In optometry, it’s always debate season. A look at some hot-button issues that challenge conventional wisdom. p. 66

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Researchers have identified the first cellular model of exfoliation glaucoma, which could lead to improved treatment. Investigators grew cells using tissue samples from trabeculectomy procedures and compared them with other cells without exfoliation glaucoma. The cells were much larger than normal and contained an abundance of disorganized vesicles—leading to the discovery of a defect in the autophagy system.


New research on the effectiveness of Humira (adalimumab, AbbVie) for noninfectious uveitis suggests it is an effective, nonsteroid alternative for eye inflammation. The study included 217 adults with active, noninfectious intermediate or posterior uveitis, or panuveitis. The researchers found that median time to treatment failure was 24 weeks in the Humira group and 13 weeks in a placebo group. Treatment failure was based on the assessment of new inflammatory lesions, best-corrected visual acuity, anterior chamber cell grade and vitreous haze grade.


Johnson & Johnson recently announced an agreement to acquire Abbott Medical Optics for $4.325 billion, which includes ophthalmic products in cataract surgery, laser refractive surgery and consumer eye health. The acquisition will allow Johnson & Johnson to enter the cataract surgery market, the company said in a release. The transaction is expected to close in early 2017.

Topical drops have always been a blessing and a curse for eye care providers. While they provide some form of treatment for patients, they come with a host of problems as well.

“The current use of topical drops for the treatment of ocular disease is certainly an issue,” says Jill Autry, OD, a partner at the Eye Center of Texas ophthalmology center. “There are concerns regarding compliance, convenience, concentration and toxicity—not to mention the poor pharmacokinetic profiles.”

Researchers have developed a possible solution to some of these age-old problems. Engineers at McMaster University in Ontario, Canada, created microscopic packets designed to sit in the base of the tear film and dissolve gradually—causing a slow release of medication. The researchers believe the new drops could make it possible for patients with conditions such as dry eye and glaucoma to receive the same amount of therapeutic effect from using drops once a week instead of daily.

“Using micelle polymers (miniature dissolvable drug packets), scientists are able to increase contact time and decrease drug concentration while still being easy on the ocular surface,” says Dr. Autry. “This could potentially enable medications to be delivered less frequently and with less systemic and ocular side effects than we have ever known.”

The team’s mucoadhesive drug delivery system, recently described in the journal Biomacromolecules, is comprised of phenylboronic-acid-based polymeric micelles that show low in vitro cytotoxicity against human corneal epithelial cells and undetectable acute in vivo ocular irritation in rats.

“My concerns are how this technology would be patented,” Dr. Autry says. “Would all companies have access to this delivery system for medications or would it be proprietary and only used in the newest, branded products? I am also concerned about how the delivery system would be affected by ocular conditions such as epiphora, dry eye, corneal scarring, artificial tear use, other drop use, etc., which can alter how the micelles are activated or retained.”

The researchers are in the final stages of investigating the safety and effectiveness of the new technology, hopefully answering many of these concerns. They aim to have it on the market in the near future.

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E ducation held the spotlight at the recent Vision Expo West (VEW), held from Sept. 14-17 in Las Vegas. This year’s annual meeting boasted the second all-time highest CE attendance in Expo’s history, with preliminary numbers coming in at 4,542, according to International Vision Expo. Total attendance figures for the entire conference were not available at press time, but will be released in a few weeks, International Vision Expo said.

Top-notch Learning
In total, VEW 2016 offered more than 320 hours of education, with clinical sessions covering everything from ocular disease diagnosis and treatment, contact lenses and therapeutics to the very latest in imaging.

“Our comprehensive and inclusive education programming, combined with our extensive exhibits, came together to offer a high value, impactful experience in Las Vegas,” said Ben Gaddie, OD, co-chair of the Vision Expo conference advisory board. “Vision Expo provides a unique and innovative approach to all aspects of clinical care education including refractive care, contact lenses, anterior segment disease, specialty dry eye management, glaucoma and retinal disease. It’s why more eye care professionals choose International Vision Expo for their education than any other conference.”

New Perspectives
VEW served up several new courses this year, including a scleral lens track with five hours of CE credit. Scleral lenses provide new opportunities and challenges for practices, and this track featured presentations by Melissa Barnett, OD, Stephanie Woo, OD, and Barry Eiden, OD, on subjects such as fitting, lens design, patient selection, specialized testing and problem solving for both full and mini-sclerals.

Another new track, “CAB Chairman Top Picks,” provided up to 21 hours of CE credit. Some of the highlights from this track included:

- The Best and the Worst Ocular Emergencies and Urgencies, presented by Vincent Young, MD, and Marc Myers, OD
- Neuro For the Rest of Us, by William Marcolini, OD
- What Do You Do If... Diagnosis and Treatment of Anterior Segment Disease You Meet Every Day! by Dr. Eiden and Andrew Morgenstern, OD
- Cloak and Dagger Retinal Clues to Systemic Disease, presented by Steven Ferrucci, OD

During the new “Lightening Rounds” five key opinion leaders offered their different perspectives in a speed-dating type of setting. Topics included glaucoma and anterior segment diseases.

“We recognize that doctors and staff are taking time away from the office to attend Vision Expo, which is why we offer a comprehensive didactic curriculum that is rich in ocular disease and contact lens courses as well as elevated business education sessions catered to bringing back actionable solutions that can be immediately implemented,” said Mark Dunbar, OD, co-chair of the Vision Expo conference advisory board. “Our continuing education is increasingly focused on the practical elements of providing vision care while running a successful business, or those wishing to open a practice.”

In the Hall
In addition to CE, the conference had 183,000 square feet of exhibit space in the Medical & Scientific Pavilion. The hall showcased the latest ophthalmic technologies and innovations, including 178 companies—24 of which were first-time exhibitors this year.

Mark your calendar for more CE in Sin City next year, as VEW will be back in Las Vegas from Sept. 13-16, 2017. For more information about VEW 2017, go to west.visionexpo.com.

The Global Contact Lens Forum—and this “State of the Contact Lens Industry in 2016” course in particular—was a hit at this year’s Vision Expo West.
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Internal Astigmatism Exposed

Internal astigmatism does not compensate for changes in the structure of the eye, according to new research. Investigators looked at 14 years of measurements and refractive error evaluations on 367 myopic patients and compared the data with one-time measurements of 204 non-myopic individuals.

Optometrists typically consider internal astigmatism a constant, yet the researchers found evidence suggesting otherwise. Internal astigmatism was greater in non-myopes who proved better able to compensate for corneal astigmatism, and internal astigmatism remained stable over time, not changing as the shape of the eye changed.

“This work finds that internal astigmatism varies by refractive error, ethnicity and the magnitude of corneal astigmatism,” says Ruth E. Manny, OD, PhD, of University of Houston College of Optometry, and lead author of the study. “Therefore, internal astigmatism should not be thought of as a constant.”

“Predicting patients who have high internal astigmatism could be important when considering sending a patient for refractive surgery or cataract surgery since neglecting this component of the refraction could produce unwanted outcomes,” Dr. Manny says.

“Using new technologies may provide a better understanding of the sources of internal astigmatism,” Dr. Manny says. “Direct measurement of internal astigmatism may also allow us to understand what is responsible for the differences in internal astigmatism by refractive error, ethnicity and corneal astigmatism.”


