneal denervation, corneal epithelial defects, diabetes mellitus, ocular surface diseases (e.g., dry eye syndrome), rheumatoid arthritis, or repeat ocular surgeries within a short period of time may be at increased risk for corneal adverse events which may become sight threatening. Topical NSAIDs should be used with caution in these patients. Post-marketing experience with topical NSAIDs also suggests that use more than 24 hours prior to surgery or use beyond 14 days postsurgery may increase patient risk for the occurrence and severity of corneal adverse events.
- **Contact Lens Wear:** BromSite® should not be administered while wearing contact lenses. The preservative in BromSite®, benzalkonium chloride, may be absorbed by soft contact lenses.
- **Adverse Reactions:** The most commonly reported adverse reactions in 1% to 8% of patients were anterior chamber inflammation, headache, vitreous floaters, iritis, eye pain, and ocular hypertension.

Please see brief summary of Full Prescribing Information on the adjacent page.

NSAID=nonsteroidal anti-inflammatory drug.

References: 1. BromSite® [package insert]. Cranbury, NJ: Sun Pharmaceutical Industries, Inc.; 2016. **2.** Hosseini K, Hutcheson J, Bowman L. Aqueous humor concentration of bromfenac 0.09% (Bromday™) compared with bromfenac in DuraSite® 0.075% (BromSite™) in cataract patients undergoing phacoemulsification after 3 days dosing. Poster presented at: ARVO Annual Meeting; May 5-9, 2013; Seattle, Washington. **3.** ClinicalTrials.gov. Aqueous humor concentration of InSite Vision (ISV) 303 (bromfenac in DuraSite) to Bromday once daily (QD) prior to cataract surgery. https://clinicaltrials.gov/ct2/show/results/NCT01387464?sect=X70156&term=insite+vision&rank=1. Accessed March 2, 2017. **4.** Si EC, Bowman LM, Hosseini K. Pharmacokinetic comparisons of bromfenac in DuraSite and Xibrom. *J Ocul Pharmacol Ther.* 2011;27(1):61-66. **5.** Bowman LM, Si E, Pang J, et al. Development of a topical polymeric mucoadhesive ocular delivery system for azithromycin. *J Ocul Pharmacol Ther.* 2009;25(2):133-139.

SUN OPHTHALMICS

BromSite® (bromfenac ophthalmic solution) 0.075%
Brief Summary

INDICATIONS AND USAGE
BromSite® (bromfenac ophthalmic solution) 0.075% is indicated for the treatment of postoperative inflammation and prevention of ocular pain in patients undergoing cataract surgery.

CONTRAINDICATIONS
None

WARNINGS AND PRECAUTIONS
Slow or Delayed Healing
All topical nonsteroidal anti-inflammatory drugs (NSAIDs), including BromSite® (bromfenac ophthalmic solution) 0.075%, may slow or delay healing. Topical corticosteroids are also known to slow or delay healing. Concomitant use of topical NSAIDs and topical steroids may increase the potential for healing problems.

Potential for Cross-Sensitivity
There is the potential for cross-sensitivity to acetylsalicylic acid, phenylacetic acid derivatives, and other NSAIDs, including BromSite® (bromfenac ophthalmic solution) 0.075%. Therefore, caution should be used when treating individuals who have previously exhibited sensitivities to these drugs.

Increased Bleeding Time of Ocular Tissue
With some NSAIDs, including BromSite® (bromfenac ophthalmic solution) 0.075%, there exists the potential for increased bleeding time due to interference with platelet aggregation. There have been reports that ocularly applied NSAIDs may cause increased bleeding of ocular tissues (including hyphemas) in conjunction with ocular surgery.

It is recommended that BromSite® be used with caution in patients with known bleeding tendencies or who are receiving other medications which may prolong bleeding time.

Keratitis and Corneal Reactions
Use of topical NSAIDs may result in keratitis. In some susceptible patients, continued use of topical NSAIDs may result in epithelial breakdown, corneal thinning, corneal erosion, corneal ulceration or corneal perforation. These events may be sight threatening. Patients with evidence of corneal epithelial breakdown should immediately discontinue use of topical NSAIDs, including BromSite® (bromfenac ophthalmic solution) 0.075%, and should be closely monitored for corneal health.

Post-marketing experience with topical NSAIDs suggests that patients with complicated ocular surgeries, corneal denervation, corneal epithelial defects, diabetes mellitus, ocular surface diseases (e.g., dry eye syndrome), rheumatoid arthritis, or repeat ocular surgeries within a short period of time may be at increased risk for corneal adverse events which may become sight threatening. Topical NSAIDs should be used with caution in these patients.

Post-marketing experience with topical NSAIDs also suggests that use more than 24 hours prior to surgery or use beyond 14 days postsurgery may increase patient risk for the occurrence and severity of corneal adverse events.

Contact Lens Wear
BromSite® should not be administered while wearing contact lenses. The preservative in BromSite®, benzalkonium chloride, may be absorbed by soft contact lenses.

ADVERSE REACTIONS
The following serious adverse reactions are described elsewhere in the Brief Summary:
- Slow or Delayed Healing
- Potential for Cross-Sensitivity
- Increased Bleeding Time of Ocular Tissue
- Keratitis and Corneal Reactions
- Contact Lens Wear

Clinical Trial Experience
Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in clinical practice.

The most commonly reported adverse reactions in 1–8% of patients were: anterior chamber inflammation, headache, vitreous floaters, iritis, eye pain and ocular hypertension.

USE IN SPECIFIC POPULATIONS
Pregnancy
Risk Summary
There are no adequate and well-controlled studies in pregnant women to inform any drug associated risks. Treatment of pregnant rats and rabbits with oral bromfenac did not produce teratogenic effects at clinically relevant doses.

Clinical Considerations
Because of the known effects of prostaglandin biosynthesis-inhibiting drugs on the fetal cardiovascular system (closure of ductus arteriosus), the use of BromSite® during late pregnancy should be avoided.

Data
Animal Data
Treatment of rats with bromfenac at oral doses up to 0.9 mg/kg/day (195 times a unilateral daily human ophthalmic dose on a mg/m^2 basis, assuming 100% absorbed) and rabbits at oral doses up to 7.5 mg/kg/day (3243 times a unilateral daily dose on a mg/m^2 basis) produced no structural teratogenicity in reproduction studies. However, embryo-fetal lethality, neonatal mortality and reduced postnatal growth were produced in rats at 0.9 mg/kg/day, and embryo-fetal lethality was produced in rabbits at 7.5 mg/kg/day. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Lactation
There are no data on the presence of bromfenac in human milk, the effects on the breastfed infant, or the effects on milk production; however, systemic exposure to bromfenac from ocular administration is low. The developmental and health benefits of breastfeeding should be considered along with the mother's clinical need for bromfenac and any potential adverse effects on the breast-fed child from bromfenac or from the underlying maternal condition.

Pediatric Use
Safety and efficacy in pediatric patients below the age of 18 years have not been established.

Geriatric Use
There is no evidence that the efficacy or safety profiles for BromSite® differ in patients 65 years of age and older compared to younger adult patients.

NONCLINICAL TOXICOLOGY
Carcinogenesis, Mutagenesis and Impairment of Fertility
Long-term carcinogenicity studies in rats and mice given oral doses of bromfenac up to 0.6 mg/kg/day (129 times a unilateral daily dose assuming 100% absorbed, on a mg/m^2 basis) and 5 mg/kg/day (540 times a unilateral daily dose on a mg/m^2 basis), respectively revealed no significant increases in tumor incidence.

Bromfenac did not show mutagenic potential in various mutagenicity studies, including the bacterial reverse mutation, chromosomal aberration, and micronucleus tests.

Bromfenac did not impair fertility when administered orally to male and female rats at doses up to 0.9 mg/kg/day and 0.3 mg/kg/day, respectively (195 and 65 times a unilateral daily dose, respectively, on a mg/m^2 basis).

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SUN OPHTHALMICS

LTP patients with high pre-treatment IOP are likely to require additional medications or surgical intervention. For example, if we expect a typical IOP lowering of 20% from SLT, a patient with a pre-treated IOP of 30mm Hg might need additional lowering if the target IOP was less than 24mm Hg. However, the lower a patient's pre-treatment IOP, the less successful they are with LTP. In one trial, 32% of patients with IOP of <14mm Hg saw a 1mm Hg increase rather than a decrease. This should be considered for prospective LTP patients with low-tension glaucoma or those with low IOP on existing treatment.[12] Despite the difficulty meeting the recommended 30% reduction from the Collaborative Normal Tension Glaucoma Study, there may be other benefits from LTP, such as blunting nocturnal IOP spikes.[13]

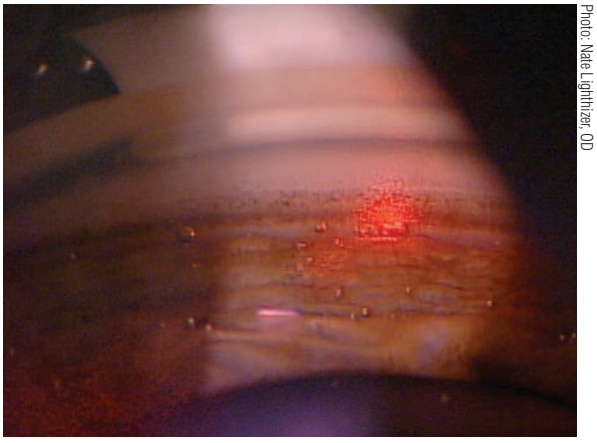

Photo: Nate Lighthizer, OD

The cavitation or "champagne" bubbles that can be seen throughout the TM during SLT lets the surgeon know the laser is properly interacting with the tissue.

The procedure's cost may also play a role in patient candidacy. One retrospective trial found LTP was slightly more expensive than medications over a 36-month period ($3,441 for LTP vs. $3,408 for medication). While LTP lowered the cost of pharmaceutical intervention ($807 for LTP patients vs. $1,467 for medication patients), it had a higher medical cost ($2,684 for LTP vs. $1,980 for medication).[14]

Complications

LTP side effects are transient and self-limited. Most tolerate the procedure without complications, but for the small percentage who do end up with complications, common possibilities including a brief increase in IOP (3mm Hg to 8mm Hg), a mild to moderate anterior chamber reaction, transient conjunctival redness and ocular pain.[1-4] Although most postoperative redness and anterior chamber inflammation is self-limited and does not require additional treatment, these side effects can be mitigated by preoperative Iopidine (apraclonidine 0.5%, Novartis), topical NSAIDs and postoperative topical Pred Forte 1% (prednisolone acetate, Allergan) if needed. Although there are case reports of IOP spikes up to 46mm Hg even two weeks post-SLT, most patients will not have complications.[14]

Additionally, patients undergoing ALT are more likely to develop peripheral anterior synechiae (PAS, 12% to 47%), than SLT patients, who rarely develop PAS.[1,4] These synechiae are different than what is seen in angle closure glaucoma or uveitic glaucoma and are not thought to cause long-term complications. PAS in secondary glaucomas are an indication of an active angle closure process, whereas PAS in LTP are localized and non-progressive.

A First-line Therapy?

While laser treatment is quickly growing in popularity—already a first-line option in Europe—it comes with limitations, the most challenging being its short duration of effect. In a short-term LTP study, SLT was as effective as medication at lowering IOP when measured for 12 months. However, research also shows LTP treatment effect diminishes over time, creating a need for a repeat procedure or more medical management.[5] Another study shows ALT was successful at lowering IOP by at least 20% in 46% of patients at one year after the procedure, but at five years, this number fell to 13%.[3] SLT has more favorable study results, with 58% of patients achieving the same IOP lowering effect as ALT at one year, and 31% maintaining the treatment effect at five years.[3] First-line topical medication is typically a prostaglandin analog, such as latanoprost. These typically lower IOP by 35% when taken in the evening. This effect lasted even five years after initiating.[16]

Lastly, a study that compared LTP with medication management found 40% of LTP patients started a glaucoma medication at 45 days post-procedure, which increased to 80% at the two-year mark. In the medication group, 81% were on two or more classes of glaucoma medication after two years compared with 27% of LTP patients.[14]

These studies highlight that while ALT and SLT may help lower IOP in the first year or more, practitioners often end up reaching for additional therapies over time.

According to the American Academy of Ophthalmology Preferred Practice Patterns, SLT should be considered as a first-line therapy in select patients, especially for those where noncompliance, cost or side effects of medication are issues.[17]

Emerging Treatments

Minimally invasive glaucoma surgeries (MIGS) are now FDA-approved to treat mild and moderate primary OAG. MIGS are *ab interno* procedures, meaning that through the use of existing corneal incisions created during phacoemulsification, the surgeon can minimize conjunctival scarring and trauma—which may occur secondary to conjunctival and sclera incisions necessary for *ab externo* procedures such as trabeculectomy.

Three major advantages of LTP over MIGS exist: it is an in-office procedure, it avoids incisional surgery and it is repeatable. This leads many practitioners to attempt laser therapy before considering surgical alternatives such as MIGS. However, MIGS do not exclude patients from future LTP applications or *ab externo* procedures.

Two MIGS procedures employ laser techniques to achieve their IOP-lowering effects:

Excimer laser trabeculostomy (ELT) uses a photoablative probe to vaporize the TM and inner wall of Schlemm's canal, resulting in a cooling effect and limiting thermal damage. The quartz fiber optic probe, connected to a xenon chloride pulsed excimer laser, is inserted through a clear corneal incision, bypassing the conjunctiva and sclera. With a gonioscopy lens, the surgeon brings the probe into contact with the TM and applies eight to 10 laser spots to create small holes that increase aqueous outflow.[18,19] This procedure can be either combined with cataract extraction or performed as a standalone procedure.

Few trials exist to demonstrate the safety and effi-

Narrow Angles? Consider Another Laser

By Nate Lighthizer, OD

While SLT has recently gained traction as a potential first-line option in the treatment of OAG patients, another laser has long been regarded as the treatment of choice in angle closure glaucoma: laser peripheral iridotomy (PI).

Laser PI is indicated for the treatment of pupillary block-caused acute angle closure, chronic angle closure, angle-closure glaucoma and narrow angles, among other conditions.[1] Recent literature has grouped anatomic narrow angles into three categories:[2]

Primary angle-closure suspect (PACS). These patients have a narrow angle—classified as failure to see the posterior TM in at least two quadrants on gonioscopy—without any IOP elevation or peripheral anterior synechiae formation.

Primary angle-closure (PAC). This is a prior suspect who has now developed either peripheral anterior synechiae or elevated IOP.

Primary angle-closure glaucoma (PACG). If primary angle-closure symptoms continue to progress and the patient develops signs of glaucomatous optic neuropathy, the patient is classified as having PACG.

A laser PI is indicated for all cases of PAC or PACG, and many believe it should even be considered for some cases of PACS.[2]

A laser PI is typically performed with either an Nd:YAG laser or an argon-green laser. Both have their pros and cons, but most eye care providers typically perform the laser PI with an Nd:YAG laser. The most common potential adverse events associated with laser PI, albeit rare, include IOP spike, inflammation, transient blur, hyphema and closure of the PI hole in the weeks following the procedure.

Preoperative medications can include an alpha-2 adrenergic agonist such as brimonidine or apraclonidine in-office to lower the risk of IOP spike, as well as pilocarpine to help stretch the iris taut prior to the procedure. Placement of the PI hole has traditionally been along the superior iris at 11 o'clock or 1 o'clock; however, newer evidence suggests a potential benefit for reduced patient dysphotopsias following the procedure when the PI hole is placed temporally.[3] The procedure can usually be completed in five to 20 shots, depending on the patient's iris color with brown irides often requiring more laser shots due to their increased thickness compared to blue irides. Appearance of the pigment plume signifies complete penetration of the iris.

Postoperative medications can include brimonidine or apraclonidine in-office as well as the use of a topical steroid for five to seven days post-procedure.

A patient with narrow angles and a concurrent visually significant cataract should be evaluated for cataract surgery, as evidence suggests that cataract extraction is superior to laser PI in opening the angles postoperatively.[4] In patients without a visually significant cataract, laser PI remains the standard for treating patients with anatomic narrow angles where the posterior TM cannot be visualized in at least two quadrants.

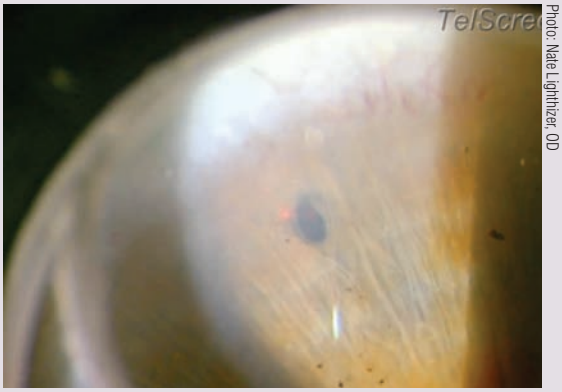

Photo: Nate Lighthizer, OD

Laser PI after two shots shows a nice pigment plume and open PI.

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cacy of ELT. In a two-year prospective study, researchers compared 180 degrees of ELT with 180 degrees of SLT. They found IOP fell from 25±1.9mm Hg at baseline to 17.6±2.2mm Hg for the ELT group and from 23.9±0.9mm Hg to 19.1±1.8mm Hg in the SLT group after 24 months. Glaucoma medications were reduced from 2.27±0.7 to 0.87±0.8 in the ELT group compared with a reduction from 2.20±0.7 to 0.87±0.8 in the SLT group.[18]

Another clinical trial involved 28 patients who underwent ELT combined with cataract extraction. Researchers split the patients into two groups: those with IOP above 21mm Hg and those with IOP below 21mm Hg at baseline. At 12 months post-procedure, the higher baseline group experienced greater ELT treatment effect than the lower IOP group.[18,19] Similar to studies involving LTP, these results suggest patients with a lower baseline IOP are less successful with ELT. None of the 28 patients in the study experienced any serious adverse events, and the most common complication was intraoperative microbleeding, which did not lead to long-term complications.

Because no trials have follow-up periods longer than 24 months, the long-term success and complication rates of ELT are still unknown. While the procedure appears effective with few side effects, many may find adopting this technology difficult because it requires a specialized probe and the use of a surgical suite. When compared with the in-office application of ALT or SLT, in which lasers often have multiple uses, ELT seems less practical.

Endocyclophotocoagulation (ECP), also known as endoscopic photocoagulation, is similar to ELT in that a probe is inserted into a clear corneal incision to spare the conjunctiva, and the procedure can be combined with cataract extraction. However, while other surgical treatment modalities focus on increasing aqueous outflow, the goal of ECP is to lower IOP by reducing aqueous production. ECP is generally used in mild to moderate glaucoma.[18]

In a two-year trial comparing cataract extraction with ECP to cataract extraction alone for OAG, IOP was reduced from 18.1±3.0mm Hg to 16.0±3.3mm Hg in the ECP/cataract extraction group compared with 18.0±3.0mm Hg to 17.3±3.2mm Hg in the cataract extraction only group at 24 months post-procedure. Medication used dropped from 1.5±0.8 at baseline to 0.4±0.7 at 24 months in the ECP group compared with 2.4±1.0 at baseline to 2.0±1.0 at 24 months for the cataract extraction only group.[20]

While postoperative IOP reduction was similar between the groups, it required more medications in the cataract extraction alone group. The ECP group did

Coding Connection

By John Rumpakis, OD, MBA, Clinical Coding Editor

Minor League Coding

With increased scope of practice comes increased risk; let's revisit the rules pertaining to minor surgical procedures?

As organized optometry continues to fight for scope of practice expansion, clinicians must arm themselves with the knowledge necessary to incorporate the new care regimens into their practices. More advanced surgical procedures such as laser trabeculoplasty, for example, call for not just new tools, but also the know-how to code for them properly.

The Stats

Laser trabeculoplasty, CPT code 65855, is defined by the CPT as "trabeculoplasty by laser surgery, 1 or more sessions (defined treatment series)." Argon laser trabeculoplasty, selective laser trabeculoplasty and diode laser trabeculoplasty are options used in the treatment of primary open-angle glaucoma (POAG)—the medical necessity you will need to prove.

These may be performed as the initial treatment, when medical therapy fails, or when a patient is unable to tolerate medications. They are considered medically necessary for these following indications:

- Primary treatment for open-angle glaucoma.
- POAG when the raised intraocular pressure is unresponsive to topical or oral medications.
- POAG with normal pressure and evidence of optic nerve damage.

The CPT characteristics that further define this code include:

- CMS National Average Maximum Allowable Reimbursement: $252.00
- Bilateral 150% procedure: this CPT designation means that if the code is billed with the bilateral modifier or is reported twice on the same day by any other means (e.g., with -RT and -LT modifiers, or with a "2" in the units field), consider the payment for these codes when reported as bilateral procedures on either (a) the total actual charge for both sides or (b) 150% of the fee schedule amount for a single code—whichever is lower. If the code is reported as a bilateral procedure and is reported with other procedure codes on the same day, apply the bilateral adjustment before applying any multiple procedure rules.
- Has a global period of 10 days following the date of surgery.
- Follows the rules for a minor surgical procedure.

Dabbling in the Minors

The rules that govern laser trabeculoplasty as a minor surgical procedure are where the compliance issues come into play. Many forget that minor surgical procedures have their own set of compliance rules, ignorance of which can lead to claim rejection, non-payment for services, inappropriate use of modifiers and, ultimately, an adverse audit result.

A minor surgical procedure has a global period of 0 or 10 days, while major surgical procedures have global periods of 90 days. While some states allow optometrists to perform major surgical procedures, the vast majority of ODs perform minor surgical procedures on a daily basis. Perhaps the two most common mistakes ODs make when billing for these minor surgical procedure is billing for an office visit on the same day as the minor surgical procedure and inappropriately using modifier -25. The National Correct Coding Initiative Policy Manual for Medicare Services clearly sets the record straight:

"If a procedure has a global period of 000 or 010 days, it is defined as a minor surgical procedure. In general E&M services on the same date of service as the minor surgical procedure are included in the payment for the procedure. The decision to perform a minor surgical procedure is included in the payment for the minor surgical procedure and shall not be reported separately as an E&M service. However, a significant and separately identifiable E&M service unrelated to the decision to perform the minor surgical procedure is separately reportable with modifier 25. The E&M service and minor surgical procedure do not require different diagnoses."[1]

Following these rules is paramount when performing any minor surgical procedures, yet many get caught up in the excitement of scope of practice expansion and access to new procedures and forget the basics. The key to successfully integrating such new techniques and care paradigms into your practice is maintaining the highest level of awareness with both patient care and medical record keeping and coding. ■

Send your own coding questions and comments to rocodingconnection@gmail.com.

1. Centers for Medicare and Medicaid Services. National Correct Coding Initiative Edits. www.cms.gov/Medicare/Coding/NationalCorrectCodInitEd/index. Accessed June 1, 2018.

report some side effects, with four patients developing postoperative cystoid macular edema (CME) compared with only one in the cataract extraction alone group. Patients with other CME risk factors such as diabetes mellitus or uveitis may not be good candidates for ECP. Otherwise, complications were uncommon.[18]

Like ELT, ECP's limitations include the need for surgical intervention and use of a specialized probe. Unless a surgeon plans to do many of these procedures, purchasing this specialized equipment may be cost prohibitive.

Today, we have more medications than ever at our disposal, traditional surgical treatments have undergone many improvements and less invasive IOP-lowering treatments such as MIGS are available for certain cases. Despite this, LTP still plays an important role for patients with mild to moderate OAG, pseudoexfoliative glaucoma and pigmentary glaucoma. In these cases, LTP can be enough to prevent more invasive intervention.

As every intervention has its pros and cons, LTP included, the future of glaucoma care will involve creative use of all modalities to individualize care. ■

Dr. DeWilde practices at the Kansas City VA Medical Center and specializes in the diagnosis and management of ocular disease.

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MIGS Madness: An Atlas of Options

With so many choices, it's a challenge to properly educate patients. These pointers and illustrations can help. **By Rachel Caywood, OD. Illustrations by Sepideh Omidghaemi, OD**

Traditionally, glaucoma management consisted of three choices, with varying levels of efficacy: eye drops, laser trabeculoplasty and, for progressing and severe glaucoma, bleb-forming surgery. However, side effects, ocular surface toxicity, out-of-pocket costs and patient noncompliance pose problems with long-term topical options.[1] Risks associated with filtering surgery, such as infection, hypotony, inflammation and bleb-related complications, may be problematic as well. Even selective laser trabeculoplasty, an excellent treatment option for many patients, has diminished efficacy over time.[2]

Luckily, patients with mild to moderate glaucoma have a new management option with the advent of minimally invasive glaucoma surgery (MIGS). These procedures have an *ab interno* approach, cause minimal trauma to the sclera and little to no scleral dissection, minimal or no conjunctival manipulation, are modestly effective and have good safety profile and rapid recovery.[3]

MIGS now consist of at least 15 different techniques, making it difficult to stay current on the advancements and efficacies of each procedure. Here, we review the current MIGS and offer guidance on patient selection and procedure recommendations.

Trabecular Meshwork Bypass

Several devices use this technique:

iStent (Glaukos). This L-shaped microstent was the first FDA-approved MIGS device in 2012 (*Figure 1d*). The surgeon inserts the lumen into Schlemm's canal, with the neck extending into the anterior chamber, to create a direct connection from the anterior chamber to Schlemm's canal. By bypassing the trabecular meshwork (TM), the stent is designed to increase aqueous outflow.[3]

iStent is approved for patients in conjunction with cataract surgery, with stable, mild-to-moderate primary open-angle glaucoma (POAG) on one to three pressure-lowering medications, and with a target intraocular pressure (IOP) of mid teens or higher. One study shows that patients who underwent cataract extraction combined with iStent had an 8% reduction in IOP and 1.3 fewer hypotensive medications after a two-year follow-up.[4] The safety profile for iStent combined with cataract extraction was equal to that of cataract extraction alone.[4] Complications include stent malposition and obstruction. To date, no adverse events have been reported in association with iStent complications.

Many factors, including proper position of the stent and its proximity to collector channels, may affect the device's efficacy.[5] Additionally, the post-op IOP will not fall below the episcleral venous pressure, which may be elevated in some POAG patients.[6] The maximum IOP lowering effect will be achieved six to eight weeks after implantation.

iStent inject (Glaukos). This device's injector houses two iStents and is designed for easier injection and to reduce both surgery time and the risk of infection (*Figure 1c*). The appearance of the second-generation stent is similar to that of a punctal plug, with a linear, not L, shape. The method of action for iStent inject is identical to that of the original iStent. These microstents are inserted in the nasal angle two clock hours apart in a procedure combined with cataract surgery. A study found a 15% IOP reduction after two years and 18.9% reduction after five years, with 1.00 and 0.21 fewer medications, respectively.[7] iStent inject, with a similar safety profile and complications as the original,

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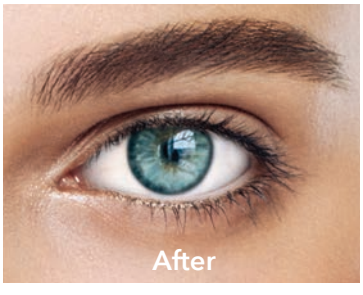

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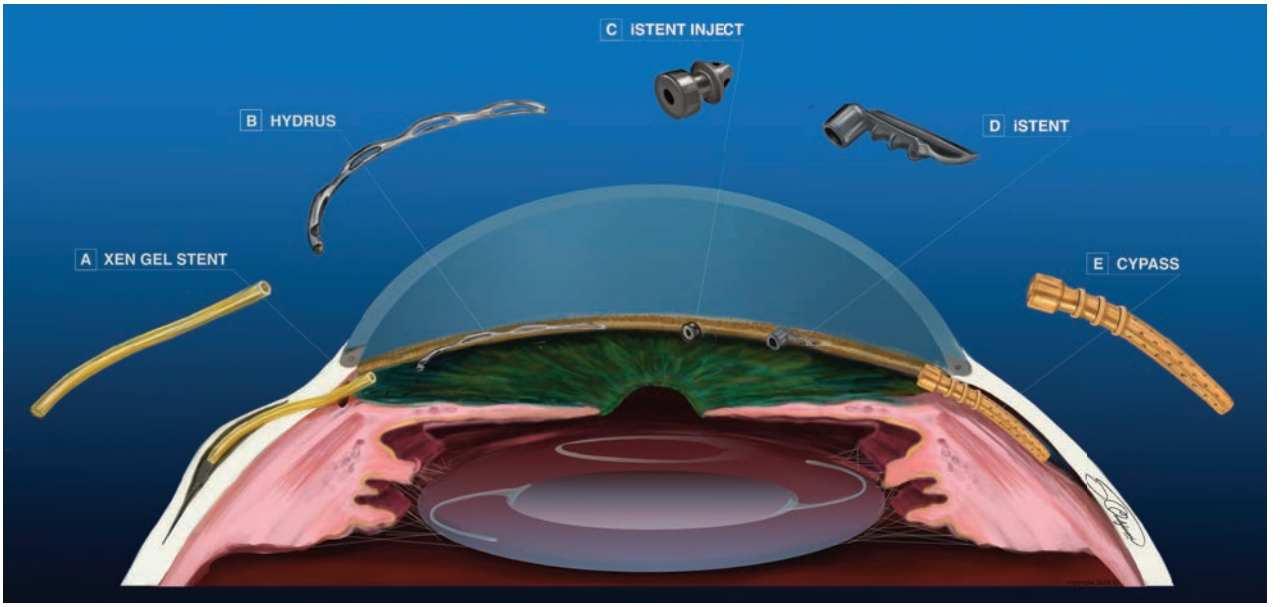

Fig. 1. Each of these MIGS procedures can reduce IOP, but in distinctly different ways.

was approved by the FDA in June.

Hydrus microstent (Ivantis). This is an 8mm device used to improve aqueous outflow in three ways (*Figure 1b*). The Hydrus provides direct connection between the anterior chamber and Schlemm's canal. Due to its length, it creates a three clock-hour scaffolding of the canal. Additionally, three windows on the anterior face of the device stretch the TM and increase aqueous outflow.

The Hydrus is projected for FDA approved in late 2018 for use in conjunction with cataract surgery. The stent is preloaded in an inserting device and is placed through the same clear cornea incision used for cataract surgery. Ideal patients would be those with similar glaucoma severity and treatment profile as iStent candidates, with the exception that, for proper device placement, Hydrus candidates should have at least moderately pigmented TM for easier anatomical location.

One study found average reduction in IOP was 12.7% with patients using 1.5 fewer medications two years after Hydrus combined with cataract surgery.[8] The most common complication found in this study was peripheral anterior synechia near the inlet segment of the device, which did not affect IOP reduction.[8]

Trabeculotomy

The efficacy of all delineated TM/Schlemm's canal procedures depends on the integrity of the collector channels downstream, as damage or occlusion limits the effect. The following TM/Schlemm's canal procedures are better suited for younger patients who are more likely to have an intact collector channel system:[9]

Trabectome (NeoMedix). This electrocautery device is used to perform a partial trabeculotomy (*Figure 2c*). The surgeon inserts the device through a temporal clear corneal incision and directs it to the nasal portion of the angle. The TM and interior wall of Schlemm's canal are removed via cauterization anywhere from 90° to 180°. Aspiration and irrigation ports on the tip of the device maintain homeostasis during the procedure. Trabectome is approved as a stand-alone procedure or in combination with cataract surgery. Contraindications include narrow angle (less than 20°), increased episcleral venous pressure, limited neck mobility, neovascularization of the angle or iris and corneal opacity obstructing the view of the angle.

Complications include blood reflux into the anterior chamber with up to 20% hyphema and partial goniosynechiae. These did not cause a significant IOP increase or failure of the procedure.[10] Two studies show 23% and 44% IOP reduction two years after trabectome.[10,11]

Kahook Dual Blade (New World Medical). This single-use knife is designed to remove the TM and Schlemm's canal internal wall without damage to adjacent tissues (*Figure 2d*). The surgeon inserts the knife through a temporal clear corneal incision during cataract surgery or in a stand-alone procedure. A section of nasal TM (up to 180°) is excised and removed with forceps or aspiration. Studies show the mean baseline IOP decreased by 26.4% with an average of 0.7 fewer medications six months post-procedure.[12]

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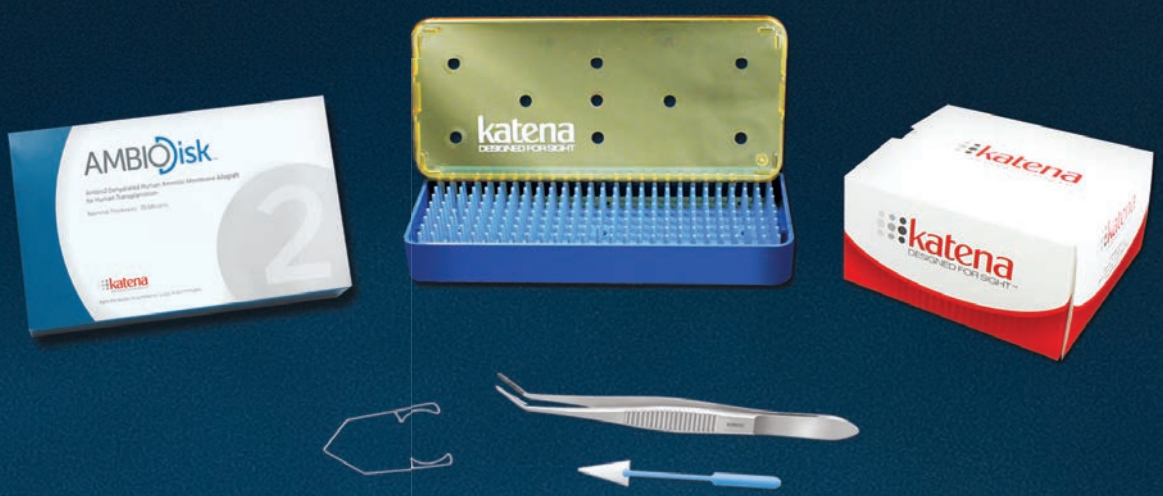

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An advantage of this procedure over Trabectome is the lack of thermal damage to the angle and reduced risk of peripheral anterior synechia postoperatively. As with all trabeculotomies, hyphema is the most common complication and typically resolves within one month.

Gonioscopy-assisted transluminal trabeculotomy (GATT). During this 360° trabeculotomy, the surgeon creates a temporal clear corneal incision and inserts forceps (*Figure 2a*). A suture or microcatheter is inserted through a separate clear corneal incision. A small goniotomy is created nasally in the TM, and the suture or microcatheter is threaded through the circumference of Schlemm's canal. The suture is then pulled through to unroof the TM.