Pediatric Eye Care Linked to Affluence, Study Shows

Children from less affluent homes are considerably less likely to obtain eye care services, according to a recent study published in Health Affairs. This results in approximately 12,800 missed strabismus diagnoses and 5,400 missed amblyopia diagnoses, researchers say.

The 10-year study divided subjects into groups based on household income. The findings show that children from the highest earning households (more than $500,000) had 19% more visits to eyecare professionals (ECPs) than those from the middle-income group (between $150,000 and $250,000). Children from the lowest earning households (less than $25,000), however, had 16% fewer visits to ECPs than children from the middle-income group.

“Most children aren’t asked or required to get a full dilated eye exam until they fail a screening either with their school nurse or pediatrician,” says Luis Trujillo, OD, who specializes in pediatric and binocular vision at The Eye Institute in Philadelphia. “Children of underserved populations may not have access to a pediatrician (continued on pg. 10)
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(continued from pg. 8)

(dpending on insurance status) and, at least here in the Philadelphia area, I have found that nurses are asked to cover more than one school within the district. This can delay when a child gets screened, to say nothing of when they will actually get an exam.”

“More attention should be directed to overcoming economic barriers that keep children from obtaining necessary eye care services,” the report concludes.1

There are ways optometrists can help level the playing field, according to Dr. Trujillo, who suggests contributing to programs, such as Infant-See, which allows patients within a certain age range access to an exam at any participating provider’s office. Another option is to reach out to your local school district. “As an assistant professor and practicing pediatric optometrist at Salus University, I have been given the opportunity and privilege over the last three years to go into the schools in the Norristown school district and give full eye exams to students who have failed their nurse’s screening. These students do not have health insurance or access to eye care, period. Along with the exam, these students get two pairs of glasses, and the entire program is funded through donations from the community,” says Dr. Trujillo.

Government funded programs, such as Head Start, also allow ODs to provide screenings to preschool-aged children.

Dr. Trujillo also recommends doctors lobby lawmakers to push for mandatory eye exams for all children.

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The oft overlooked Demodex diagnosis is elemental to setting patients on the path to relief, if you know what to look for.

By Victoria Roan, OD

78 Spotlight on Demodex: Eliminating the Mite-y Menace

The oft overlooked Demodex diagnosis is elemental to setting patients on the path to relief, if you know what to look for. By Victoria Roan, OD
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ANDREW S. GURWOOD, OD
Classic beta blocker adjunctive therapy for the right patient at the right time

The concomitant use of two topical beta-adrenergic blocking agents is not recommended.

Indications and Usage
ISTALOL® (timolol maleate ophthalmic solution) is a non-selective beta-adrenergic receptor blocking agent indicated in the treatment of elevated intraocular pressure in patients with ocular hypertension or open-angle glaucoma.

Preservative-free TIMOPTIC® (timolol maleate ophthalmic solution) in OCUDOSE® (dispenser) is indicated in the treatment of elevated intraocular pressure in patients with ocular hypertension or open-angle glaucoma. It may be used when a patient is sensitive to the preservative in TIMOPTIC (timolol maleate ophthalmic solution), benzalkonium chloride, or when use of a preservative-free topical medication is advisable.

Important Safety Information for Istalol and Timoptic in Ocudose
• Both ISTALOL® (timolol maleate ophthalmic solution) and TIMOPTIC® (timolol maleate ophthalmic solution) in OCUDOSE® (dispenser) are contraindicated in patients with: bronchial asthma; a history of bronchial asthma; severe chronic obstructive pulmonary disease; sinus bradycardia; second or third degree atrioventricular block; overt cardiac failure; cardiogenic shock; hypersensitivity to any component of the product.

• The same adverse reactions found with systemic administration of beta-adrenergic blocking agents may occur with topical administration. Severe respiratory reactions and cardiac reaction, including death due to bronchospasms in patients with asthma, and rarely death in association with cardiac failure, have been reported following systemic or ophthalmic administration of timolol maleate.

• Patients with a history of atopy or severe anaphylactic reactions to a variety of allergens may be unresponsive to the usual doses of epinephrine used to treat anaphylactic reactions.

• Timolol has been reported rarely to increase muscle weakness in some patients with myasthenia gravis or myasthenic symptoms.

• Beta-adrenergic blocking agents may mask signs and symptoms of acute hypoglycemia or certain clinical signs of hyperthyroidism. Patients subject to spontaneous hypoglycemia, or diabetic patients receiving either insulin or oral hypoglycemic agents, or patients suspected of developing thyrotoxicosis, should be managed carefully, with caution.

• In patients undergoing elective surgery, some authorities recommend gradual withdrawal of beta adrenergic receptor blocking agents because these agents impair the ability of the heart to respond to beta-adrenergically mediated reflex stimuli.

• The most frequently reported adverse reactions have been burning and stinging upon instillation. This was seen in 38% of patients treated with ISTALOL and in approximately one in eight patients treated with TIMOPTIC in OCUDOSE. Additional reactions reported with ISTALOL at a frequency of 4 to 10% include: blurred vision, cataract, conjunctival injection, headache, hypertension, infection, itching and decreased visual acuity.

Please see Brief Summary of Prescribing Information for ISTALOL and TIMOPTIC in OCUDOSE on the following pages.
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**INDICATIONS AND USAGE**

Preservative-free TIMOPTIC in OCUDOSE® is indicated in the treatment of elevated intraocular pressure in patients with open-angle glaucoma or ocular hypertension.

Preservative-free TIMOPTIC in OCUDOSE® may be used in patients when a preservative is contraindicated in the prescriber's judgment.

**CONTRAINDICATIONS**

Preservative-free TIMOPTIC in OCUDOSE® is contraindicated in patients with:

- Bronchial asthma
- A history of bronchial asthma
- Severe chronic obstructive pulmonary disease (see WARNINGS)
- A history of angina pectoris
- A history of congestive heart failure
- A history of thyrotoxicosis

- Patients with bronchial asthma, a history of bronchial asthma, severe chronic obstructive pulmonary disease, or a history of thyrotoxicosis should be advised not to take this product. (See CONTRAINDICATIONS).

**WARNINGS**

As with any topically applied ophthalmic drug, this drug is absorbed systemically. The clinical consequences of systemic administration of beta-adrenergic blocking agents may occur with topical administration. For example, severe respiratory reactions and cardiac reactions, including death due to bronchospasm and cardiac arrest, and death due to cardiac arrest alone, have been reported following topical administration in cardiac failure, have been reported following systemic or ophthalmic administration of timolol maleate (see CONTRAINDICATIONS).

Cardiac Failure:

The risk of death from cardiac failure is the essential risk of patients receiving beta-adrenergic receptor blocking agents. The potential for serious cardiac decompensation in patients with cardiac failure has been reported following systemic or ophthalmic administration of timolol maleate (see CONTRAINDICATIONS).

**ADVERSE REACTIONS**

**Local Reactions**:

Patients treated with TIMOPTIC should be observed for ocular irritation such as burning and stinging. Patients have reported occasional burning and stinging with TIMOPTIC (timolol maleate ophthalmic solution) resulting in systemic effects similar to those seen with systemic beta-adrenergic blocking agents such as dizziness, headache, nausea, shortness of breath, bradycardia, bradycardia, and cardiac arrest (see ADVERSE REACTIONS).

**OVERDOSAGE**

There have been reports of inadvertent overdosage with Ophthalmic Solution TIMOPTIC (timolol maleate ophthalmic solution) resulting in systemic effects similar to those seen with systemic beta-adrenergic blocking agents such as dizziness, headache, nausea, shortness of breath, bradycardia, bradycardia, and cardiac arrest (see ADVERSE REACTIONS). Overdosage may be treated with pilocarpine 1% eye drops.