The procedure can be performed during cataract surgery or alone in a phakic or pseudophakic eye. Researchers report an average of 39.8% IOP reduction and 1.1 fewer medications in postoperative GATT patients with POAG.[13] The most common complication is postoperative hyphema, which typically self resolves within a month. Advantages include its low cost, especially with suture material, and the full 360° treatment of the angle.

Trab360 (Sight Sciences). This 360° trabeculotomy is performed through one clear corneal incision temporally, where the Trab360 device is inserted into the anterior chamber (*Figure 2b*). The tip pierces the TM nasally and a probe contained within the device is advanced 180° into Schlemm's canal; the surgeon then extracts the device from the eye, cutting through the TM and creating a 180° trabeculotomy. The surgeon can retract the probe and use the device to treat the remaining 180° of TM. Trab360 can be performed in conjunction with cataract surgery or as a stand-alone procedure. Initial results show an average IOP reduction of 31.8% and patients required 0.9 fewer meds.[14]

Table 1. Summary and Results of Select MIGS Studies

Procedure	Stent	w/cataract surgery or stand-alone	Target structure	Pre-op IOP +/- SD	Post-op IOP +/- SD	IOP reduction	Pre-op meds +/- SD	Post-op meds +/- SD	Total medication reduction	Post-op period
iStent[4]	Yes	Combined	TM/Schlemm's canal	18.6 +/-3.4	17.1 +/-2.9	8.1%	1.6 +/-0.8	0.3 +/-0.6	1.3	Two years
iStent inject[7]	Yes (2)	Combined	TM/Schlemm's canal	20.0 +/-3.7	17.0 +/-2.3	15%	1.3 +/-0.7	0.5 +/-0.6	0.83	Two years
Hydrus[8]	Yes	Combined	TM/Schlemm's canal	18.9 +/-3.3	16.5 +/-2.9	12.7%	2.0 +/-1.0	0.5 +/-1.0	1.5	Two years
Trabectome[10]*	No	Either	TM/Schlemm's canal	27.6 +/-7.2**	15.2 +/-2.4	44.9%	Not reported			Two years
Kahook[12]	No	Either	TM/Schlemm's canal	17.4 +/-5.2	12.8 +/-2.6	26.4%	1.6 +/-1.3	0.9 +/- 1.0	0.7	Six months
GATT[13]*	No	Either	TM/Schlemm's canal	25.6 +/-6.1	15.7 +/-4.5	38.7%	3.2 +/-0.9	1.5 +/-1.2	1.7	One year
Trab360[14]	No	Either	TM/Schlemm's canal	19.8 +/-6.4	13.5 +/-4.0	31.8%	1.1 +/-1.2	0.2 +/-0.5	0.9	One year
ABiC[16]	No	Either	TM/Schlemm's canal/collector channels	28.6 +/-5.9	13.8 +/-3.2	51.7%	1.9 +/-1.2	0.5 +/-0.8	1.4	One year
CyPass[18]	Yes	Combined	Suprachoroidal space	24.5 +/-3.0	17.1 +/-3.4	30.2%	1.4 +/-0.9	0.2 +/-0.6	1.2	Two years
iStent Supra[19]	Yes	Combined	Suprachoroidal space	20.4 +/-2.4	11.9	41.6%	2	1	1	Two years
Solx gold shunt[20]	Yes	Either	Suprachoroidal space	30.8 +/-8.8	13.7 +/-3.0	55.5%	2.8 +/-1.1	1.5 +/-0.8	1.3	Two years
Xen[21]	Yes	Either	Subconjunctival	22.5 +/-3.7	13.1 +/-2.4	41.8%	2.5 +/-0.09	0.4 +/-0.8	2.1	One year
InnFocus Microshunt[23]	Yes	Either	Subconjunctival	23.8 +/-5.3	10.7 +/-3.5	55%	2.4 +/-0.9	0.7 +/-1.1	1.7	Three years
ECP[26]	No	Either	Anterior cilliary processes	18.1 +/-3.0	16.0 +/-3.3	11.6%	1.5 +/-0.8	0.4 +/-0.7	1.1	Two years

* reported results for stand-alone procedure, ** non-medicated IOP

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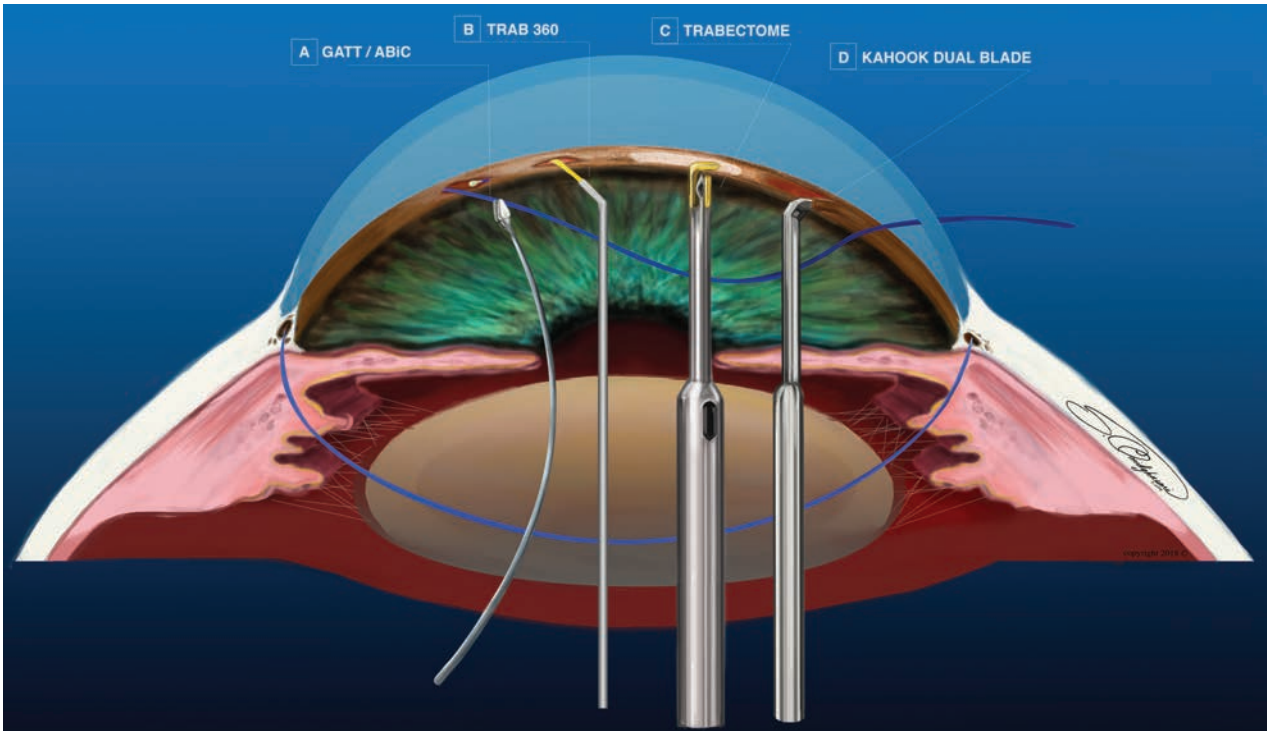

Fig. 2. Rather than use an implanted device, many MIGS use a blade, laser or sutures to complete the procedure.

Natural Outflow Enhancement

A slight variation on traditional **ab externo** ***canaloplasty,*** **ab interno** ***canaloplasty (ABiC) (iTrack, Ellex)*** treats all aqueous outflow structures (*Figure 2a*). As with GATT, a microcatheter device is advanced through Schlemm's canal 360°. Instead of pulling the microcatheter to cut the TM, the surgeon extracts it as viscoelastic is gently injected into Schlemm's canal. This viscodilation breaks adhesions within Schlemm's canal, stretches the trabecular plates and creates microperforations within the inner wall. It also improves outflow by irrigating collector channel blockages.[15]

With ABiC, all structures of the aqueous pathway remain intact. Currently, it is the only MIGS that addresses collector channel blockages. ABiC can be a stand-alone procedure or done in conjunction with cataract surgery in primary and secondary glaucoma. The procedure is not suitable for angle closure, neovascular or traumatic glaucoma. Results of ABiC alone one year after surgery show average IOP fell by 19.3%, and patients used 1.4 fewer medications. ABiC combined with cataract surgery can provide an average IOP reduction of 51.7% and 1.4 fewer medications after one year.[16]

In the same fashion as ABiC, ***Visco360 (Sight Sciences)*** is designed to viscodilate Schlemm's canal and collector channels using the Trab360 device to perform the surgery.

Multiple MIGS

It is becoming increasingly popular for surgeons to perform multiple MIGS in the same surgery to combine different methods of action or target tissue to maximize IOP lowering effect. Two common combination procedures include the Omni device (Sight Sciences), which is a combination of Visco360 and Trab360 procedures, and ICE, which involves iStent implantation in conjunction with cataract extraction and ECP.

Suprachoroid

The ***CyPass (Alcon)*** is an *ab interno* suprachoriodal shunt made of brown polymide material (*Figure 1e*). It has a 0.3mm lumen, is 6.35mm in length and comes preloaded in a curved inserter. The surgeon inserts the device through a temporal clear corneal incision into the nasal angle posterior to the scleral spur, with the distal portion of the device embedded in the suprachoroidal space. The shunt diverts aqueous from the anterior chamber into the suprachoroidal space using the uveoscleral outflow system. Aqueous not only flows though the lumen of the stent, but also through fenestrations in the distal portion of the device.[17] The COMPASS trial two-year outcomes show mean IOP was reduced by 30.2% and 1.2 fewer medications.[18] Infrequent and largely transient adverse events included reduced vision by more than two lines,

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visual field progression, iritis and corneal edema. Stent obstruction occurred in 2.1% of patients due to focal peripheral anterior synechia.[18] CyPass is FDA-approved for use in open angle glaucoma with cataract surgery.

iStent Supra (Glaukos). This suprachoroidal shunt is comprised of polyethersulfone and titanium. The placement and mechanism of action are identical to the CyPass stent. iStent Supra is currently available in Europe and Canada and is in US clinical trials for insertion combined with cataract surgery.[19]

Solx gold shunt (Solx). Made of 24-carat gold, this 5.5mm by 3.2mm flat stent is inserted ab externally through a scleral incision in any quadrant of the eye. The anterior portion of the stent is placed 1mm into the anterior chamber with the posterior end of the stent in the suprachoroidal space. Created for use in patients with refractory glaucoma, this device is approved for use in Europe and Canada and is currently in clinical trials for FDA approval as a stand-alone procedure or combined with cataract surgery.[20]

Subconjunctiva

The Xen gel stent (Allergan) is a gelatin and glutaraldehyde 6mm tube, which is preloaded in a disposable injector (*Figure 1a*). The surgeon inserts the injector through a clear cornea incision and tunnels through the sclera at or anterior to Schlemm's canal to deploy the distal portion of the stent within the subconjunctival space. This creates a pathway for aqueous to flow from the anterior chamber to the subconjunctival space, forming a bleb. The procedure may include subconjunctival injection of mitomycin C to prevent scar formation and bleb failure.[21]

Xen is FDA approved for refractory glaucoma. It is useful in patients with primary, pseudoexfoliative or pigmentary glaucoma with open angles. It is reserved for patients who have progressive disease despite maximum medical treatment, and who failed previous surgical interventions. A recent study of 41 eyes with the Xen implant combined with cataract surgery shows an average IOP reduction of 41.8% and 2.1 fewer medications post-procedure.[22] Complications were rare in this study, and included one case of each of the following: bleb requiring needling, device explant, device migration, device obstruction, transient hypotony and transient choroidal detachment.[22]

The InnFocus MicroShunt (InnFocus, Santen) is a 8.5mm long implant made of poly(styrene-block-isobutylene-block-styrene). The surgeon inserts this device into the anterior chamber through an *ab externo* approach, creating a bleb in the subconjunctival space. During the procedure, mitomycin C is applied to the area of the intended bleb to reduce the risk of scar formation and bleb failure. The procedure may be performed in combination with cataract surgery or alone, and is approved for use in Europe. It is currently in clinical trials for approval in the United States.[23]

Cyclophotocoagulation

The ***endoscopic cyclophotocoagulation (ECP, Endo Optiks)*** probe is a reusable device consisting of a laser, aiming beam, light source and field view endoscope. The surgeon inserts the endolaser device through a clear corneal incision, directs the probe posterior to the iris and treats the anterior cilliary processes with photocoagulation (*Figure 3*). Roughly 240° to 300° of cilliary body processes can be treated with a single incision; a full 360° treatment requires a second incision. Research shows a direct correlation between the amount of cilliary processes treated and the effectiveness of the procedure.[24] It can be used in open- or closed-angle glaucoma, as an initial treatment option for mild-to-moderate glaucoma, or for advanced glaucoma for which previous management has failed.[25] One study shows a mean IOP reduction of 11.6% and medication reduction by 1.1 for patients who had ECP with cataract surgery.[26]

While a useful procedure, it comes with possible complications, including: chronic inflammation, cystoid macula edema, cataracts, hypotony and phthisis bulbi. ECP may be performed during cataract surgery or alone. Due to the risk of cataract development, ECP it is better suited for phakic patients or in combination with cataract surgery.

A similar procedure, ***micropulse transscleral cyclophotocoagulation (Cyclo G6/MicroPulse P3, Iridex)***, staggers laser energy output into pulses to minimize iatrogenic damage. An investigational technique called ***ultrasound-mediated cyclomodification*** uses ultrasonic energy to produce controlled thermocoagulation without deleterious effects to surrounding tissue.[26]

Patient Selection

While the final decision is often in the surgeon's hands, optometrists are key players in comanagement. ODs will be faced with the first patient encounter and must consider many education factors, including the fact that cataract surgery alone can lower IOP.[27] For those with cataracts and mild-to-moderate glaucoma who are stable on one medication, iStent combined with cataract extraction would be a good option as well.[27] Referral to a skilled cataract surgeon with iStent experience or a glaucoma specialist would be appropriate.

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Glaucoma patients on multiple medications with a goal IOP of mid teens or higher and visually significant cataracts would benefit from iStent inject, Hydrus, Trabectome, Kahook, GATT, Trab 360, ABiC, CyPass or ECP combined with cataract surgery rather than cataract surgery alone. Pseudophakic patients whose goal IOP is not met with medical management or are noncompliant with medications could be considered for Trabectome, Kahook, GATT, Trab 360, ABiC or ECP. Xen gel stent is best suited for patients with refractory glaucoma.

Patients with a target IOP of 12mm Hg or lower will achieve minimal benefit from a TM/Schlemm's procedure and would be better served with CyPass combined with cataract surgery or Xen stent.[28]

A MIGS referral should include the following, whenever possible:

- Maximum IOP and IOP on current treatment.
- Current glaucoma medications and any that were ineffective or not tolerated.
- Baseline and current visual field and any fields demonstrating progression.
- Gonioscopic assessment of the angle structures.
- Baseline and current optic nerve OCT and glaucoma progression analysis.
- Eye surgery/injury history.

Providing this information in your referral will eliminate repeat testing and inform the surgeon of the patient's disease process, allowing the surgeon to choose the best MIGS procedure for the patient.

It is an exciting time to treat glaucoma, as patients now have a middle road to travel for management. A basic understanding of the available MIGS procedures and their individual niches in glaucoma management can help you educate patients properly, refer with confidence, and ultimately reduce the need for medication and thus the costs and inconvenience to the patient. ■

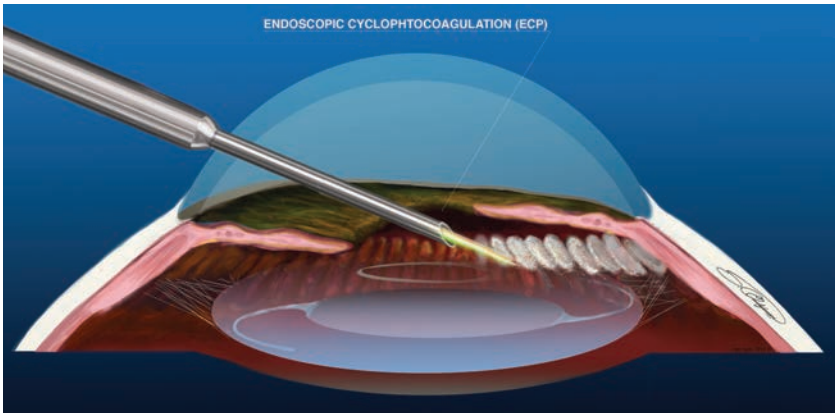

Fig. 3. ECP is the only MIGS procedure that decreases aqueous production.

Dr. Caywood is a staff optometrist at the Oklahoma City VAMC.

Dr. Omidghaemi is a talented artist and a staff optometrist at the Oklahoma City VAMC.

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PEER • REVIEWED PRA ARTICLE

Comanaging Invasive Glaucoma Surgeries

When the easy options aren't enough, it's time to call in the heavy artillery. Here's what surgeons can do for those patients most in need. By Justin Schweitzer, OD

Glaucoma treatment has undergone significant changes in both pharmaceutical and surgical arenas over the last few years. New topical glaucoma drugs with multiple mechanisms of action are helping to lower intraocular pressure (IOP), but patient adherence, ocular toxicity and lifestyle issues continue to be detriments. Minimally invasive glaucoma surgeries (MIGS), targeting mild-to-moderate glaucoma, are providing ophthalmologists with a wide array of devices to lower IOP. Technological advances allow for earlier detection, yet patients progress and severe glaucoma continues to exist. In these cases, traditional incisional surgeries are necessary to prevent debilitating vision loss.

Optometrists must understand why these procedures are necessary, how they are performed and our roles in the pre- and post-op care of traditional incisional surgeries.

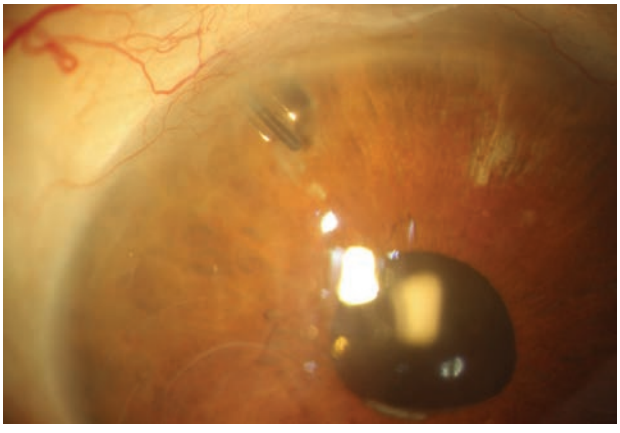

Fig. 1. Here you can see a Baerveldt aqueous tube shunt with the nylon "ripcord" suture visible within the lumen of the tube shunt. If additional IOP lowering is needed, the suture can be removed to increase flow through the tube shunt.

Preoperative Considerations

Surgical intervention is usually not the first choice for treating a glaucoma patient. Topical glaucoma drops or selective laser trabeculoplasty are initially good options for high-risk glaucoma suspects and both mild and moderate glaucoma patients. However, when a patient displays above-target IOP and VF progress and when OCT shows retinal nerve fiber layer (RNFL) thinning and topical medications are maxed out, clinicians should start a conversation about surgery. The emergence of MIGS has given surgeons an option for patients with mild-to-moderate glaucoma. However, when these procedures are not effective enough, a more aggressive approach is necessary.

The decision on what type of filtration surgery often comes down to surgeon preference and the procedure they are most comfortable with. As this article will show, all of the procedures have the ability to effectively lower IOP. At times, the anatomy of the eye can dictate the decision as well. If the patient has a small globe or a particularly narrow palpebral fissure, placing an aqueous tube shunt can be difficult and

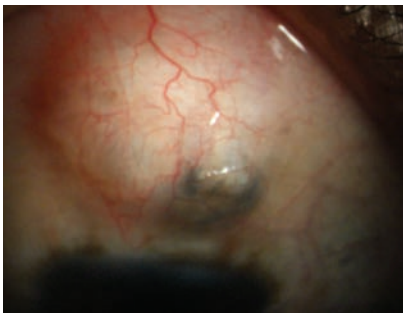

Fig. 2. The filtering bleb of a patient with aphakic glaucoma and nystagmus. Note the area of pigment in the bleb secondary to iris pigment liberation.

a trabeculectomy may be optimal. As the primary eye care provider, it's the optometrist's responsibility to remind patients that these surgeries have an extended healing period. It is not uncommon for the patient to have a foreign body sensation or discomfort in the eye for at least one week. It can take months before IOP stabilizes, and at times can fluctuate from too low to too high. If expectations are set before surgery, the postoperative period for both the OD and patient will go smoother.

Devices and Procedures

Aqueous tube shunts divert aqueous humor from the anterior chamber to an external reservoir. A fibrous capsule forms about one month after implantation over the plate of the tube shunt to regulate flow.

Tube shunts come in a variety of sizes, materials and designs. Some familiar ones include the Molteno implant (Katena), the Baerveldt implant (AMO/Johnson & Johnson Vision) and the Ahmed glaucoma valve (New World Medical).

The Molteno and Baerveldt implants are nonvalved devices and, until the fibrous capsule forms, have no way of controlling aqueous flow, which can lead to early postoperative hypotony (*Figure 1*). For that reason, the devices are partially occluded with a nylon suture and tied off with a four-to six-week dissolvable vycryl suture. This technique causes postoperative IOP to remain unchanged, requiring patients to continue all of their preoperative glaucoma medications until the fibrous capsule forms to regulate aqueous flow and the suture dissolves. The nonvalved devices are a good choice for glaucoma patients who do not need immediate IOP control. The surgeon has the ability to attempt to predict or guide the postoperative IOP with a Baerveldt implant by selecting different sizes.

The Ahmed glaucoma valve provides a more immediate IOP response. The valve mechanism consists of multiple thin membranes that are pretensioned to open and close in response to IOP variations, in the range of 8mm Hg to 12mm Hg.[1] A lower rate of hypotony is seen with Ahmed glaucoma valves compared with Baerveldt implants.[2]

Implanting these devices requires a conjunctival incision to create a conjunctival flap between two recti muscles, typically in the superotemporal quadrant.[3] Tenon's capsule is dissected from the episclera and episcleral vessels are gently cauterized. The implant is placed 8mm to 10mm from the limbus and sutured to the sclera.[3] The drainage tube is trimmed to 2mm to 3mm insertion and bevel cut to an angle of 30 degrees to allow access to the anterior chamber. A 22g to 23g needle is used to create a tract for tube insertion in the anterior chamber.[3]

Trabeculectomy

This filtering surgery creates an opening into the anterior chamber from underneath a scleral flap (*Figure 2*). The opening allows aqueous to flow out of the eye, into the subconjunctival space and creates a filtering bleb.[4]

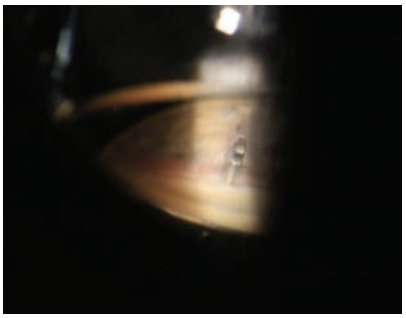

Fig. 3. The lumen of an Express mini glaucoma shunt visible in the anterior chamber.

According to a 2011 survey by the American Glaucoma Society, trabeculectomy started to decrease in popularity between 1996 and 2008.[5] Concerns with bleb-related complications has lead to an increased use of tube shunts. The survey reveals members prefer trabeculectomy with mitomycin C (MMC) in 74% of patients compared with 11% placement of tube shunts in 2008.[5] In 2016, the survey was repeated, and tube shunt surgery increased to 23% compared with a decrease of 59% use of trabeculectomy with MMC.[6]

The surgical technique begins with a creation of a superior fornix-based conjunctival flap, and MMC is applied to the bare sclera for one to four minutes. MMC is an antimetabolite agent that prevents subconjunctival fibrosis and subsequent failure of the trabeculectomy. Next, a partial thickness scleral flap is created, the anterior chamber is entered with a thin metal probe called a stylet and a peripheral iridectomy (PI) is performed. The final steps of the procedure involve the scleral flap being sutured and a bleb being formed by suturing the conjunctiva to the limbus.

Express mini glaucoma shunt (Alcon) is a device with characteristics of an aqueous tube shunt and a trabeculectomy (*Figure 3*). It is a stainless steel 3mm biocompatible device with an external diameter of 400µm.[7] It is non-valved, with a 50µm lumen and an external disc or plate at one end and a spur-like extension on the other end to prevent extrusion.[7] The device shunts aqueous from the anterior chamber to a subconjunctival reservoir similar to a trabeculectomy, but without the removal of scleral or iris tissue.[8]

The surgeon makes a superior fornix-based conjunctival incision to allow the creation of a scleral flap in the same manner as a trabeculectomy. A temporal paracentesis wound is created through the cornea and MMC is applied to the bare sclera. The scleral flap is lifted and a 26g needle is inserted into the anterior chamber to allow access for the device. It comes preloaded on an injector and its lumen gains access to the chamber through the wound created by the needle. The plate of the device is placed flush with the scleral bed and the scleral flap is sutured into place. One to three releasable sutures are placed and can be removed depending on the aqueous flow required. The conjunctiva is closed and, using the paracentesis wound, the bleb is inflated with balanced salt solution.[8-10]

In The Lit

One major analysis of the efficacy and safety of tube shunt surgery and trabeculectomy, the Tube Vs. Trabeculectomy (TVT) study, was designed prospectively over a five-year period.[11] It looked at 212 patients with uncontrolled glaucoma who had previously undergone cataract extraction, had failed filtering surgery or both.[11] Patients were randomized to receive either a Baerveldt glaucoma implant or a trabeculectomy with MMC.[11] The goal of the study was to provide information to assist with surgical decisions between tube shunt surgeries versus trabeculectomy with MMC.[11]

The degree of IOP reduction was effective and similar between treatment groups at five years. The baseline IOP was 25.1mm Hg +/-5.3mm Hg in the tube group and 25.6mm Hg +/-5.3mm Hg in the trab group. At five years, IOP was 14.4mm Hg +/-6.9mm Hg in the tube group and 12.6mm Hg +/-5.9mm Hg in the trabeculectomy group.

The majority of patients enrolled in the TVT study had advanced glaucoma; thus, many had initiated near maximal medical therapy. The researchers noted a significant reduction in medication use in both groups, but no significant difference in mean number of medications between the groups at five years. Baseline medications were 3.2 +/-1.1 in the tube group and 3.0 +/-1.2 in the trabeculectomy group. At five years, mean number of medications was 1.4 +/-1.3 in the tube group and 1.2 +/-1.5 in the trab group.

The cumulative probability of failure at five years was 29.8% in the tube group and 46.9% in the trabeculectomy group. Failure included patients with persistent hypotony, reoperation for glaucoma, or loss of light perception vision. The higher failure rate of the trab group at five years was consistent with data at one year and three years.[12,13]

The TVT study does not show a clear superiority of one glaucoma procedure over the other, but a shift in the use of tube shunts has occurred since 2008.[5,6] One reason may be the increased failure rates of trabeculectomy surgery compared with tube shunt surgery.[5,6]

The Primary Tube vs. Trabeculectomy (PTVT) study is a randomized clinical trial comparing the safety and efficacy of tube shunt surgery and trabeculectomy with MMC in eyes without prior ocular surgery.[14] The study is ongoing. Similar to the

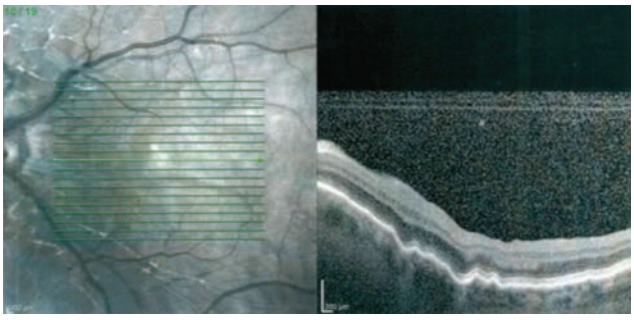

Fig. 4. The macular OCT of a patient with a Express mini glaucoma shunt and hypotony maculopathy. Note the choroidal folds and retinal striae.

TVT study, the PTVT study goal is to assist with surgical decisions between tube shunt surgeries and trabeculectomy with MMC.[14]

The degree of IOP reduction was effective and similar between treatment groups at one year. Baseline IOP was 23.3mm Hg +/-4.9mm Hg in the tube group and 23.9mm Hg +/-5.7mm Hg in the trab group. At one year, IOP was 13.8mm Hg +/-4.1mm Hg in the tube group and 12.4m Hg +/-4.4mm Hg in the trabeculectomy group. One year IOP reduction results are similar to what was found in the five-year results of the TVT study, showing the effective and powerful IOP lowering ability of both procedures.[11]

Medication reduction occurred in both groups, but significantly greater use of medication was seen in the tube shunt group vs. the trabeculectomy group at one year. Baseline medications were 3.1 +/-1.1 in the tube group and 3.2 +/-1.1 in the trabeculectomy group. At one-year follow-up, mean number of medications was 2.1 +/-1.4 in the tube group and 0.9 (+/-1.4) in the trabeculectomy group.

The cumulative probability of failure at one-year follow-up was 17.3% in the tube group and 7.9% in the trabeculectomy group. The result of failure rates is opposite to what was seen in the TVT study five-year results.[11] Ultimately, failure rates will depend on follow-up for the PTVT study.

Similar to the TVT study, PTVT does not demonstrate a clear superiority for one procedure over the other. In contrast to the TVT study, PTVT has made a case that not only is trabeculectomy efficacious in lowering IOP, but demonstrates a lower failure rate than tube shunt surgery at one year. A longer follow-up period is necessary to fully compare the two studies.

Express mini glaucoma shunt vs. trabeculectomy. The Express shunt was designed to be a simpler aqueous filtration device with the efficacy of a trabeculectomy, with reduced rates of complications. Multiple studies have reported no significant difference in IOP reduction between the procedures.[15-18] The Express shunt has been shown to have reduced rates of early postoperative hypotony, thought to be because of uniform filtration through the lumen of the device.[15,16,18,19] There have also been studies showing that less inflammation occurs postoperatively with the Express shunt as no iridotomy is performed.[15,16,18,19] Long-term hypotony, bleb morphology, visual acuity and bleb-related complications have been shown to be similar to trabeculectomy.[15-19]

With many studies showing equal efficacy and safety of Express shunt and trabeculectomy, economic differences have been a topic often discussed. One study reported a 3.5x higher disposable cost for Express vs. trabeculectomy.[20]

Postoperative Considerations and Complications

Trabeculectomy's and Express shunts' typical post-op care involves visits according to the IOP, anterior chamber depth and bleb characteristics. A low-lying diffuse bleb with minimal vascularity, cystic changes, low teens IOP, formed anterior chamber and a Seidel-negative conjunctival closure is an ideal bleb.[21,22] The most common complications occur due to changes in bleb structure. These complications include hypotony, bleb leaks and blebitis.[21,22]

Hypotony can present secondary to a conjunctival wound leak in the early postoperative period. The key finding is a low IOP—less than 5mm Hg—without a visible bleb. A Seidel's test will localize the site of the leak, and conservative treatment is initially recommended. Conservative treatment involves placing a bandage contact lens, aqueous suppressant drops and the initiation of antibiotic drops. If the eye fails to respond, a referral back to the surgeon is warranted for possible surgical intervention.[23]

Hypotony that is present for three months or longer is classified as chronic hypotony. Chronic hypotony can lead to hypotony maculopathy, which is associated with a decrease in visual acuity, choroidal folds, retinal striae and the absence of edema (*Figure 4*).[24,25] Chronic hypotony is a challenge to manage as surgical intervention (bleb size reduction by cryotherapy, compression sutures, closing of the scleral flap or applying a scleral patch graft) results are variable and, at times, can lead to unsafe levels of IOP elevation.[26-28]

The use of MMC can cause thin-walled blebs, increasing the risk of a bleb leak and subsequent hypotony, blebitis or endophthalmitis (*Figure 5*).[29] A bleb leak is detected by using a fluorescein strip and cobalt blue illumination. The fluorescein strip is applied to the bleb; if a leak is present, subtle unstained aqueous will be seen flowing from the bleb with dark green fluorescein stained tear film surrounding it.

Treatment should be conservative, with the initiation of aqueous suppressants, topical antibiotic, application of a bandage contact lens or use of an amniotic membrane graft.[30] If conservative treatment does not resolve the leak, a referral back to the surgeon is best, and closure of the leak by cyanoacrylate glue, fibrin tissue glue or surgical revision can be attempted.[31-33]

Bleb-related ocular infections such as endophthalmitis and blebitis have been reported at 2% and as high as 6% to 7.5%.[34] The most common symptoms are ocular pain, foreign body sensation, and blurry vision. If an ocular infection is suspected examination of the bleb for a milky white appearance and loss of clarity is critical, as well as a careful examination for any anterior chamber or vitreous involvement.

A blebitis is an infection limited to the filtering bleb and will likely respond well to aggressive antibiotic treatment. If there is involvement of the anterior chamber, vitreous and bleb, the main concern is endophthalmitis. A referral to the surgeon should be made so a fluid tap can be done. Topical, systemic, and, likely, intravitreal injections of antibiotics will be necessary.

Aqueous tube shunt complications share some of the issues encountered with trabeculectomy, but also have some unique issues.

The majority of complications with aqueous tube shunts are hypotony related. Hypotony can occur in the early or late post-op period and with valved or non-valved shunts. On examination, if the anterior chamber is shallow or flat, or choroidal effusions exist, the surgeon will inject a small volume of viscoelastic into the anterior chamber. If hypotony persists after numerous injections, the patient may need tube ligation.

In the TVT study, 6% of patients receiving an aqueous tube shunt had persistent diplopia after three years. Bleb height is one likely reason for diplopia. It is argued that proper placement of the tube plate under the muscles will prevent diplopia. In certain cases of persistent and transient diplopia, spectacle lenses with prism may need to be prescribed to give patients relief.

Other complications may include tube-corneal touch, uncontrolled high IOP, endophthalmitis, corneal edema and tube migration.

In cases of tube-corneal touch, the

Cataract Surgery's Windfall

Surgeons have developed many invasive procedures specifically to lower IOP. However, one of the oldest procedures in eye care—in fact, in human history—has always had that effect. Though never designed to lower IOP, cataract surgery alone can reduce it by 16.5%.[1] The mechanism is not precisely known, but researchers believe it improves the function of the trabecular meshwork (TM) itself, rather than aqueous access to the TM.[2] However, for patients with pre-existing glaucoma, the change is slight at best and can be complicated by prior surgery and elevated IOP at the outset.[3,4] Clinicians must carefully manage expectations for cataract patients who already have glaucoma before touting it as a stone that can kill two birds.