**TIME OF ADMINISTRATION**

In patients with angle-closure glaucoma, the immediate post-dose rise in intraocular pressure is due to the response of the accommodating mechanism rather than a true ocular response. If IOP is not controlled with the initial dose of TIMOPTIC, the dosage may be increased to one drop of 0.5 percent after the patient’s response to the initial dose has been determined. If the intraocular pressure is not reduced after the second dose, the dosage should be increased to one drop of 0.5 percent every 12 hours. The dosage may be increased up to a maximum of two drops of 0.25 percent twice a day generally have not been shown to produce further reduction in IOP.

**Preparations**

Parenteral—Topical—Ophthalmic—Otic—Topical—Otic—Novartis Pharmaceuticals Corporation, a company of Novartis AG, has announced the development of a novel therapeutic approach for the treatment of glaucoma.

**Administration and Dosage**

For the ophthalmic drug administration. Because of the potential for serious adverse reactions from timolol in raising intraocular pressure, if the patient’s intraocular pressure is not reduced by a reduction in the dose of timolol, the drug should be discontinued and another treatment regimen employed. Dosages above the dose of 0.5 percent TIMOPTIC (timolol maleate ophthalmic solution) have not been shown to produce further reduction in IOP. If the intraocular pressure is not reduced by a reduction in the dose of timolol, the drug should be discontinued and another treatment regimen employed.

**Contraindications**

**Cardiac Failure:**

If signs or symptoms suggesting reduced cerebral blood flow are present, patients suspected of developing thyrotoxicosis should be managed carefully to avoid abrupt withdrawal of beta-adrenergic blocking agents that would exacerbate the clinical condition.

**Hypotension during anesthesia:**

Difficulty in restarting and maintaining the heartbeat has been reported following systemic administration of timolol maleate (see CONTRAINDICATIONS).

**Nursing Mothers:**

The potential for serious cardiac decompensation in patients with cardiac failure has been reported following systemic or ophthalmic administration of timolol maleate (see CONTRAINDICATIONS).

**Cardiac Insufficiency:**

If signs or symptoms suggesting reduced cerebral blood flow are present, patients suspected of developing thyrotoxicosis should be managed carefully to avoid abrupt withdrawal of beta-adrenergic blocking agents that would exacerbate the clinical condition.

**Hypotension during anesthesia:**

Difficulty in restarting and maintaining the heartbeat has been reported following systemic administration of timolol maleate (see CONTRAINDICATIONS).

**Nursing Mothers:**

The potential for serious cardiac decompensation in patients with cardiac failure has been reported following systemic or ophthalmic administration of timolol maleate (see CONTRAINDICATIONS).

**Cardiac Insufficiency:**

If signs or symptoms suggesting reduced cerebral blood flow are present, patients suspected of developing thyrotoxicosis should be managed carefully to avoid abrupt withdrawal of beta-adrenergic blocking agents that would exacerbate the clinical condition. In a lifetime oral study in mice, there were statistically significant increases in the incidence of benign and malignant pulmonary tumors, benign prostate tumors and malignant adenocarcinomas in female mice of 50 mg/kg (approximately 71,000 times the systemic exposure following the maximum recommended human intraocular dose). Similar differences were not observed in rats or mice doses equivalent to approximately 14,000 times the maximum recommended human intraocular dose.

**In a one year oral study in monkeys, male, significant increases in the incidence of bronchial adenomas and malignant pulmonary tumors, benign prostate tumors and malignant adenocarcinomas in female monkeys of 50 mg/kg (approximately 71,000 times the systemic exposure following the maximum recommended human intraocular dose), but not at 5 or 50 mg/kg (approximately 7,000 or 700 times, respectively, the systemic exposure following the maximum recommended human intraocular dose); in a subacute study in dogs in which rats were not given the timolol maleate and the dogs, a statistically significant increase in the incidence of pulmonary tumors was observed in both species. The increased occurrence of malignant adenocarcinomas was associated with elevations in serum prolactin which occurred in female mice administered timolol maleate at 50 mg/kg, but not at 5 or 50 mg/kg (approximately 7,000 or 700 times, respectively, the systemic exposure following the maximum recommended human intraocular dose). In a subsequent study in female mice in which post-mortem examinations were limited to the skin and the lungs, a statistically significant increase in the incidence of pulmonary tumors was again observed and among females.

**Beta-adrenergic blockade has been reported to potentiate muscle weakness;**

Beta-adrenergic blocking agents may mask certain clinical signs (e.g., bradycardia and hypotension); however, patients administered beta-blockers will not necessarily be completely protected from heart attack, stroke, or other serious complications of myocardial infarction.
Istalol® (timolol maleate ophthalmic solution) 0.5% 0.9% Instant U.S. Approval: 1979

STORAGE

INDICATIONS AND USAGE
Istalol (timolol maleate ophthalmic solution) 0.5% is a non-selective beta-adrenergic blocker indicated in the treatment of elevated intraocular pressure (IOP) in chronic open-angle glaucoma or ocular hypertension or open-angle glaucoma.

CONTRAINDICATIONS
4.1 Asthma, COPD: Istalol is contraindicated in patients with bronchial asthma; a history of bronchial asthma; severe chronic obstructive pulmonary disease (see WARNINGS AND PRECAUTIONS, 5.1, 5.3); bronchial asthma or a history of bronchial asthma in which Istalol is contraindicated (see CONTRAINDICATIONS, 4.2). This Brief Summary does not include all the information needed to complete a course of topical therapy.

WARNINGS AND PRECAUTIONS
4.3 Hypersensitivity Reactions: Hypersensitivity reactions, including anaphylaxis, have been reported following systemic administration of beta-adrenergic blocking agents or following administration of the drug by the intravenous or intramuscular route. Hypersensitivity reactions include angioedema, anaphylactic reaction, bronchospasm, hypotension, and shock. Hypersensitivity reactions to beta-adrenergic blocking agents may also occur with topical ophthalmic administration. A history of atopy or a history of severe anaphylactic reactions to a variety of allergens, including foods and a variety of non-beta adrenergic blocking agents, such as nonsteroidal anti-inflammatory drugs or bronchodilators, makes patients particularly susceptible to this reaction. In clinical trials, adverse reactions noted were in association with cardiac failure, have been reported following systemic or intravenous administration. For example, severe respiratory reactions and cardiac reactions including death due to bronchospasm in patients with asthma, and rarely death in association with cardiac failure, have been reported following systemic or ophthalmic administration of timolol maleate (see CONTRAINDICATIONS, 4.1). The potential for local and systemic reactions found in association with systemic administration of beta-adrenergic blocking agents may occur with topical administration. For example, severe respiratory reactions and cardiac reactions including death have been reported following systemic administration of timolol maleate in patients with asthma, and rarely death in association with cardiac failure, have been reported following systemic or ophthalmic administration of timolol maleate (see CONTRAINDICATIONS, 4.1).

5.1 Potentiation of Respiratory Reactions Including Asthma: Istalol may potentiate respiratory reactions including asthma. In patients with pre-existing bronchospastic disease, respiratory failure, nasal congestion, cough and upper respiratory infections, Escalation of exacerbations of respiratory disease in patients with beta-adrenergic blocking agents (see WARNINGS AND PRECAUTIONS, 5.3). Bronchial asthma or a history of bronchial asthma in which Istalol is contraindicated (see CONTRAINDICATIONS, 4.2), should in general, not receive beta-blocking agents, including timolol.