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tube will likely need to be trimmed as corneal endothelial decompensation can occur, along with corneal edema. If the corneal edema persists after the tube is trimmed and a conservative approach of topical steroid is initiated, a partial thickness corneal transplant procedure, such as Descemet's membrane endothelial keratoplasty or Descemet's stripping endothelial keratoplasty, may be necessary.

A phenomenon called the ocular hypertensive phase usually occurs one to three months following surgery. It is due to a capsular fibrosis, and treatment is with aqueous suppressants and topical steroids.

Tube migration or tube exposure can lead to endophthalmitis. If the device is partially or completely exposed it may need to be removed to prevent an ocular infection.

Traditional incisional glaucoma surgeries continue to be an indispensable tool in glaucoma, particularly when medications, laser and MIGS fail to lower IOP adequately and stop progression. Although many of these procedures have significant complications that need to be managed in the postoperative period, they also have powerful pressure-lowering abilities. ■

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24TH ANNUAL GLAUCOMA REPORT
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GLAUCOMA:
LIFESTYLES OF THE ANTIOXIDANT RICH AND FAMOUS

Nutrition and other health choices can affect your patients' risk of disease and progression. Here's what you need to know to educate them properly.

By Matthew Hochwalt, OD, Heather Spampinato, OD, and Meaghan Horton, OD

Environmental factors, diet and lifestyle habits have long been associated with chronic health conditions such as diabetes and cardiovascular disease.[1-5] Today, a number of studies are focusing on how these factors affect glaucoma as well, and while research is growing, many commonly accepted associations have yet to be proven in the literature.

Optometrists must arm themselves with the knowledge patients seek so they can answer questions about diet and lifestyle factors, and guide patients toward healthy choices that help to work against glaucomatous progression. Although patient access to health information is ever increasing in the digital age, your recommendations as a healthcare provider likely make a bigger impact on treatment compliance.[6]

The Theory of Everything Glaucoma

Glaucoma is a complex chronic disease currently understood based on two simplistic models. The vascular model suggests that nerve damage occurs due to reduced or fluctuating blood flow to the optic nerve, while the mechanical model states that intraocular pressure (IOP) compresses and kills the optic nerve neurons.[7] Research is also discovering differences at the chemical level of glaucoma patients compared with controls, such as an increase in free radicals (oxidation).[8,9] Until we have a better grasp on glaucoma's mechanism of action, any management with micronutrients through diet or supplementation is still based mostly on theory.

Antioxidants are the focus of most research regarding diet and glaucoma. Oxidation—when an electron is transferred and creates free radicals—is a byproduct of normal cell functions, but can also be produced by stress or environmental toxins.[10] Retinal ganglion cell (RGC) mitochondria produce a large amount of free radicals due to their immense energy demands.[11] Research suggests free radicals interact with light

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Goal Statement: While more research is needed to better understand nutrition and lifestyle's role in glaucoma, what we do know suggests some modifications—such as eating foods rich in antioxidants, adding some supplements and even sleeping differently—can be beneficial. This article discusses the current literature on the effects of nutrition and lifestyle on glaucoma risk and progression, so practitioners can be prepared to educate and counsel patients appropriately.

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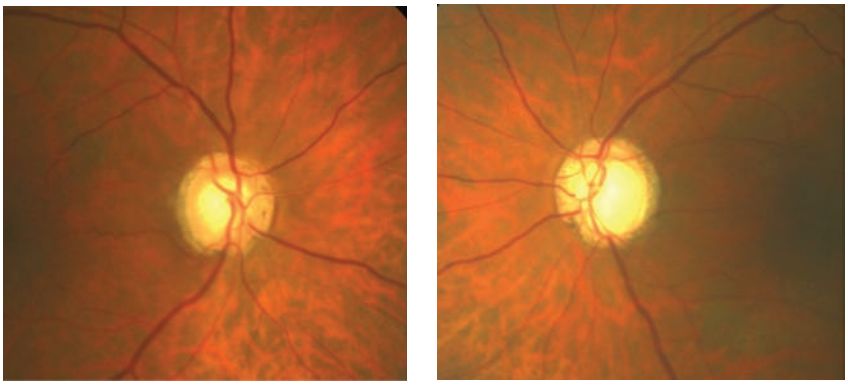
to directly damage RGCs, and they may also damage the trabecular meshwork, which could compromise aqueous outflow and increase IOP.[9] Antioxidants help to stabilize these free radicals by donating electrons to reduce their reactivity.[10]

While increased IOP is currently the main target of glaucoma therapy, research suggests neurodegeneration, including vascular dysregulation, oxidative stress and apoptosis secondary to glutamate toxicity, is also a contributing factor to glaucomatous damage.[12-14]

Retinal blood vessels' vascular tone is closely regulated by nitric oxide and endothelin interaction, and patients with normal tension glaucoma (NTG) have an increase in plasma endothelin sensitivity and a decrease in nitric oxide levels.[15] On a molecular level, endothelin-1 binds to ETA receptors located on vascular smooth muscle cells, causing an influx of calcium and thus vasoconstriction, decreased blood flow and ischemia, the last of which leads to cellular dysfunction and glutamate retention in intercellular spaces. In glaucoma patients, plasma concentrations of endothelin-1 are increased, reducing blood flow to the optic nerve head and impairing axoplasmic transport.[14]

Another dietary consideration is glutamate—a major excitatory neurotransmitter in the retina. Excess glutamate results in increased intracellular calcium levels, increased free radical accumulation, oxidative stress, mitochondrial dysregulation and activation of nitric oxide synthase.[13,16] Investigators suspect glutamatergic injury is the main cause of RGC death, or apoptosis, in glaucoma.[16] When neuronal cells undergo apoptosis secondary to ischemia or circulation disruption, they release additional glutamate, calcium, nitric oxide and free radicals into the extracellular environment, further propagating RGC damage and, ultimately, death.[17]



Glaucoma patients with asymmetric cupping/thinning, as seen here, may benefit from some diet modifications such as increased intake of foods rich in antioxidants.

Regardless of the exact mechanism, RGC death is the final common pathway of all forms of glaucoma. Therefore, any therapeutic approach to protect these cells—whether medicinal, dietary or lifestyle—can significantly widen the field of glaucoma treatment.[18]

You Are What You Eat

Science continues to look for specific foods that can help manage glaucoma in conjunction with traditional medical intervention. Unfortunately, the effects of diet on glaucoma are difficult to prove scientifically for several reasons. A typical diet provides more than 25,000 bioactive food constituents and an infinite combination of macro and micronutrients.[19] Given this complexity, it is nearly impossible to isolate variables. Added to that, nutrient absorption and use from a certain food can be affected by other foods in the diet. Patient variability is also a challenge, as not all individuals absorb and use nutrients the same way. Thus, a dietary change for one individual will not have the same effect as it might for another individual.[20]

Despite these hurdles, research has uncovered interesting data concerning antioxidant-rich foods and the dietary intake of carbohydrates and essential fats and their effects on IOP and neuroprotection.

Plants. A diet low in fruits and vegetables is associated with a higher prevalence of many chronic systemic and ocular diseases.[11] Researchers believe this is due to the high concentrations of bioactive constituents called phytochemicals, a majority of which are antioxidants, in plant-based foods.[21] Glaucoma patients in particular show a lower total plasma antioxidant level.[8]

Studies on the association between a higher intake of fruits and vegetables and decreased odds of glaucoma offer mixed results. One study of older African American women shows a significantly decreased risk of glaucoma with a diet high in fruits and vegetables.[22] Another study found specific vegetables such as carrots and leafy green vegetables may be associated with a decreased risk of glaucoma.[23] However, the Rotterdam study shows only a decreased risk of glaucoma with dietary vitamin A intake, with all other antioxidants showing no significant benefit.[24] They also found a possible increased risk of glaucoma with dietary magnesium intake.[24] Other investigators found diets high in carotenoids, vitamin C and vitamin E had no significant correlation with the risk of developing glaucoma.[9] Some even found an increased risk of glaucoma

with higher consumption of orange juice and spinach.[23]

A vegetarian diet is an excellent option for incorporating foods high in antioxidants. While fruits and vegetables are phytochemically dense, other plant-based foods show high antioxidant levels as well. Tea, coffee, nuts and seeds contain a significant amount of antioxidants, and some spices and herbs are extremely high in antioxidants as well.[21]

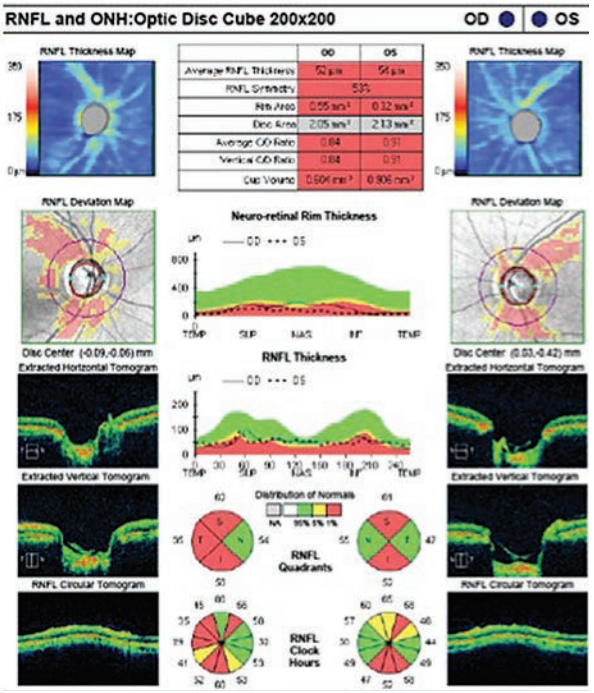
Nitrates are substances found in high concentrations in green leafy vegetables and may also be protective against glaucomatous damage, especially in individuals with early paracentral visual field loss.[25]

Carbohydrates. Instead of using antioxidants to neutralize free radicals, limiting carbohydrates can reduce the production of free radicals altogether. Although little research examines the direct relationship of high carbohydrate diets and glaucoma, some investigations show that individuals on a high protein diet have better insulin sensitivity and B-cell function, decreased oxidative stress and inflammatory cytokines compared with those on high carbohydrate diets.[26] Researchers also believe mitochondrial dysfunction secondary to oxidative stress may be a risk factor for glaucoma.[27]

A high glycemic index is also associated with an increase in advanced glycation end products (AGEs) in the retina and brain.[28] AGEs are formed from proteins that are nonenzymatically modified by carbohydrates and are associated with cytotoxicity and oxidative stress. These AGEs can induce oxidation and inflammation within the body.[29]

The ketogenic diet, typically consisting of approximately 80% fat, 15% protein and 5% carbohydrates, reduces oxidative stress through the restriction of carbohydrate intake.[30-32] Over time, the body's energy is mainly derived from the oxidation of fatty acids in the mitochondria, producing ketone bodies as the main energy substrate, a process called ketosis.[30] This can be neuroprotective in diseases such as Alzheimer's and Parkinson's, and is possibly neuroprotective of the RGCs.[33,34] One study found that during ketogenic conditions, the brain develops metabolic efficiency by stimulating mitochondrial biogenesis.[35] Other investigators found beta-hydroxybutyrate and acetoacetate, compounds produced during metabolism of fatty acids, can also protect neuronal cells from oxidative stress in a rat model.[36,37]

Fats. The two essential unsaturated fats most frequently studied are omega-3 and omega-6.[10] Dietary omega-3 fats, which produce arachidonic acid, are competitive substrates for many enzymes within the eyes. Arachidonic acid and omega-6 derived eicosanoids are precursors of prostaglandin F2-alpha, a well-established ocular hypotensive agent similar to frequently prescribed prostaglandin analog medications.[38,39] A diet with a lower omega-3:omega-6 ratio has been shown to reduce the risk of glaucoma in some studies.[38,40] Omega-3s, however, have been shown to possibly reduce the risk of glaucoma and have been associated with lower IOP, increased ocular blood flow and improved optic neuroprotection.[40,41] One study shows primary open-angle glaucoma (POAG) patients tend to have lower levels of circulating omega-3s.[42] Most studies agree that a 1:4 ratio of omega-3 to omega-6 is ideal for an individual's health, but most western diets range from 1:15 to 1:30.[38,43]



Significant RNFL loss in a patient with advanced glaucoma. While the exact mechanism of glaucoma is unknown, the final result is always death of retinal ganglion cells.

The Mediterranean diet, high in antioxidants and omega-3s, has long been studied for its many proven health benefits, one of which may be protection for glaucoma patients.[44] The diet consists mainly of olive oil, legumes, unrefined cereals, fruits, vegetables, fish and moderate consumption of dairy and wine—creating an omega-3:omega-6 ratio of 1:2.60.[45] Others found that individuals with type 2 diabetes on the Mediterranean diet did have a reduced risk of glaucoma.[46]

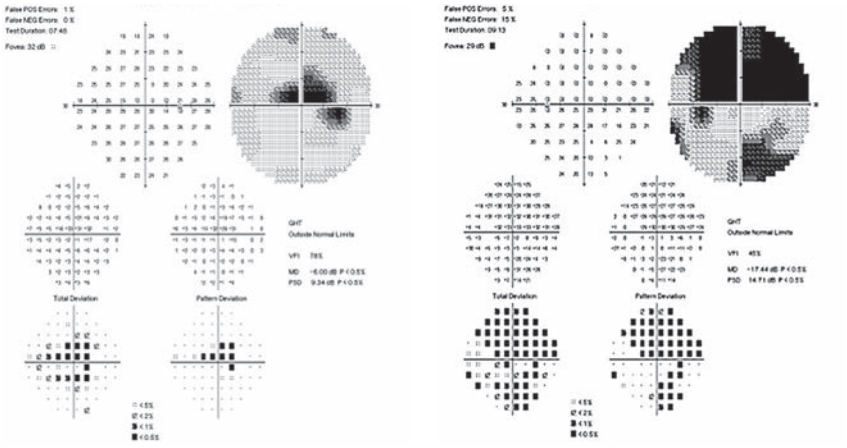
Dietary Additions

Patients often ask about supplements touted to have beneficial health effects. These options have some promising research to support their possible positive effects on patients with glaucoma:

Gingko biloba extract (GBE). This contains flavonoids and the terpene lactones ginkgolides and bilobalide, along with several other organic acids. Flavonoids act primarily as free radical scavengers, particularly of nitric oxide, and dilate blood vessels through the release of endothelium-derived relaxing factor and prostacyclin, while also decreasing blood viscosity by antagonizing platelet activating factor.[18,47-49] Inhibition of platelet activating factor reduces glutamate excitotoxicity, thereby offering a neuroprotective effect.[49] One study demonstrated neuroprotective effects of GBE in RGCs of rats with moderately elevated IOP, primarily through reduction of nitric oxide toxicity.[18]

Studies evaluating the effect of GBE on ocular blood flow show increases in both peak systolic velocity and end diastolic velocity in the ophthalmic artery of healthy subjects.[47] GBE may also increase blood flow through the central retinal and short posterior ciliary arteries in patients with ocular vascular deficiencies such as glaucoma.[47] In patients with NTG, GBE can increase peripapillary blood flow, volume and velocity.[48] Researchers demonstrated that 40mg of GBE TID for four weeks resulted in a statistically significant improvement in both mean deviation (MD) and pattern standard deviation (PSD) on visual field testing compared with baseline in NTG patients who previously had progressive visual field damage with no significant IOP change.[50] However, the visual field improvements were not maintained after discontinuation of GBE treatment, suggesting therapy would have to be long term.

While efficacy and safety reports suggest a daily dose of 120mg is acceptable and sufficient for most healthy adults, further studies are needed to better define glaucoma treatment recommendations.[14]



Nitrates found in green leafy vegetables may be beneficial for patients with early paracentral loss to help avoid advanced field loss, as seen here.

Flavonoid anthocyanins. These have strong antioxidant properties, inhibit platelet activation and promote collagen synthesis and decreased capillary permeability.[17,49] Anthocyanins from *Vaccinium myrtillis*, or bilberry, can improve best-corrected visual acuity in patients with NTG, likely through effects on ocular blood flow.[49] Oral bilberry extract may also provide a neuroprotective effect to RGCs after optic nerve damage through the reduction of oxidative stress and endoplasmic reticulum stress, both of which can signal apoptosis.[51]

Anthocyanins in black currants can induce endothelin-dependent vessel dilation, resulting in increased blood flow to the inferotemporal rim of the optic nerve head in glaucoma patients.[52] Research also shows daily systemic administration of 50mg of black currant anthocyanins can decrease IOP by an additional 0.6mm Hg in glaucoma patients treated with prostaglandins.[52]

Coenzyme Q10. Also known as CoQ10 or ubiquinone, this is an antioxidant that specifically targets mitochondria and can exhibit neuroprotective activity in a number of neurological disorders, including Parkinson's and Huntington's disease.[53] It maintains mitochondrial membrane potential and inhibits reactive oxygen species generation, thereby protecting neuronal cells from oxidative stress.[16] The level of CoQ10 in the human retina declines by up to 40% with age, suggesting RGCs become more susceptible to glaucomatous neurodegeneration over time.[16,53] In one study, dietary supplementation with CoQ10 over the course of six months in glaucomatous mice promoted RGC survival through the inhibition of oxidative stress, glutamate excitotoxicity and apoptosis.[16]

Another recent study demonstrates a significant neuroprotective effect against RGC loss with topical application of CoQ10 in rats with surgically induced ocular hypertension.[53] After three weeks of once-daily application, the number of apoptotic RGCs in the rats was significantly reduced without any effect on IOP.[53]

Magnesium. This supplement works to inhibit intracellular calcium influx and glutamate in the retina, thereby reducing vascular dysfunction, oxidative stress and RGC apoptosis.[54,55] Magnesium deficiency has been associated with retinal pigment epithelium necrosis and myelination disorders of the optic nerve in rats, as well as pigmentary retinal degenerations such as retinitis pigmentosa in

humans.[55] Magnesium supplementation is relatively safe compared with prescription calcium channel blockers, which have been known to cause hypotension, bradycardia and decreased cardiac output.[54,55]

In one study, researchers evaluated the effect of oral magnesium on visual field and ocular blood flow in normotensive glaucoma.[54] Patients received 300mg of oral magnesium citrate in suspension for one month. Within that time, patients had a statistically significant improvement in both MD and PSD on visual fields compared with the control group.[54] Another study showed that 121.5mg of magnesium BID for one month improved peripheral circulation as measured by blood flow velocity and number of capillaries.[56]

Melatonin. This antioxidant acts as a scavenger of light-induced free radicals in the retina, protecting photoreceptor outer segment membranes.[15,57] Retinal melatonin levels are regulated by the interaction between the circadian clock and light exposure, rapidly rising during dark and decreasing after light exposure.[16] Evidence suggests neuroprotective benefits of melatonin through the reduction of nitric oxide and subsequent increase in the uptake of glutamate from the retina.[14,58]

Hormone replacement therapy. Females have a lower incidence of POAG compared with males up until the age of 80, which has led researchers to investigate the possible role hormones play in the pathogenesis of glaucoma.[59] Studies show that estrogen both reduces RGC loss and decreases IOP in women who undergo hormone replacement therapy.[60]

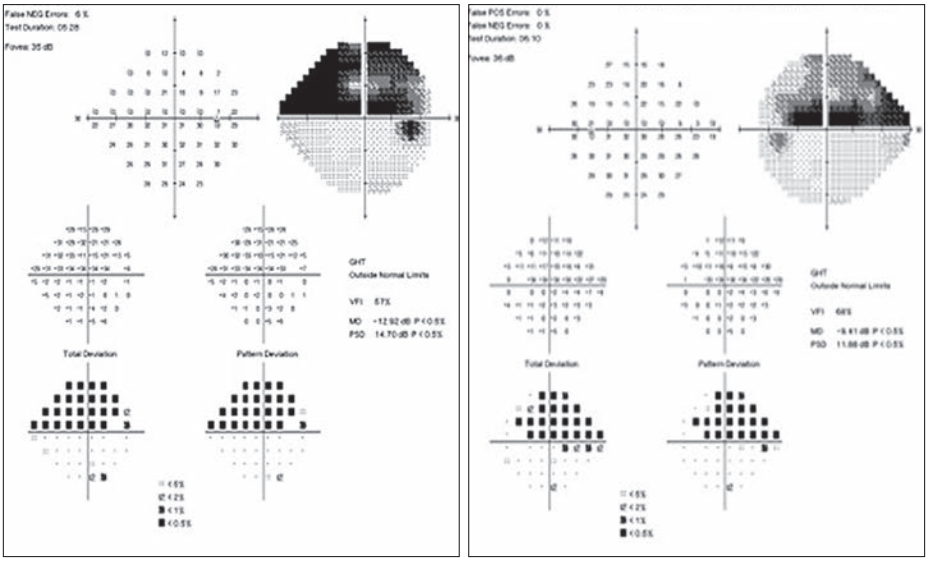
While the exact mechanism through which estrogen provides a neuroprotective effect in glaucoma remains unknown, a few hypotheses exist. One is that estrogen activates the growth of collagen and increases the elasticity of various ocular tissues, resulting in decreased IOP. Within the lamina cribrosa, collagen changes lead to a decrease in compression on RGC axons, thereby increasing survival rates of these axons in glaucomatous disease.[60] In addition, some studies suggest estrogen causes greater blood flow within retinal arteries, particularly the inferotemporal retinal artery, in postmenopausal women on hormone replacement therapy.[61] This effect may be due to upregulation of nitric oxide, which causes vasodilation and the antagonizing effects on endothelium-derived contracting factor, which leads to vasoconstriction.[61] Researchers also observed increased neuroretinal rim volume, mean retinal nerve fiber layer (RNFL) thickness and RNFL cross-sectional area, especially in the inferotemporal region of the optic nerve head.[61]

Get Your Life Together

Several lifestyle habits and environment factors can affect IOP.[62-65]

Sleep position. Research shows IOP is higher when sleeping in the supine position compared with other sleep positions; sleeping with the head elevated 20 to 30 degrees may lessen the effect of the supine position.[63,65-67] A recent study of normal tension and high tension glaucoma patients with asymmetric visual field loss found that of those patients preferring to sleep on their side, approximately two-thirds had worse field loss on their sleep-dependent side.[68] However, another study found worse visual field loss in the non-dependent side eye.[69] Other researchers found a small, but significant, increase in IOP for the eye on the sleep-dependent side relative to the non-dependent side.[70] Clinical application of these findings is not well defined, but it is reasonable to recommend a slight head elevation and possible avoidance of sleeping on the side of the worse eye when significant asymmetry is present.

Elevated body mass index (BMI). Associations between BMI and glaucoma are far less consistent than those



This patient with advanced POAG has a history of selective laser trabeculoplasty OU and is currently on maximum topical therapy. Dietary and lifestyle changes may be particularly important in this case to help supplement the patient's medical management.

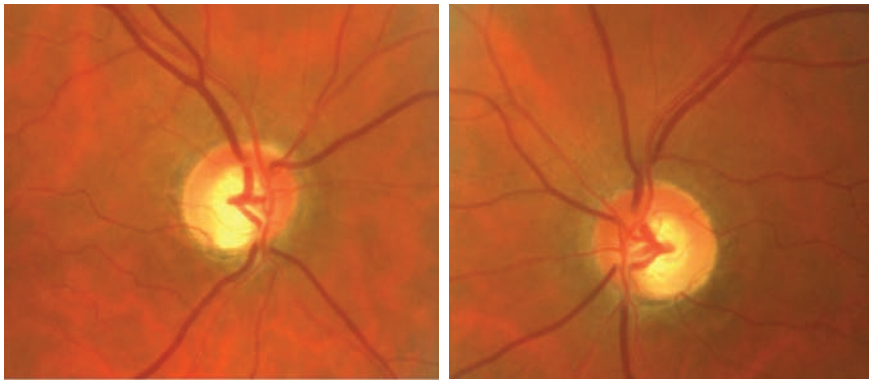
with stroke, hypertension, coronary artery disease and type 2 diabetes.[71] Studies have found elevated IOP is associated with obesity, but others found a decrease in the incidence of glaucoma with obesity.[72-76]

Smoking. While this lifestyle habit affects almost all organs, insufficient evidence currently exists to link cigarette smoking to POAG.[77,78] Small case-controlled studies led to mixed results, and both large population studies and further evaluation of observational studies by meta-analysis show no association between cigarette smoking and POAG.[79-83]

Some early studies of cigarette smoking and IOP found a transient but significant increase in IOP following inhalation.[84,85] However, a more recent study in young healthy smokers found no significant increase in IOP immediately after smoking.[86] The Blue Mountain Eye Study found a 0.26mm Hg higher IOP in chronic smokers vs. non-smokers.[87] Similar results were found in a large UK-based cross-sectional study published in 2016 with more than 100,000 patients; the authors found a 0.19mm Hg higher IOP in smokers vs. non-smokers.[88]

A retrospective study of glaucoma progression in a Japanese population found an association between smoking and progression of inferior visual field defects in glaucoma patients.[89] Although another study found smoking was associated with faster glaucoma progression, this is an area with a scarcity of research.[90]

Alcohol. Several studies show a dose-dependent and transient lowering of IOP shortly following consumption of alcohol.[90-93] This lowering effect is not dependent on the type of alcohol consumed, lasts for two to five hours and may be from one to several mm Hg.[91,92] Consuming large quantities of alcohol, though, may increase IOP through a hypervolemic effect.[94] Although alcohol causes a temporary decrease in IOP, large population studies show no association between alcohol consumption and POAG risk.[75,79,80] Some studies even show an inverse relation in glaucoma risk and alcohol consumption.[95,96] Even though there may be some health benefits to moderate alcohol consumption, there is not convincing evidence to suggest a benefit for glaucoma patients.

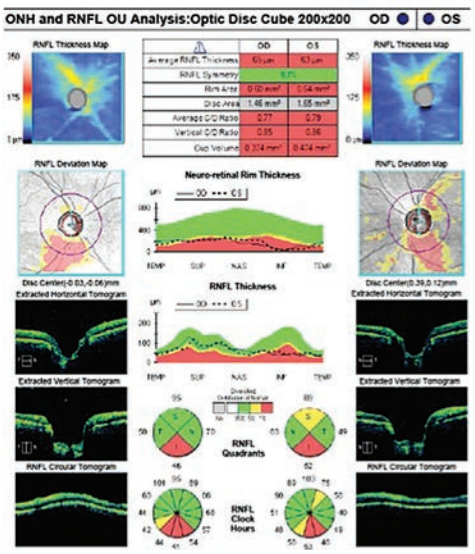


This glaucoma patient has significant inferior notching.

Exercise. Moderately intense aerobic exercise lowers IOP in individuals with and without glaucoma, though the effect may be more pronounced in glaucomatous eyes.[69,97,98] Exercise of increasing intensity shows a larger reduction in IOP.[99] The effect of exercise may only last an hour in unconditioned individuals and will also fade in fit individuals within weeks of deconditioning.[100-102] Exercise does cause a decrease in blood pressure and possibly ocular perfusion pressure, but whether this impacts glaucoma risk or progression is unknown.[103]

Some specific forms of exercise that induce positional exertion and Valsalva type maneuvers increase IOP. Weight lifting can cause a transient increase in IOP by several mm Hg that dissipates following completion of the exercise.[104] Yoga positions that involve a headstand or that place the eyes below the heart can almost double IOP temporarily, but the IOP returns to baseline a few minutes later.[65,94]

Although research continues to lag behind patient interest, optometrists must remain up-to-date on the information currently available on the many ways a patient's diet, nutritional supplementation, exercise and habits can affect their IOP and glaucoma risk. For most patients,



Anthocyanins in black currants may increase blood flow, particularly to the inferotemporal rim of the optic nerve head in glaucoma patients such as this one with glaucomatous thinning predominantly in the inferior and inferotemporal region of the optic nerve head.

broad recommendations for lifestyle modification are not warranted due a lack of compelling evidence linking them to glaucoma. Clinicians can instead focus on patients at substantial risk for progression and those with advanced disease. Regardless of the patient's disease status, proper education and prudent lifestyle counseling can ensure they remain healthy to ward off glaucomatous progression whenever possible. ■

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1. What structure of the eye is believed to be affected by free radicals?
a. Trabecular meshwork.
b. Retinal ganglion cells.
c. Ciliary body.
d. Both a and b.

2. Excess glutamate results in all of the following, except:
a. Activation of nitric oxide synthase.
b. Decreased free radical accumulation.
c. Increased intracellular calcium.
d. Mitochondrial dysregulation.

3. Which of the following is true about smoking and glaucoma?
a. It may cause a small chronic decrease in IOP.
b. It can increase the risk of developing POAG.
c. Observational studies have failed to show a significant link between smoking and POAG.
d. Both a and c.

4. Sleeping with the head elevated ____ degrees has been shown to lessen the effect of the supine position on IOP.
a. 10.
b. 20 to 30.
c. 30 to 40.
d. 45.

5. The Mediterranean diet is high in:
a. Antioxidants.
b. Omega-3s.
c. Antioxidants and omega-3s.
d. Antioxidants and omega-6s.

6. A diet high in _____ has been shown to increase oxidative stress and inflammatory cytokines.
a. Proteins.
b. Carbohydrates.
c. Fats.
d. Fruits and vegetables.

7. Research shows the addition of which supplement can provide an additional IOP reduction of 0.6mm Hg in glaucoma patients already treated with prostaglandins?
a. Black currant.
b. Ginkgo biloba extract.
c. Magnesium.
d. Melatonin.

8. Ginkgo biloba extract has all of the following effects, except:
a. Dilation of blood vessels.
b. Decreased blood viscosity.
c. Increased glutamate excitotoxicity.
d. Inhibition of platelet activating factor.

9. What effect does alcohol consumption have on IOP?
a. It increases IOP transiently.
b. It decreases IOP transiently.
c. It has no effect.
d. It decreases IOP transiently except in cases where large quantities are consumed.

10. Which of the following has been shown to increase the risk of POAG?
a. Alcohol consumption.
b. Physical inactivity.
c. Elevated IOP.
d. High BMI.

11. In patients with normal tension glaucoma, the addition of which supplement resulted in a statistically significant improvement in MD and PSD on visual field testing?
a. Coenzyme Q10.
b. Magnesium.
c. Ginkgo biloba extract.
d. Both b and c.

12. Approximately how many bioactive food constituents are there in a typical diet?
a. 25,000.
b. 10,000.
c. 500,000.
d. 2,500.

13. Which of the following is not a good source of dietary antioxidants?
a. Fruits and vegetables.
b. Nuts and seeds.
c. Lean meats.
d. Coffee.

14. Hormone replacement therapy with estrogen may result in:
a. Increased neuroretinal rim volume.
b. Increased retinal neve fiber layer thickness.
c. Increased blood flow in the inferotemporal retinal artery.
d. All of the above.

15. Nitrates, which are found in high

OSC QUIZ

concentrations in _____, may be protective against glaucoma damage, especially in individuals with early paracentral visual field loss.
a. Coffee.
b. Olive oil.
c. Orange juice.
d. Green leafy vegetables.

16. Which of the following is true about aerobic exercise and glaucoma?
a. It increases IOP.
b. It may increase ocular perfusion by temporarily increasing blood pressure.
c. The effect on IOP will persist even after an individual is deconditioned.
d. None of the above.

17. Which of the following does not occur as a result of apoptosis secondary to ischemia?
a. Release of glutamate.
b. Increase of nitic oxide.
c. Decreased intracellular calcium.
d. Increased free radical accumulation.

18. What exercise can almost double IOP temporarily before returning to baseline after a few minutes?
a. Weight lifting.
b. Yoga in head down positions.
c. Aerobic exercise.
d. Curling.

19. What term is used to describe bioactive dietary constituents, most of which are mainly composed of antioxidants?
a. Superfoods.
b. Ketone bodies.
c. Phytochemicals.
d. Nutramolecules.

20. Coenzyme Q10 works to protect neuronal cells from oxidative stress by:
a. Maintaining mitochondrial membrane potential.
b. Promoting collagen synthesis.
c. Reducing vascular dysfunction.
d. Upregulation of nitic oxide.

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4. Ⓐ Ⓑ Ⓒ Ⓓ
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19. Ⓐ Ⓑ Ⓒ Ⓓ
20. Ⓐ Ⓑ Ⓒ Ⓓ

Post-activity evaluation questions:

Rate how well the activity supported your achievement of these learning objectives:
1=Poor, 2=Fair, 3=Neutral, 4=Good, 5=Excellent

21. Improve my understanding of the role of antioxidants in glaucoma. ① ② ③ ④ ⑤
22. Add to my knowledge of the role diet plays in a patient's risk for glaucomatous damage. ① ② ③ ④ ⑤
23. Recognize the positive effects of certain supplements in protecting ocular structures. ① ② ③ ④ ⑤
24. Better understand the effects of sleep position on glaucoma progression. ① ② ③ ④ ⑤
25. Improve my knowledge of lifestyle changes such as smoking and alcohol consumption on glaucoma risk. ① ② ③ ④ ⑤
26. Improve my ability to counsel patients on their diet and lifestyle choices that affect their risk of glaucoma. ① ② ③ ④ ⑤

Rate the quality of the material provided:
1=Strongly disagree, 2=Somewhat disagree, 3=Neutral, 4=Somewhat agree, 5=Strongly agree

27. The content was evidence-based. ① ② ③ ④ ⑤
28. The content was balanced and free of bias. ① ② ③ ④ ⑤
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Lesson 116991 RO-OSC-0718

Concerns About the Choroid

Here are three important things to look for. **By Bisant A. Labib, OD**

We all know the highly vascularized choroid supplies blood to nourish the outer retina with oxygen and nutrients.[1,2] But it also serves other purposes, and its dysfunction is the source of several pathological conditions we often come across in practice.[2] Be especially vigilant for these three.

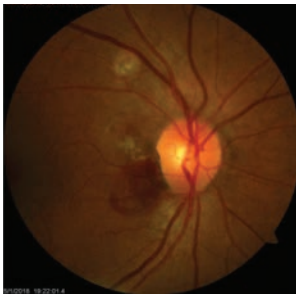

Fig. 1. PCV signifies subretinal bleeding.

1. Vascular Abnormalities

The choroid comprises the most posterior portion of the uvea. It is predominantly made up of blood vessels and, to a lesser degree, fibroblasts, melanocytes and supporting connective tissue. In fact, the choroid is the most highly vascularized structure in the entire body, given the high rate of blood flow necessary to nourish the retina.[2] This is made possible through the presence of choriocapillaris, a highly anastomosed network of fenestrated blood vessels, which is thickest at the fovea and thins peripherally.