5.2 Pulmonary Edema: Patients with chronic obstructive pulmonary disease (e.g., chronic bronchitis, emphysema) of mild or moderate severity, bronchospastic disease, or a history of bronchospastic disease (other than those with a history of bronchial asthma in which Istalol is contraindicated (see CONTRAINDICATIONS, 4.2)) should, in general, not receive beta-blocking agents, including timolol.

5.4 Hypotension: Severe hypotension has been reported more frequently in patients with cerebrovascular insufficiency. If signs or symptoms of cerebrovascular insufficiency develop following initiation of therapy with timolol, alternative therapy should be considered. In patients with cerebrovascular insufficiency, close observation of the patient is recommended when a beta blocker is administered to patients receiving CYP2D6 inhibitors because of possible atrioventricular conduction disturbances, left ventricular failure, and hypotension. In patients with impaired cardiac function, co-administration should be avoided.

5.5 Potentiation of Muscle Weakness: Beta-adrenergic blockade has been associated with exacerbation of myasthenia gravis patients. Beta-adrenergic blocking agents such as timolol may exacerbate symptoms of myasthenia gravis, paresthesia, somnolence, insomnias, nightmares, behavioral changes and psychiatric disturbances including depression, confusion, hallucinations, anxiety, delirium, and nervousness and memory loss; skin Allopurinol and panthothenate or pantothenic acid; hypopituitarism; Signs and symptoms of systemic allergic reactions, including angioedema, urticaria, laryngeal edema, bronchospasm, shock, anaphylactic reaction, and anaphylactoid reaction, have been reported in patients with pre-existing bronchospastic disease, respiratory failure, nasal congestion, cough and upper respiratory infections, Escalation of exacerbations of respiratory disease in patients with beta-adrenergic blocking agents. In a study in patients with pre-existing bronchospastic disease, Escalation of exacerbations of respiratory disease in patients with beta-adrenergic blocking agents (see WARNINGS AND PRECAUTIONS, 5.3). Bronchial asthma or a history of bronchial asthma in which Istalol is contraindicated (see CONTRAINDICATIONS, 4.2).

5.6 Preexisting Cardiac Disease: Patients with pre-existing cardiac disease (e.g., previously documented silent myocardial infarction, coronary artery disease, angina pectoris, ventricular arrhythmias, cardiogenic shock, heart failure, or a disruption of the ocular epithelial surface (see PRESERVATION IN MAY, 2014)) should, in general, not receive beta-blocking agents, including timolol.

5.7 Atenolol: Atenolol has been shown to decrease the plasma concentration of oral theophylline and oral theophylline and oral theophylline to decrease the plasma concentration of oral theophylline and oral theophylline in patients treated with atenolol. When intravenous theophylline is used, patient should not be used alone in the treatment of angle-closure glaucoma.

5.10 Administration of Beta-blocking Agents During Angiographic Procedures: Beta-blocking agents have been used with caution in patients with cerebrovascular insufficiency. If signs or symptoms suggesting reduced cerebral blood flow develop following initiation of therapy with timolol, alternative therapy should be considered.
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1 Harmful Blue Light falls between 415-455nm on the light spectrum and is suspected to be most toxic to retinal cells. See Arnaul t E, Barrau C, Nanteau C, Gondouin P, Bigot K, et al. (2013). Phototoxic Action Spectrum on a Retinal Pigment Epithelium Model of Age-Related Macular Degeneration Exposed to Sunlight Normalized Conditions. PLoS ONE 8(8): e71398. doi:10.1371/journal.pone.0071398. Harmful Blue Light is the blue-violet wavelengths believed most toxic to retinal cells, which lie between 415-455nm on the light spectrum. 2Combining a Smart Blue Filter™ lens with Crizal Prevencia No-glare coating reduces exposure to Harmful Blue Light by at least 30% and provides UV protection.

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Letters to the Editor

High Praise
I read the 125th anniversary issue (July 2016) on planes to and from Nova Scotia. I think your staff did a superb job in the coverage. They really did.

The profession of optometry has made miraculous strides in the 70 or so years since I started practicing. The changes in goals and in scope of practice, in my opinion, have been greater than any other healthcare profession. I do hope that the younger generation does not forget the early years.

I have been a friend of Frank Fontana for years and years, and both Drs. Gurwood (the father and the son) are on my list of colleagues. It was a pleasure to see them featured in the issue.

Thank you for taking the time, and making the effort, to construct the theme of this issue. Congratulations to you and your colleagues for a job well done.

—Irving Bennett, OD
Sarasota, FL

Out With the New, In With the Old
"Speaking Frankly: A Conversation with Frank D. Fontana, OD" (July 2016) was priceless! For us “old geezers,” who’ve been practicing for more than 50 years, hearing Dr. Fontana relate to the old days is something we greatly appreciate.

—Stan Pugh, OD
Tacoma, WA

Son of A. Fitch
Congratulations on 125 years of your eminent magazine. While reading about optometry’s evolution, I saw many prominent optometric names mentioned, including that of Albert Fitch. I attended the Pennsylvania State College of Optometry (1958-1962). This unique institution required a total of six years of education (about a third of the class had BA/BS degrees). All the other institutions required five years. PSCO included full courses in organic chemistry, biochemistry, neuroanatomy, human anatomy (dissected cadaver), mammalian anatomy (dissected cat), general pathology, ocular histopathology, ocular pathology and public health. In addition, all courses were appropriately instructed by individuals with PhD and MD degrees. Think of it! Sixty years later and today’s colleges have curricula patterned from PSCO’s model. That is why, in my humble opinion, Dr. Fitch’s name, for his many important contributions, should be more prominently recognized.

—Nathan Solat, OD
Staten Island, NY

Physician, Eat Thy Vitamins
I read with great interest the nutrition and roundtable discussion in the February issue (“Transforming Eye Health Through Proven Ocular Nutrition Strategies,” sponsored by MacuHealth). The discussion regarding the benefits of nutritional supplementation, and the role of optometrists, was very insightful. It was especially enlightening to read about the benefits of improved nutrition, even for apparently young and healthy people, regarding the increase in macular pigment optical density, correlating to improved mental acuity.

I have taken a multivitamin and 500mg of time-
In Defense of Retinal Scanning

Response to “The Dilation Dilemma,” June 2016:

When debating the value of retinal screening vs. dilated fundus exams (DFE), don’t underestimate the impact retinal scans have on patient education. Figure 1 demonstrates what the results of a DFE looks like to a patient with diabetic retinopathy. Figures 2 and 3 demonstrate what the results of an Optomap (Optos) scan, without DFE, look like for the same patient. The images speak for themselves.

I routinely dilated eyes for 25 years before I obtained an Optos retinal scanner for my practice in 2006. What I found that I—as well as other respected practices in my area—had been missing most by relying on DFE was mid-peripheral lesions. In addition, some prior DFE records from my own and other practices had the lesions in different locations than they actually were. These “clerical” errors of position simply do not happen with Optos.

Although I still have my indirect ophthalmoscope and lenses, Optos has been the one technology in 36 years of practice that has most dramatically improved my ability to diagnose and educate patients. It has allowed for immediate consultations with retinal specialists, even on Saturday afternoons. It provides the opportunity for that all-important “second look” by yourself or a colleague.

In the minute it takes to explain an Optomap image to a patient, I see conditions that I may have missed during a DFE.

I began practicing when it was illegal to dilate. I remember opposing camps in optometry regarding retinoscopy and autorefrac- tion. I remember seeing my first OCT.

Standards of care in health care are always evolving, and each step in this technological evolution faces its critics. It is not mercenary of us to embrace these technologies—it is visionary and vital for the future of our profession. Optometry needs these new methods of examining eyes. We need engineers to create these technologies and practitioners that have the courage to adopt them.

The insurance coverage a patient does or does not have should not determine what we feel is the most comprehensive level of care. If insurance companies only covered a routine eye exam every three years, should that be the new standard of care?

Dilation and Optos each have their advantages and disadvantages. Please don’t refer to Optos as “a crutch” while our current standard of care provides no permanent record, of a fleeting image of a small part of the retina that we only see upside down and backwards.

— Robert Conway, OD
Rochester, NY
Technical Difficulties

Imagine this scenario: You bring your car in for routine maintenance. The basic services are covered under your warranty, so you don’t expect to lay out any cash unless there’s a repair needed. The car’s in good shape, so you figure you’ll be out of there without much hassle. But before you’ve even gotten your complimentary cup of coffee, the receptionist hands you some intimidating forms. Apparently, there are several optional tests the mechanic can do if you’re willing to pay. One detects engine problems sooner. Another could make your steering more responsive. A third will get you out the door today faster—and might also reveal wear and tear better. But maybe these tests do nothing. It’s confusing. And the decision is on your shoulders.

You study the forms quizzically, trying to parse the technical jargon, and worry about making the wrong call. It feels like a lose-lose situation: either you waste money on unnecessary tests or risk missing out on valuable information.

Sound familiar? This is exactly what many patients feel when presented with the array of options for elective diagnostic screening tests.

This didn’t really dawn on me until a few weeks ago when my wife needed an eye exam, her first in several years. After checking in with the receptionist, she was asked to make three decisions about her own care—and wallet—while still making decisions about her own care—and wallet—while still waiting. Like many practices, this one offered wavefront scanning, visual field screening and ultra-widefield retinal imaging. Other offices might offer OCT and macular pigment optical density too, either bundled into a “wellness package” or offered a la carte.

It’s a lot of info to absorb, especially before the patient has spoken to anyone with clinical expertise.

I know this is common practice. That doesn’t make it good practice, however. Shouldn’t the patient be able to discuss these procedures with the doctor or a well-informed tech, to make a better decision or at least put their mind at ease? My wife was busy texting me for advice instead of having a conversation with a healthcare professional.

There’s nothing wrong with offering elective services or out-of-pocket charges. Many patients are happy to pay top dollar for high-fashion eyewear, multifocal contact lenses, “spa-like” dry eye treatments and other lifestyle enhancements. But when an optional fee concerns diagnostic data, it’s harder for patients to gauge the value on their own, especially if the handouts imply a possible missed diagnosis for those who decline the test. They need personal attention, not form letters.

Give patients the courtesy of a conversation. Look at their history. Talk about their goals. Make recommendations. Let them know they have an advocate at the practice; otherwise, they may consider it a stressful, “hard-sell” experience instead of the chance to take advantage of some cutting-edge technology to learn more about their eyes.

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Optometrists are thinkers. We sit around chewing on the least important issues as if our entire world will most certainly collapse if we choose the wrong side dish at Taco Bell. So, imagine the turmoil the poor OD faces when a sales rep wants him to order a lens bank. What to do?!

I have developed a few random thoughts that might help you gain the confidence to make weighty decisions such as this:

1. Tackle every computer decision with the clear understanding that you will eventually drop the monitor onto your arthritic big toe. Choose accordingly.
2. When you think, “that was the stupidest idea I’ve ever had,” know that you’ll top that someday, I promise.
3. Studies show that, if you decide your answer is no you will be right 87.45% of the time.
4. No decision you make will make sense to your spouse.
5. If “just say yes!” pops into your head, it’s the Holy Spirit talking—unless it’s a weight loss infomercial at 3am.
6. Never make your final decision based on how much it costs you because your patients are paying for it! They will not be happy unless it benefits them.
7. Try new lenses on every candidate as your first choice. Only keep the fitting set if eight of every 10 patients love it.
8. If you are deciding what multifocal contact lens to choose for a patient, you are already in over your head, my friend.
9. A written policy gives you confidence in tricky situations. If a patient’s phone rings, my policy is to leave the room for at least an hour for their privacy.
10. Once you decide on a new phone system, unload and remove all firearms from your house to prevent some other kind of “cide.”
11. Never decide that patients are snotty-heads based on their front desk interactions. They are probably just mad at their spouse, running late for a tennis lesson—or maybe they are, in fact, snotty-heads.
12. Each day wake up and decide to be at peace. Smile and laugh, enjoy each moment and, if all else fails, there is always tequila.
13. One theory is that the more you learn about your profession the better decisions you will make. Or you can stay stupid and be just as successful.
14. If in doubt, trust #2 more than #1.
15. There’s a good reason that restaurant’s parking lot is empty.
16. When buying a new car, test drive it to a lumberyard and compare it with all the other vehicles. Still like it? Buy it.
17. Always refer to the wisdom of the punk band, The Clash. If I can’t decide, Should I Stay or Should I Go, my fallback position is Rock the Casbah. It works for me.
18. Nancy Reagan was correct: just say no.
19. When the decision has the potential to be a life game changer, I look at all the facts, carefully research the alternatives, lay out the myriad sequelae—and then I do what my wife says.
20. Drink tons of water. The quiet times my kidneys have afforded me have contributed so much to my decision making process.

You are now prepared to make outstanding decisions—or at least have something to do while you procrastinate: Google The Clash. That will give you a break in the action as you Rock the Casbah.
Announcing BromSite, the first product from Sun Ophthalmics.
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Preventing a Total Melt Down
Keratolysis is a significant complication that demands immediate attention. Here’s a primer on monitoring and treating it. By Justin Schweitzer, OD, and Richard Mangan, OD

The vast majority of refractive surgery cases go according to plan, but when patients do experience complications, optometrists are there to play an integral role in comanagement. Corneal melting, or keratolysis, is one such complication that may occur after LASIK. It can lead to scarring, irregular astigmatism, photophobia and decreased vision. Improvements in surgical techniques and technology have reduced the incidence of postoperative complications after LASIK, but corneal melt remains an emergent threat to a patient’s vision. Prompt recognition and aggressive treatment can prevent permanent visual loss. In this column, we review the pathophysiology and treatment of this potentially vision-threatening condition.

Pathophysiology
The incidence of a corneal melt following LASIK is difficult to quantify accurately, as reports in published literature are often small series or case reports discussing a single event. The melting process often starts at the rim of the flap and is commonly associated with a variety of conditions, such as epithelial ingrowth. The migration of epithelial cells under the LASIK flap increases following enhancement surgery, specifically following lifting of the flap. Patients with certain preexisting corneal conditions present a greater risk of developing ingrowth and are at a greater risk for corneal melting after LASIK flap creation (Figure 1). These conditions include epithelial basement membrane dystrophy, collagen vascular diseases and autoimmune diseases. Additionally, dry eye disease creates a poor healing environment of the cornea, which may potentially make corneal melt more likely. These conditions should be resolved before LASIK surgery is performed.