The blood vessels are classified into two layers: *Haller's layer*, which lies adjacent to Bruch's membrane and contains large diameter vessels, and *Sattler's inner layer*, which houses medium and small vessels that feed the capillary network and lies closer to the scleral surface.[1,2]

Due to this abundance of blood supply and flow, the choroid is the most common site for metastatic disease, accounting for over 90% of uveal metastasis.[3] Cancers originating in the breast (47%), lung (21%) or gastrointestinal tract (4%) are the most common to metastasize by hematogenous spread, so highly vascularized areas such as the choroid are most likely to be affected.[4]

Other vascular abnormalities may arise in the choroid as well, such as bleeding or the formation of choroidal neovascular membranes. For example, polypoidal choroidal vasculopathy (PCV) is an idiopathic condition where bleeding occurs in the peripapillary subretinal area *(Figure 1)*.[5]

2. Lymphatic Dysfunction

In addition to the choriocapillaris, the choroid contains multiple lacunae. In animal studies, these lacunae have been identified as a site of lymphatic drainage.[2] The presence of lymphatics in the human choroid is controversial and not yet well established, though most recent evidence points to the existence of nontraditional lymphatics.[6] The lymphatic system generally functions to preserve fluid homeostasis and plays a key role in immunosurveillance.[6,7] In vertebrates it may also be implicated in tumor metastasis, further contributing to choroidal metastasis.[7]

An imbalance of tissue fluid homeostasis may alter permeability as well as choroidal thickness.[2] Increased choroidal permeability is also implicated in central serous chorioretinopathy, which manifests as a serous retinal detachment with or without the presence of pigment epithelial defects.[8]

Choroidal thinning has been associated with many pathological conditions, such as age-related macular degeneration, high myopia and ocular growth.[9] Additionally, since lymphatics are known to play a fundamental role in immunosurveillance, the choroid is susceptible to uveitis and other choroidal inflammatory conditions.[6]

3. Melanoma

Unlike in animals, the human choroid represents the site of large and abundant melanocytes, which give the choroid its dark pigmentation and appearance. These melanocytes are situated in close proximity to the choroidal blood vessels.[2] Besides pigmentation and light absorption, the function of melanocytes is unknown. However, their presence allows for cancer to arise in the form of a primary malignant choroidal melanoma.[10] ■

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NEW WORLD MEDICAL

Don't Berry this Clinical Finding

Retinal astrocytic hamartomas are the most common ocular finding in tuberous sclerosis complex. **By Carlo J. Pelino, OD, and Joseph J. Pizzimenti, OD**

Tuberous sclerosis complex (TSC) is an inherited, multisystem disorder characterized by hamartomatous growths that can occur in virtually any organ or tissue. Ophthalmic features associated with TSC can be divided into retinal and non-retinal.[1] This case highlights a classic presentation of posterior segment findings in a young patient with TSC.

The Case

A 15-year-old male was referred to the eye clinic with no ocular or visual complaints. He had recently been diagnosed with TSC by his primary care provider (PCP) and neurologist, and his PCP wanted to rule out any ocular complications. His history was negative for epilepsy and intellectual disability. Unaided visual acuity was 20/20 OD and OS. Gross exam showed skin adenoma sebaceum along his cheeks extending to his chin. Fundus examination revealed a white, mulberry-shaped lesion in the superior retina of the right eye consistent with retinal astrocytic hamartoma (*Figure 1*).

We informed the patient's primary physician about the clinical findings and scheduled the patient for a six-month follow-up visit.

A Bushel of Associations

The term *hamartoma* refers to abnormal growth of mature cells native to that area of the skin, blood vessels or nervous system. Phakomatoses are a group of hereditary conditions characterized by the presence of hamartomas. Some of the more common phakomatoses, several of which have prominent retinal manifestations, include neurofibromatosis types I and II, tuberous sclerosis (Bourneville's disease), encephalotrigeminal angiomatosis (Sturge-Weber syndrome), angiomatosis of the retina and cerebellum (von Hippel-Lindau disease) and racemose angioma of the midbrain and retina (Wyburn-Mason syndrome).[1,2]

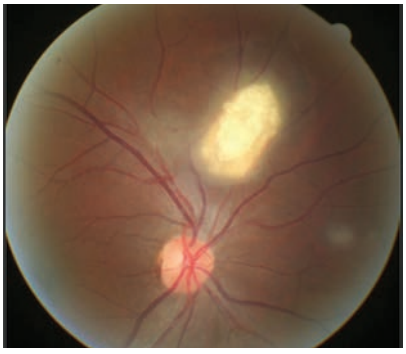

Fig. 1. A mulberry-like lesion consistent with retinal astrocytic hamartoma.

The first description of TSC is usually attributed to Bourneville in 1880; however, Vogt described the classic triad of "epilepsy, mental retardation, and adenoma sebaceum" (now called angiofibromatosis) in 1908.[2,3]

The clinical manifestations of TSC are now known to be more numerous and diverse. TSC is inherited as an autosomal dominant trait—the genes are located on chromosomes 9q and 11q—with an incidence of one in 15,000 live births. There is no race or sex predilection.[2,4,5] Systemic manifestations of TSC may include ash-leaf spots, café-au-lait spots, shagreen patches, subungual fibromas, cortical tubers of the basal ganglia, lateral ventricles and third ventricle, obstructive hydrocephalus and tumors of the bone, lung, heart, liver and kidney (*Table 1*). [5]

Ocular Fruits of TSC

Retinal associations of TSC were first noted by Van der Hoeve in 1921.[6] He termed these lesions phakomas (derived from the Greek *phakos*, meaning spot) and introduced the concept of phakomatosis. These lesions are now known to be retinal astrocytic hamartomas (RAH), sometimes called retinal astrocytoma.[1,2,4] RAH is a benign, minimally progressive, neoplastic lesion of the retinal nerve fiber layer caused by proliferation of glial cells.

These (typically) elevated yellowish-white calcific "mulberry-like" lesions are the characteristic ophthalmic finding in TSC. RAH is found in 50% to 90% of TSC patients, where they most commonly reside in the posterior pole.[1]

Three morphological types of retinal hamartomas are described in the literature: (1) the relatively flat, smooth, non-calcified, gray, translucent lesion; (2) the elevated, multinodular, calcified, opaque lesion resembling mulberries; and (3) a transitional lesion that has morphological features of both.[2,4,5]

Other posterior segment findings associated with TSC include retinal pigmentary disturbances such as hyperpigmented areas (probably

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†Oxygen levels for single vision spherical (SVS) lenses only.
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REFERENCE: 1. Data on file. Bausch & Lomb Incorporated. 3rd Party Industry Report. 2016-2018.

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congenital retinal pigment epithelium hypertrophy) to "punched out," hypopigmented areas at the posterior pole or midperiphery.[1,2]

Other ophthalmic findings include eyelid angiofibromas, iris, lens and choroid coloboma, strabismus, poliosis of eyelashes, optic disc edema and sector iris depigmentation.[1,2]

Many Hands Make Light Work

Any care provider can make a presumptive diagnosis of TSC if a patient has two of the following: infantile spasms, seizures, areas of increased attenuation in the cerebral cortex, calcified lesion in the cerebral cortex, ash-leaf spots, retinal hamartoma, dental enamel pits and multiple renal tumors.[3,5] Clinicians should consult with a pediatric neurologist for a definitive diagnosis.

Patients experience a normal life expectancy. The seizures usually present in infancy and progress to tonic-clonic seizures later in life. A patient with TSC should have an extensive evaluation that includes dermatology, optometry or ophthalmology, neurology, cardiology, urology and pulmonology.[5] These subspecialties help ensure a proper interdisciplinary approach to the patient's care. Some patients may need a shunt procedure for obstructive hydrocephalus.

There is no cure for TSC, although treatment is available for a number of the symptoms. Antiepileptic drugs may be used to control seizures. Sabril (vigabatrin, Lundbeck) is a particularly useful medication and has been approved by the Food and Drug Administration for treatment of infantile spams in TSC, although it has significant side effects.[5]

Tending Your Patients

Patients may seek routine eye care with or without an established diagnosis of TSC, and in some cases RAH, detected by the OD, may be the presenting sign of TSC. RAHs usually remain stable over time and have no effect on visual function in the majority of cases. Patients with TSC may experience visual loss secondary to cortical tubers (brain hamartomas), astrocytic hamartomas that involve the macula, optic nerve gliomas and optic atrophy secondary to hydrocephalus.[1,2]

In patients with a known diagnosis of TSC, clinicians should perform a complete ophthalmic evaluation, including dilated funduscopy, to assess for retinal lesions and visual field deficits. Ongoing periodic surveillance is needed after initial diagnosis for optimal care and prevention of secondary complications. Management of specific complications of TSC will often require input from the multidisciplinary team.[2,4,5]

The FDA recommends a baseline ophthalmic evaluation and follow-up testing every three months for patients on Sabril.[5,7,8] Its use is complicated by an irreversible, dose-dependent visual field constriction from photoreceptor toxicity. Optic neuropathy and macular pigment epithelial changes are the most common ophthalmoscopic findings.[7,8] Screening for these in young children can be challenging, especially considering many children who need the medication are non-verbal and often uncooperative. Methods of screening include serial fundus examination, serial automated static perimetry, OCT, VEP and ERG.[7,8]

Currently in the United States and many other countries, specialized TSC clinics have been established. Ideally, all TSC patients would have access to one of these clinics to ensure appropriate care and treatment. You can help patients locate a clinic at www.tsalliance.org/individuals-families/tsc-clinics. ■

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Table 1. TSC Review of Systems[1-5]

Ophthalmic: At least 50% have ocular abnormalities; most common lesions are retinal astrocytomas that become calcified over time.
Pulmonary: cystic pulmonary abnormalities occur in up to 40% of women with TSC.
Renal: Four types of lesions can occur: autosomal dominant polycystic kidney disease, isolated renal cyst(s), angiomyolipomas (AMLs) and renal cell carcinomas.
Dental: Pitting of the dental enamel; gingival fibromas.
Gastrointestinal: Hamartomas and polyposis of the stomach, intestine and colon.
Hepatic: Hepatic cysts and hepatic AMLs, typically asymptomatic and nonprogressive, with a marked 5:1 female predominance.
Skeletal: Sclerotic and hypertrophic lesions of bone.
Neurologic: Growth of tubers and the presence of subependymal nodules (SENs) and subependymal giant cell astrocytomas (SEGAs).
Cutaneous: Adenoma sebaceum, which often does not appear until late childhood or early adolescence.
Cardiac: Of individuals with TSC, 50% to 60% have evidence of early cardiac disease, mostly rhabdomyomas.

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PTC Revisited

Recognize when all the signs add up to a rare condition.

By Alan G. Kabat, OD, and Joseph W. Sowka, OD

A 25-year-old woman presented for evaluation reporting daily, recurrent, predominantly left-sided headaches that had been worsening over the past two years. She indicated that the headaches interfered with her ability to work and were associated with blurry vision and eye pain. The patient denied any diplopia, motor or cognitive issues, but did report a "fluttering" sound that was more noticeable in her left ear. She also recalled that she had suffered transient visual "blackouts" since her teen years. According to her records, a previous optometrist had referred her for a neurological evaluation and magnetic resonance imaging (MRI) of the brain, but these scans were reportedly negative. Her medical history was positive for seasonal allergies and associated sinus congestion, but was otherwise unremarkable. The only medication that the patient admitted to using was an implanted birth control device.

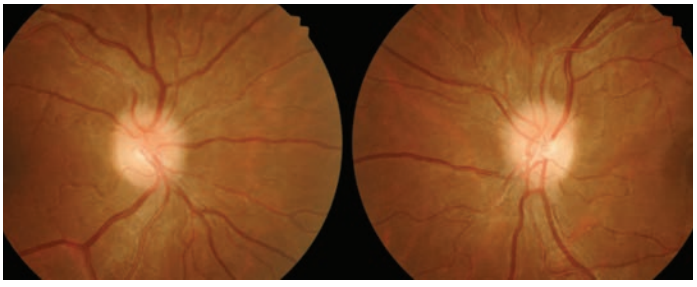

Fig. 1. Fundus photos from January 2016. Note the blurred disc margins.

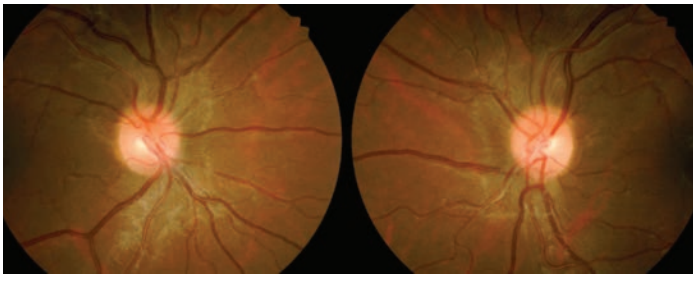

Fig. 2. Fundus images from February 2018. Note disc edema resolution.

Evaluation

The patient's uncorrected visual acuity was 20/20 OD, OS and OU. Pupils were equal and reactive, without afferent defect. Ocular motilities were smooth and unrestricted. Visual fields by confrontation were full. Her blood pressure was 118/90 and she was 5' 3" and weighed 200 lbs. Intraocular pressures (IOP) measured 18mm Hg OD and 20mm Hg OS. Dilated funduscopy revealed mildly elevated and hyperemic optic nerve heads in both eyes, with indistinct margins and a cup-to-disc ratio of approximately 0.2/0.2.

Prior records were obtained and the fundus photographs taken 21 months before were consistent with our funduscopic examination (*Figure 1*). B-scan ultrasonography had also been performed at the prior visit, which showed no evidence of buried optic nerve head drusen. While the patient had been referred for both MRI and neurologic consultation by the former optometrist, she admitted that she never followed up after learning that the MRI was "normal." Given the overall presentation, we advised the patient that a neurologic assessment was imperative, and referred her for the appropriate clinical evaluation and testing.

Diagnosis

This patient was diagnosed with pseudotumor cerebri (PTC), sometimes referred to as idiopathic intracranial hypertension (IIH). It is considered a diagnosis of exclusion. It is often suspected in cases of optic disc edema associated with headache, transient visual obscuration, intermittent horizontal diplopia, tinnitus, nausea and vomiting in young, overweight females.[1-6] However, to rule out other conditions that may present similarly (e.g., brain tumors, hydrocephalus and cerebrovascular malformations), this diagnosis requires careful neurologic assessment and radiographic evaluation.[1-5]

While the pathophysiology of PTC is not completely understood, some things about the condition are well known. True IIH (which is a specific subset of PTC) occurs most frequently in obese young women

between the ages of 15 and 45.[1-6] Overall, the condition is rare, with a worldwide incidence of just 1 to 3 per 100,000, but the number of affected women outnumber men by a ratio of up to 9:1.[6] Moreover, the incidence of IIH is approximately 15 times greater in women who are at least 10% above their ideal body weight.[6] Certain medications—such as tetracycline derivatives, cyclosporine, lithium, naladixic acid, nitrofurantoin, oral contraceptives, levonorgestrel, danaxol and tamoxifen—may precipitate PTC in susceptible individuals.[2,5] One study shows patients with mild visual loss could be controlled with a combination of acetazolamide and a low sodium diet.[7] Additionally, patients taking acetazolamide had significantly improved papilledema, quality-of-life measures and lower cerebrospinal fluid pressure.[7]

Background

The topic of birth control and PTC has drawn considerable interest over the years. Reports of neurological sequelae in association with oral contraceptives date back to the 1960's.[8-11] More recently, long-term implantable contraceptives such as Norplant (levonorgestrel, Wyeth-Ayerst) Jadelle (levonorgestrel, Bayer), Implanon and Nexplanon (etonogestrel, Merck) have likewise been implicated in some cases of PTC.[12,13] Norplant was discontinued in the United States in 2002, and Jadelle (also known as Norplant II) was never marketed, but both of these products are available in other regions throughout the world. Implanon was discontinued in the United States in 2010 and replaced by Nexplanon, a newer version of the implant designed to reduce the risk of insertion errors. Although no direct cause-and-effect relationship has been established between any of these medications and PTC, the levonorgestrel-containing products do carry a specific warning regarding IIH. Implanon and Nexplanon appear to be safer in this regard, but cases of PTC have still been reported; the authors have suggested that the mechanism of PTC in association with etonogestrel-containing products may be rapid weight gain rather than direct hormonal influence.[13]

Diagnostic Criteria for PTC (Modified Dandy Criteria)[4]

- Signs and symptoms of increased intracranial pressure (headache, nausea, vomiting, transient obscurations of vision, papilledema).
- No localizing neurological signs otherwise, with the single exception being unilateral or bilateral sixth nerve paresis.
- Cerebrospinal fluid which can show increased pressure, but with no cytologic or chemical abnormalities otherwise.
- Normal to small symmetrical ventricles must be demonstrated (demonstrated by computed tomography).

Treatment

Given that our patient displayed numerous features commonly associated with PTC, we felt confident that neurologic consultation was appropriate. After obtaining additional MRI studies of the brain and performing a lumbar puncture to assess the intracranial pressure (which ultimately was elevated), the patient was diagnosed with PTC and initiated on a therapy of oral acetazolamide (250mg QID). Due to a possible causal effect, she was also referred by the neurologist to her primary care provider for explantation of her subdermal birth control device. The patient returned to our clinic about five months after we had last seen her, reporting improvement in symptoms. Examination showed markedly improved disc edema, both on funduscopic evaluation and optical coherence tomography (*Figure 2*). Visual field testing showed no discernible pattern of loss.