Diffuse lamellar keratitis (DLK), which is characterized by diffuse infiltrates at the flap margin, can lead to pain, photophobia, blurred vision and, eventually, corneal melting. Clinically, patients with DLK will present with progressive hyperopia and irregular astigmatism. The stages of DLK, based on clinical appearance relative to the intensity of inflammation, are broken down as follows:
- **Stage I** includes an infiltrate in the periphery of the flap.
- **Stage II** is when the infiltrate involves the periphery and visual axis.
- **Stage III** is identified by a cluster of inflammatory cells in the central cornea.
- **Stage IV** is severe inflammation and the beginning of corneal melting, followed by corneal scarring, loss of visual acuity and irregular astigmatism (Figure 2).

Treatment
The goal of treatment is to remove the agent contributing to the corneal melt process. The cause can be multifactorial and will demand a variable approach. One approach to treatment is observation only. If the corneal melt is not progressing or causing visual complications, the process may be self-limiting. Obtaining accurate slit lamp photographs of the corneal melt will aid in deciding if progression is occurring. If the condition progresses or causes degradation in vision, surgical intervention may be necessary.

Epithelial ingrowth can be aggressive, so the need for treatment is immediate. Since it is more common following LASIK, these patients require careful observation. If the epithelial ingrowth involves 30% of the flap, or is associated with corneal melting proven by clinical or topographical examination, consider treatment.
If a patient is having changes in vision or if topographical changes are noted, consider surgical intervention. The general procedure for removing epithelial ingrowth requires a surgeon to lift the flap and scrape the epithelial cells from the stromal bed and undersurface of the flap. At the conclusion of the procedure, a bandage contact lens is typically placed and the patient is started on topical antibiotics and steroids.

Other treatments, such as ethanol, mitomycin and phototherapeutic keratectomy, have been suggested for epithelial ingrowth, but adverse events are possible. These should all be considered secondary options, if the risk associated with scraping is too great.

DLK treatment in the early stages (I or II) involves a topical steroid to reduce the inflammatory event and decrease the likelihood of a corneal melt process. If a patient presents with advanced stage III or IV DLK, surgical intervention of lifting the LASIK flap, cleaning the interface, and treatment with both a topical corticosteroid and an antibiotic will be necessary.

Before treating post-LASIK patients who have autoimmune disorders, corneal dystrophies or dry eye syndrome—all conditions that can lead to a corneal melt or have led to a corneal melt episode—you must address the underlying condition first. This may involve consultation with the patient’s rheumatologist in regards to more specifically targeted control of the patient’s autoimmune disorder. In the case of dry eye syndrome and other corneal ocular surface diseases, treatment may include artificial tears, punctal plugs, topical cyclosporine drops, topical corticosteroid drops, autologous serum topical drops, meibomian gland dysfunction treatment or the use of amniotic membrane grafts.

Corneal melting following LASIK is a significant complication that requires prompt recognition. A detailed preoperative examination for refractive surgery patients is necessary to identify underlying systemic and ocular conditions that can predispose a patient to a corneal melt process. Patients who have undergone refractive surgery and have conditions that predispose them to corneal melting should have close follow up. Early identification and prompt treatment can prevent permanent vision loss from this rare but serious refractive complication.

**Fig. 2. Stage IV inflammation is the most severe DLK classification and shows the beginning of corneal melt.**

**A Corneal Melt Case Report**

A 28-year-old female presented to clinic urgently with a complaint of an irritated right eye for about three days. Her ocular history was significant for LASIK in both eyes in 2012, and a LASIK enhancement in the right eye in 2015. Her general medical history was unremarkable. Visual acuity uncorrected was 20/40 OD and pinhole visual acuity was 20/20 OD. Examination of the anterior segment identified a small amount of epithelial ingrowth superior to the nasal, along the flap edge, as well as an inferior temporal corneal melt along the flap edge with a small amount of epithelial ingrowth present.

Treatment options discussed with the patient included lifting the flap and removing the epithelial ingrowth or, a more conservative approach, aggressive ocular surface treatment with close observation. Ultimately, an initial conservative approach was decided on and a treatment of topical corticosteroid, topical cyclosporine, punctal plugs and preservative artificial tears was initiated.

The patient was monitored on a weekly basis for six weeks. No changes of the epithelial ingrowth or corneal melt were noted. Her vision improved from 20/40 OD uncorrected to 20/20 OD uncorrected after six weeks. The foreign body sensation resolved and the patient was tapered off the topical steroid.

Topical cyclosporine was continued and no changes of the cornea melt process or epithelial ingrowth has been noted in four months.

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**Dr. Schweitzer is a cornea, glaucoma, cataract and refractive surgery specialist at Vance Thompson Vision in Sioux Falls, SD.**
When Your Patient’s Expecting

Pregnancy changes things—especially IOP. But when an expectant mom has glaucoma, how to do you proceed before and after baby? Edited by Paul C. Ajamian, OD

I just diagnosed early open angle glaucoma in a 34-year-old patient who is six months pregnant. What are my options on drops, and does anything change after she has the baby and begins nursing?

With 6.3 million pregnancies reported in the U.S. each year, doctors face the prospect of tailoring therapy to both mother and baby during an especially vulnerable time, says Caroline Pate, OD, Associate Professor at University of Alabama School of Optometry.

Address potential concerns early and let patients know which ocular changes may be in store, she says. Among these changes: a natural reduction in IOP. “It's rare to make a diagnosis of glaucoma during pregnancy, because of a natural decrease in intraocular pressure (IOP),” says Dr. Pate. She explains that the increased uveoscleral outflow pathway and decreased episcleral venous pressure, thought to be governed hormonally, typically results in a 19.6% IOP reduction in healthy patients and a 24.4% decrease in ocular hypertensives.

“We often set a target IOP 20% to 30% lower than baseline when initiating glaucoma treatment. Pregnant patients’ IOP may actually drop this amount without therapy!” This may persist several months postpartum.

Risk vs. Reward

Though a need for IOP reduction is rare in these patients, carefully consider the benefits and risks of drugs in the pregnant patient, she advises. The FDA’s risk categories, though recently abandoned, can still help. “Medications in Category A or B are generally accepted safe to use during pregnancy, whereas Category C are prescribed only when the benefit justifies potential risks to the patient and baby,” says Dr. Pate. “Categories D and X are unsafe during pregnancy.”

Drugs approved after June 30, 2013, no longer use this classification system. “Doctors must now read the package inserts and analyze the safety data to make an informed decision,” she says. Drugs approved on or after June 30, 2001 will be phased-in, Dr. Pate explains.

Since no new topical agents have been approved since the new labeling system was initiated, “we can still refer to the more familiar pregnancy category labeling,” says Dr. Pate. Alphagan (brimonidine, Allergan) is the only available drop that falls into the Category B, she explains. “Generally considered safe during pregnancy, avoid Alphagan during lactation since it’s been linked to CNS depression and sleep apnea in breastfeeding infants.”

Oral prostaglandins are sometimes used to induce labor. Though it’s not proven that ocular topical prostaglandins result in a similar effect, it is probably wise to avoid them, she notes. Topical β-blockers should also be avoided, due to the risk of fetal cardiac arrhythmias. Oral carbonic anhydrase inhibitors given during pregnancy have been linked to congenital malformations, so it’s best to avoid the topical counterparts as well, Dr. Pate explains.

New FDA labeling is more sensitive to risk profiles but puts the onus on ODs.

What is considered safe during pregnancy may not be safe during lactation and vice-versa, says Dr. Pate, and she recommends a free, peer-reviewed online database of from the US National Library of Medicine. “The LactMed database includes helpful information such as levels of a particular drug in breast milk, infant levels in blood, potential effects in breastfeeding infants and on lactation itself. Useful apps also exist for ease of use.”

Though topical IOP-lowering drugs generally pose little risk to the fetus, one must still consider the risks and benefits when prescribing, says Dr. Pate. “Treating these glaucoma patients can be challenging. If in doubt, consult the patient’s OB/gyn or PCP prior to treatment.”

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Focus on Refraction

Eye on the Ball
When an athlete presents with significant refractive error, how do we bring his vision up to par? By Marc B. Taub, OD, MS, and Paul Harris, OD

We consider the challenge of providing excellent vision care to be vital for all of our patients. But in athletes, the stakes are especially high: their performance in the chair often correlates directly with their performance on the field. And in dangerous contact sports, sharp vision can keep them free of injury or incident. Sports vision cases are illuminating in helping us understand the unique challenges athletes face and teach us valuable lessons applicable to everyone.

This installment of Focus on Refraction draws from our recent experiences in sports vision at Southern College of Optometry (SCO), which now provides comprehensive sports vision care for athletes attending the University of Memphis (UM).

We recently screened the first one-third of the student athletes—147 athletes—and identified roughly one-third of these 147 to be in need of comprehensive sports vision evaluations. Vision correction and vision therapy was then provided, if determined necessary.

One athlete, J.D., noted on the screening questionnaire that he has a refractive prescription but hasn’t worn glasses or contacts for more than one year. In response to the question, “Do you ever feel yourself making visual errors?” he responded, “Yes, when finding the football.” J.D. plays an inside position on the football team’s defensive line and has two more years of time left in his college career.

J.D.’s unaided visual acuities were 20/22 OD and 20/40 OS. He saw nothing on the Random Dot 3 stereo test and had some intermittent suppression Brock String. When he did see the two strings, they met closer to him when he looked at the bead furthest away from him, but they met further away from him on the near and intermediate beads. Based on the results of this screening, J.D. was brought into SCO’s University Eye Care center where the UM athletic vision program (AVP) is being conducted.

Player’s Stats
During our 90-minute AVP evaluation, we took a thorough history. We found that J.D., an interdisciplinary studies major with an emphasis in health, holds a GPA in the high twos. When asked about the strongest aspect of his play on the field, he said it was getting to the quarterback. He denied having suffered from TBI but stated that, on at least four separate occasions, he wondered if he had sustained injury following hard hits on the field.

He denied seeing double. He did not have his glasses with him; he hadn’t worn them for more than a year. He never wore contact lenses. His last comprehensive visual evaluation occurred in 2013 in his home state, prior to college. We performed our refractive workup on J.D. (Table 1). His visual acuities were found to be nearly identical to what was found at the visual screening: 20/21 OD and 20/39 OS. At near, he showed 20/20 in all conditions, but he held the target much closer than normal working distance—nine inches. His cover test varied at times, which showed near-ortho and moderate to high exophoria. His near point of convergence showed an eight-inch break and a 14-inch recover; his left eye went out objectively, though he never reported seeing double.

After a battery of tests, the most significant finding was this patient’s performance with the ReadAlyzer (Compevo), an infrared eye movement recording device. We had to drop to an eighth grade level reading card in order for J.D. to score the minimum 70% on the comprehen-
sion test. In fact, he performed much higher—90%—on the eighth grade scorecard.

His reading speed was 140 words per minute—one-half the speed expected for an adult-level reader. He stopped 123 times to read 100 words, 37% more than expected—a fifth grade level. He showed only six regressions (going backward within a line of text to reread it), which is actually better than what we expect for an adult-level reader. His average duration of fixation was 0.34 seconds, the expected value for a first grader. This usually signals that the person discusses the story and data (to themselves) during the reading to help them remember.

The Game Plan
Herein lies the primary dilemma:

What do we prescribe? We had a conundrum, and a thorny one at that. The patient shows very poor binocularity. We cannot correlate the cause and effect because this patient was new and we did not have access to his previous exam data. Thus, whether or not the poor binocularity led to his suppression and blurring of the left eye’s input or vice versa did not weigh into what was prescribed.

We do know from experience, however, that if all of a sudden he gets two clean streams of data, he doesn’t have the software to use them seamlessly. And that’s not taking into account the spatial changes one gets with cylinders like that.

Fortunately, we were at least three months from the football season, and the patient is in his junior year. A third member of our sports vision team, Christina Newman, OD, will fit him with contact lenses for maximum visual acuity as we simultaneously commence a vision therapy program.

The contact lenses alone will not address J.D.’s severe binocular dysfunction, which manifests as dual convergence and accommodative insufficiencies. We initiated an intensive vision therapy program, to help J.D. learn to balance use of his two eyes together and to make quick spatial adjustments on the field and in the classroom.

We considered whether or not it would suffice to prescribe one contact lens on his left eye. However, we felt the jump from 20/21 unaided to 20/14 with the cylinder in place would be quite significant in high-level, division I NCAA sports, so we elected to fit contacts for both eyes.

Lastly, we considered whether to prescribe glasses at all or opt only for contact lenses. Of course, we recognize that all patients who wear contact lenses will face circumstances when they should not wear their contacts, in which case their glasses become an emergency backup. At this point in his care, the spatial distortions caused by glasses

Table 1. Refractive Workup
Distance retinoscopy:
OD +0.50 -1.75 x 180
OS +3.25 -2.00 x 175

Binocular balance (most plus to the first good 20/20 done binocularly):
OD +0.50 -1.00 x 180
OS +2.75 -2.50 x 180 20/20 OU

Second refractive endpoint**—i.e., the lens through which he saw the 20/20 letters to be perceptually the largest:
OD plano -1.00 x 180 20/14
OS +2.25 -2.50 x 180 20/19 VA OU 20/14

Following this, we did the rest of our binocular testing. The key findings included:
Distance base out: x / 18 / 2
Distance base in: x / 12 / 4
Near base out: x / ?? (he never reported it doubling)
Near base in: x / 30 / 12
PRA: -0.50
NRA: +1.50
Stress point retinoscopy: +1.50

Large shapes are graded from 600 to 400 seconds of arc. These 10 circles on the top grade down to 12.5 seconds of arc. The three lower rows have Lea symbols with intermediate stereo values.
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Performing the Random Dot 3 (left) and Brock string tests (right).

could amplify the binocular issues too much to be practical for J.D.

4th and Goal

This clinical experience tells us two important things. First, when working with athletes it is important to instill confidence that we can and will help them from the get-go. In this light, prescribing glasses that would amplify J.D.’s problems was not conducive to a good working relationship. Second, this case tells us that once the binocular problems have been addressed sufficiently, glasses can be prescribed, which will be adapted to rather easily.

Note: The visual acuities reported here have finer gradations than are part of normal charts. We use the M&S Technologies Smart System with a program that allows for continuously variable-sized Sloan letters; the user employs a step program to find thresholds which are quite accurate and repeatable.

**For more on refractive targets, see our prior column “The Endpoint Endgame,” December 2015, p. 28.”

The ReadAlyzer saccadic test being used on a student athlete.

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Nothing can ruin a day faster than being notified you have been referred to an insurance carrier’s Special Investigations Unit because of over-use of special ophthalmic tests. This is particularly prevalent in glaucoma testing. Understanding a few key rules will help protect yourself, your patient and your practice.