While PTC is uncommon, it remains of particular importance to optometrists because visual disturbances and headache are typically the primary symptoms, and papilledema is the most readily observable sign. While one cannot make this diagnosis without complete and thorough testing, the clinical scenario is often strikingly prototypical. We urge all readers to avoid jumping to conclusions and making this diagnosis in "a typical patient profile" without obtaining the necessary evaluations detailed here. Recognizing the crucial elements of the medical history and facilitating prompt and appropriate care is the key to successfully managing these patients. ■

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The Gene Genie

A patient has experienced vision troubles all his life. Could he come by it naturally?

By Eric Dillinger, OD, and Mark T. Dunbar, OD

A 34-year-old Caucasian male presented to the clinic with complaints of progressive blurry central vision he has experienced since childhood. He's also experienced trouble seeing at night. The patient reported a male cousin and maternal grandfather suffering similar vision loss, which progressed later in life.

On examination, the patient's best-corrected visual acuities were 20/40 OD, 20/60 OS with a prescription of -7.00D OD, -6.75D OS. Pupils were sluggish, with minimal response to light, confrontation visual fields were constricted to a central 30 degrees in each eye and extraocular muscles were full in the presence of horizontal nystagmus in both eyes.

Color vision, measured with D-100 color plate, showed only eight correct with the right eye, and seven with the left. Intraocular pressures (IOPs) were 16mm Hg OD and 14mm Hg OS with Tonopen (Reichert). Dilated fundus exams were also performed (*Figure 1*). Additionally, we obtained OCT images (*Figure 2*).

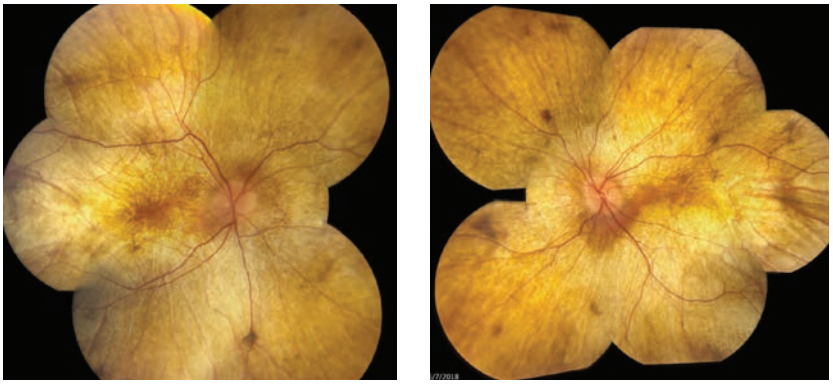

Fig. 1. Fundus photos show our 34-year-old patient's right, at left, and left eyes.

Take the Retina Quiz

1. Which best describes the fundus appearance in both eyes?
a. Diffuse retinal whitening, sparing the central macula.
b. Diffuse outer retinal atrophy with exposed sclera, sparing the macula.
c. Vascular attenuation, peripheral bony spicules and disc pallor.
d. Pisciform flecks with central retinal atrophy.

2. What is the correct diagnosis?
a. Gyrate atrophy.
b. Choroideremia.
c. Ocular albinism.
d. Pathologic myopia.

3. How would you describe the OCT findings?
a. Thin inner retina with mild cystoid macular edema.
b. Subretinal hyperreflectivity with a thin choroid.
c. Significant outer retinal atrophy with choroidal thinning and outer retinal tubulations.
d. Thin retina with abnormally thick choroid.

4. Based on the images and presentation, what is the appropriate next step?
a. Send for genetic testing.
b. Send for stroke evaluation.
c. Send for low vision evaluation.
d. Intravitreal anti-VEGF injection.

For answers, see page 90.

Diagnosis

Based on the clinical presentation and history, our patient was tentatively diagnosed with chorioderemia (CHM) and sent for genetic testing, which confirmed the diagnosis. CHM is an x-linked recessive chorioretinal dystrophy, which affects approximately one in 50,000 to 100,000 individuals.[1,2] Since the development of genetic confirmation, more than 100 variations in the CHM gene have been discovered, opening the door for gene therapy.[1,2]

The CHM gene is responsible for the production of Rab escort protein-1 (REP-1), which is one of only two Rab escort proteins throughout the body. REP-1 helps to transport lipid membrane-bound structures (Rab) intracellularly, which plays a role in the transport of proteins from the Golgi apparatus to the outer segments in photoreceptors, and phagocytosis/degradation of

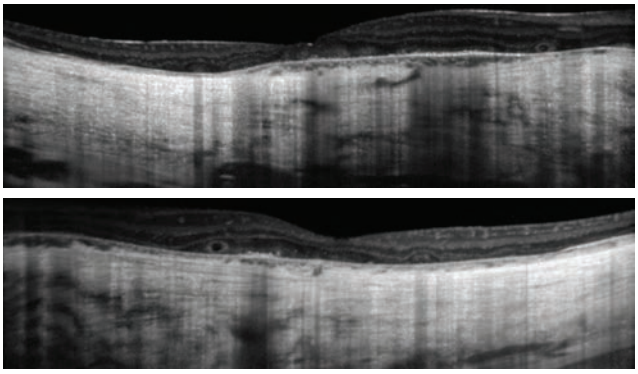

Fig. 1. These OCT images show our patient's right, at top, and left eyes. The patient has had visual complaints since childhood.

outer segments shed by the RPE cells. Unfortunately, the other Rab escort protein cannot take over the function in the event of a defective REP-1.[3] Ultimately, this results in degeneration to the photoreceptors (rods and cones), RPE and choriocapillaris.

Initial signs of diffuse RPE pigment clumping can be confused with bone-spicule pigment clumping associated with retinitis pigmentosa (RP), but can differentiated because RP pigment clumping is typically perivascular.[4]

Large areas of atrophy soon develop at the equator, extending centrally to the arcades, then the peripapillary region revealing sclera and choroidal vessels. The fundus appears stable, with an intact outer retina, RPE and choroid foveally until late-stage progression results in central atrophy as well.

Female carrier signs can vary from no changes to mild peripheral RPE changes/clumping, with or without atrophy. Other associated findings include posterior subcapsular cataracts and, rarely, choroidal neovascular membrane. Often, the pathophysiology and ocular signs directly correlate to visual symptoms.

In advanced CHM the fundus has a classic appearance of yellow/white sclera and large choroidal vessels with or without foveal sparing, depending on the degree of progression.

However, early in the disease, imaging can help in diagnosis and management (*Table 1*).

Prevalence

Due to the x-linked inheritance, patients are typically male. Females are rarely affected, but are often carriers. Initial symptoms involve nyctalopia (correlated to peripheral pigment clumping) beginning in the first decade of life, that progresses to peripheral vision loss (correlating to peripheral atrophy) throughout the second and third decades of life. Central vision is preserved until the fifth to seventh decades of life, when color vision, visual acuity or both, can rapidly deteriorate (correlating to foveal atrophy). A large variation in symptoms and disease progression exists, even between family members affected by the condition. Female carriers will be largely asymptomatic, or have mild changes to their night vision and peripheral vision correlating to peripheral clumping and atrophy.[1]

Follow Up

Genetic testing has become the standard for confirming a CHM diagnosis. As CHM has just a single gene mutation, clinicians hope to one day see development of a successful gene therapy treatment. Adeno-associated virus subtype 2 is used in ophthalmic gene therapy research, in large part due to its affinity for photoreceptors and RPE.

Our patient was subsequently enrolled in one of the current clinical trials involving gene therapy via subfoveal injection of an adeno-associated virus subtype 2 for chorioderemia. He is being followed closely to determine whether he experiences any improvement in his visual function. ■

Table 1. Diagnostic Findings for CHM Staging

	Early	Advanced
OCT	Slight central retinal and choroidal thickening Parafoveal outer retinal atrophy Cystoid macular edema	Central outer retinal atrophy Choroidal thickening Cystoid macular edema Outer retinal tubulations
ERG*	Reduced scotopic followed by reduced photopic	Extinguished
FAF**	Patchy hypo-AF with scalloped, distinct edge-sparing macula	Extensive hypo-AF involving macula and periphery

*Electroretinogram for carriers is normal, except in some symptomatic carriers, which may show reduced full-field ERG 30-Hz flicker response in 15% of symptomatic carriers.[4]

**Fundus autofluorescence is extremely useful in early diagnosis and predicting disease progression. Areas of hypo-autofluorescence may precede outer retinal atrophy, thus there may be minimal to no signs on fundus exam or photography. Carriers can show speckled Hyper-Autofluorescence resulting from mild RPE degeneration, thus lipofuscin accumulation.[4]

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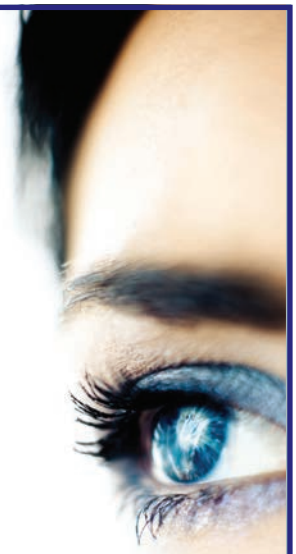

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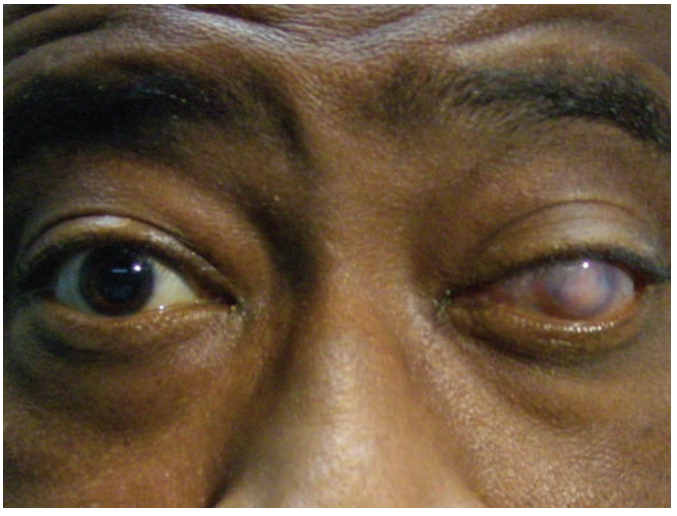
The Incredible Shrinking Eye

By Andrew S. Gurwood, OD

History

A 67-year-old black female was brought to the office because her family felt her left eye was "shrinking." The patient explained that the eye had been injured a number of years ago and that she also had a history of infection in the cornea, for which she had been hospitalized. Since the injury, the eye had lost its vision and only became painful after the corneal infection. She explained she used drops for a while but had stopped them herself when they became too expensive to afford and the pain stopped. Her systemic history was remarkable for hypertension, diabetes and dyslipidemia, which were all controlled medically. She denied allergies of any kind.



This 67-year-old woman is blind in her left eye, but her family was concerned that the eye itself was shrinking. Can this external examination help explain her condition?

Diagnostic Data

Her best-corrected entering visual acuities were 20/40 OD at distance, 20/25 OD at near. She had no light perception in her left eye. The external examination of her eye is demonstrated in the photograph. We did detect an afferent pupil defect in the left eye, seen using the indirect technique (the right eye dilated when the light was placed in front of the left eye). Her refraction was stable and measured +1.00/+3.00 OD. Confrontation visual fields for her right eye were normal.

The biomicroscopic examination of the anterior segment of her right eye was also normal and showed no evidence of iris neovascularization. The left eye demonstrated dense corneal opacity, elements of band keratopathy, a large central corneal depression with evidence of tissue thinning, a flat anterior chamber and evidence of old hyphema. Goldmann applanation tonometry measured 15mm Hg OD and 4mm Hg OS. The dilated examination of the patient's right eye was normal and demonstrated no evidence of peripheral pathologies. However, it was not possible to get a view of the left eye.

Your Diagnosis

Does the case presented require any additional tests, history or information? What steps would you take to manage this patient? Based on the information provided, what would be your diagnosis? What is the patient's most likely prognosis? To find out, please visit us online at www.reviewofoptometry.com. ■

Retina Quiz Answers *(from page 83):* 1) b; 2) b; 3) c; 4) a.

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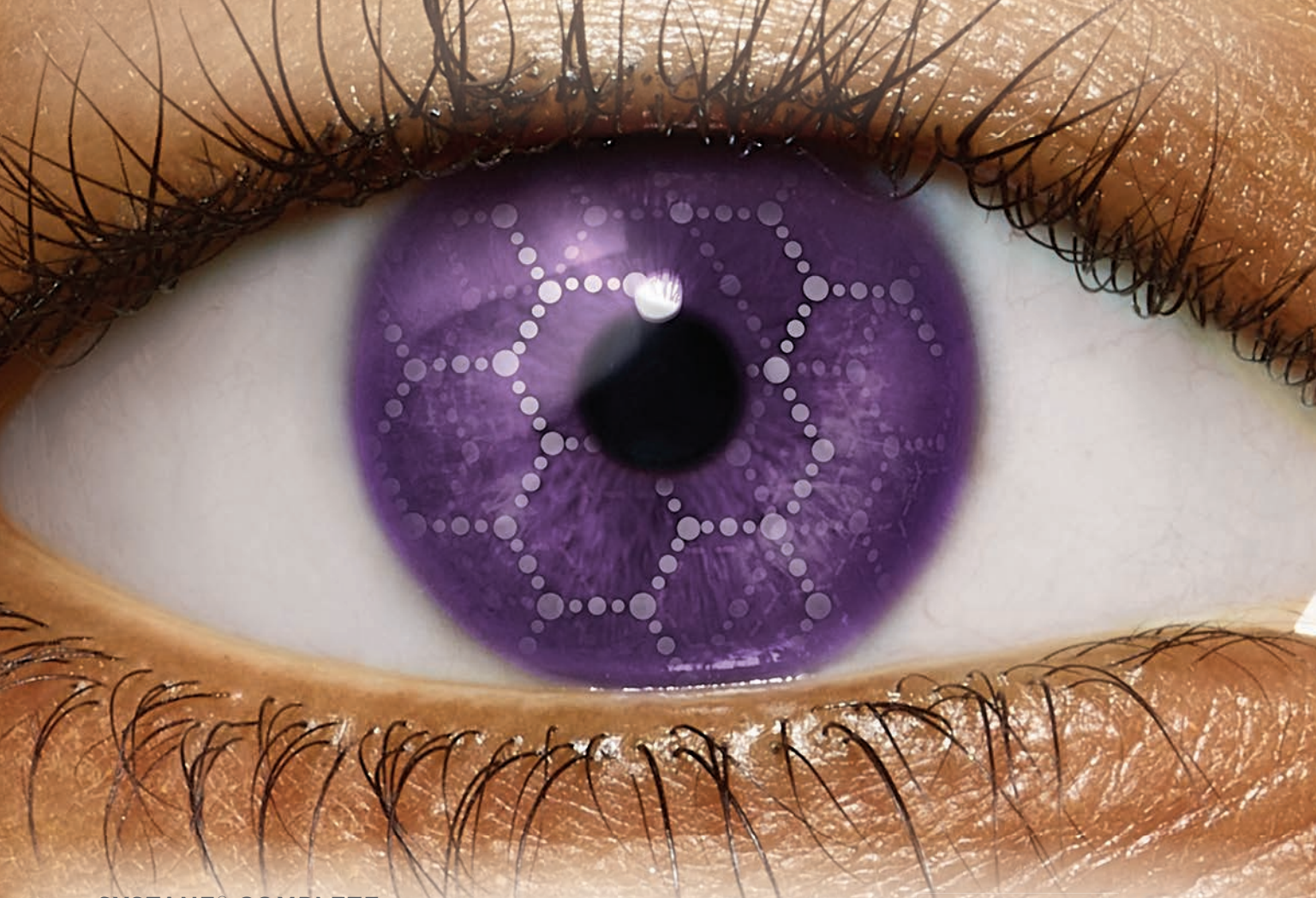

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1. Ketelson H, Rangarajan R. Pre-clinical evaluation of a novel phospholipid nanoemulsion based lubricant eye drops. Poster presented at: The Annual Meeting of the Association for Research in Vision and Ophthalmology (ARVO); May 7–11, 2017; Baltimore, Maryland, USA. **2.** Data on file. Alcon; 2017. **3.** Fernandez KB, Epstein SP, Raynor GS, et al. Modulation of HLA-DR in dry eye patients following 30 days of treatment with a lubricant eyedrop solution. *Clin Ophthalmol*. 2015;9:1137-1145. **4.** Davitt WF, Bloomenstein M, Christensen M, Martin AE. Efficacy in patients with dry eye after treatment with a new lubricant eye drop formulation. *J Ocul Pharmacol Ther*. 2010;26(4):347-353. **5.** Korb D, Blackie C, Meadows D, Christensen M, Tudor M. Evaluation of extended tear stability by two emulsion based artificial tears. Poster presented at: 6th International Conference of the Tear Film and Ocular Surface: Basic Science and Clinical Relevance; September 22-25, 2010; Florence, Italy. **6.** Lane S, Paugh J, Webb JR, Christensen MT. An evaluation of the in vivo retention time of a novel artificial tear as compared to a placebo control. Poster presented at: The Annual Meeting of the Association for Research in Vision and Ophthalmology (ARVO); May 3-7, 2009; Fort Lauderdale, FL. **7.** Craig JP, Nichols KK, Akpek EK, et al. TFOS DEWS II definition and classification report. *Ocul Surf.* 2017;15:276-283. **8.** Torkildsen G. The effects of lubricant eye drops on visual function as measured by the Inter-blink interval Visual Acuity Decay test. *Clin Ophthalmol*. 2009;3:501-506.

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