Rule #1 – Medical Necessity Rules The Day
When ordering any special ophthalmic test you will submit to a third party for payment, clearly establish why you ordered the test and why it’s necessary in this patient’s case. Each test you order and perform must individually meet the requirement for medical necessity, which is based upon a clinical finding discovered during the patient exam. Your medical record must contain a written statement of this necessity.

Rule #2 – Individual I/R
Each ophthalmic test you perform requires its own interpretation and report (I/R) to be considered complete or billable. Each test and I/R must stand on its own, and be reflected in the medical record. An I/R should contain:
- Clinical findings: pertinent findings regarding the test results
- Reliability of the test
- Comparative data: comparison to previous results, if applicable
- Clinical management: how the results will affect management of the condition/disease, i.e.:
  - Change, increase or stop medication
  - Recommendation for surgery
  - Recommendation for further diagnostic testing
  - Referral to a specialist for additional treatment

Rule #3 – Choose Tests Wisely
You should not apply a standard battery of tests on every glaucoma suspect patient. Choose your tests based upon their individual validity in that specific case, since you must demonstrate necessity for each test that you order and perform.

Rule #4 – Understand Your Provider Contracts
When you became a participating provider with a third party carrier, you received a document typically called a provider agreement—essentially a contract defining the parameters of what you can and cannot do with a patient with respect to covered services under that carrier’s plans. Policies and requirements are contained within this document or are tied to other references used by the carrier. Keep yourself up to date with current contract requirements, as they change frequently.

Rule #5 – Don’t Fudge A Diagnosis To Get Coverage
The number of times I am contacted by doctors asking what diagnosis to use to get a particular test paid for would amaze you. The ICD-10 is quite unforgiving—it is specific enough you can accurately report the diagnosis to the carrier, and it should support the necessity for the specific test in question. Take the time to learn the ICD rules—they are not just codes to get reimbursed.

Rule #6 – Love Your Sales Reps, Know Your Carrier Rules
Often, equipment manufacturers feel a test should be reimbursable for a specific disease state. They will have literature and studies that look impressive clinically, yet a carrier may not have a policy or reimburse you for the test. Manufacturers can make a clinical case to the carrier’s medical directors and demonstrate their technology’s efficacy in the diagnosis of disease, but they don’t always do this. It’s important to know your carriers’ specific policies.

Overtesting is a big concern today and is contributing to carriers’ rising costs.¹ The CMS comparative billing reports highlight how carriers are looking at the frequency of testing and the combination of tests used on individual patients and in the aggregate within your practice. Outcome-based care rewards those who demonstrate the best outcomes in the most efficient manner. If you were paid a fixed fee per year for a patient with a specific diagnosis, would you still test to the same level, even without compensation for each test? The answer is important today, and will be for years to come.

Send questions and comments to ROcodingconnection@gmail.com.

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Angle-closure Glaucoma: Are You Ready?

Diagnosing and managing these challenging cases is inevitable. Be prepared with these clinical pearls. By Michael Cymbor, OD

On a fine, spring Sunday evening about 17 years ago, I was on call at our eye care group and on the phone with the daughter of an 82-year-old who had complained of eye pain early that morning. The daughter said her mother’s eye was red, vision seemed cloudy and she felt nauseous and had a headache. I remember giving her a directive: “Look at your mom’s pupils and see if they look different.” She returned and said, “Yes, the eye that’s red has a larger pupil.” Individually, each symptom could take the clinician down many paths. Taken together, an acute angle closure must be strongly considered. I told her to meet me at the office in 15 minutes.

Fortunately, with a prompt diagnosis we were able to break that patient’s angle-closure attack and get her in for a laser peripheral iridotomy (PI) quickly.

Because most optometrists will see an angle-closure attack similar to this at some point, an understanding of the current thinking in the management of narrow-angle and angle-closure glaucoma and how optometrists are poised to stabilize and manage these patients is vital.

Angle-closure Up Close

Angle-closure glaucoma (ACG) affects 20 million people worldwide, and about four million are bilaterally blind.1 Reports suggest the total number of people between the ages of 40 and 80 affected by angle-closure glaucoma will increase to 23 million by 2020 and 32 million by 2040.2 ACG causes nearly half of all glaucoma blindness worldwide.3 Even though there are three times more people worldwide with primary open-angle glaucoma (POAG) than ACG, angle-closure’s increased morbidity causes blindness in about the same number.4 Patients undergoing an acute symptomatic angle-closure attack present with symptoms of ocular or periocular pain, reduced vision with halos, eye redness and nausea.

![Diagram of the eye showing CB, SS, PTM, ATM, SL.](image)

Fig. 1. The ciliary body (CB) is the most posterior structure visible, followed by the scleral spur (SS), posterior trabecular meshwork (PTM), anterior trabecular meshwork (ATM) and, finally, Schwalbe’s line (SL).
or vomiting. Ocular signs include elevated intraocular pressure (IOP), corneal edema, mid-dilated pupil, shallow anterior chamber and conjunctival injection with ciliary flush.3

There are three currently accepted categories for angle-closure disease: primary angle-closure suspect, primary angle-closure and angle-closure glaucoma.17 An ACG suspect has an angle where the trabecular meshwork cannot be seen for half or more of the angle gonioscopically, indicating at least 180 degrees of iridotrabecular contact. These patients will not have peripheral anterior synchiae, which are the result of long-term iridotrabecular contact.8 A primary angle-closure patient will have a closed angle with a rise in IOP, possibly with peripheral anterior synchiae. Patients with ACG will have a closed angle, peripheral anterior synchiae and evidence of glaucomatous damage in either the disc or field. The glaucomatous damage of the nerve in patients with ACG is similar in nature to glaucomatous damage in patients from POAG, while the field defect may be more diffuse in ACG.9

Demographic risk factors include female gender, advanced age and Asian ancestry.10-12 Ocular risk factors include narrow angles, shallower axial and limbal anterior chamber depth, thicker lens, shorter axial length, more anteriorly positioned lens, smaller corneal diameter and hyperopic refraction.3 Population-based studies suggest a genetic component, but the exact genetic pattern remains elusive.13

Anatomy

Although an underused procedure, gonioscopy remains the standard for viewing the angle and making the diagnosis of angle-closure. One study found that less than half of all eye care providers performed gonioscopy on their glaucoma patients.14 Understanding the anatomy is crucial to help identify variances associated with ACG (Figure 1).

In an open angle, the most posterior structure visible is the ciliary body (CB), which is found between the iris root and the scleral spur. It can vary from light gray to brown and may reduce complete visualization. The second most posterior structure, the scleral spur, is found in the posterior margin of the scleral sulcus, between the CB and the trabecular meshwork (TM). It is made up of collagen tissue, serves as the anchor for the ciliary muscle and can vary in color from white to gray. The TM is next, found between the scleral spur and Schwalbe’s line. It can be subdivided into anterior and posterior TM. It is typically light gray in younger patients and becomes more pigmented over time. The anterior third of the TM is non-functional, while the posterior two-thirds filters aqueous into Schlemm’s canal. Schwalbe’s line is the most anterior angle structure and represents the end of a clear cornea.

There are three main classification systems—Scheie, Shaffer and Spaeth (Tables 1-3) for ACG—each with its own strengths and weaknesses.15-17 In general, using these grading systems may complicate comanagement between clinicians, as a grade 1 can mean two vastly different angle configurations. A good rule is always to describe the last structure seen.

Mechanisms

Angle-closure refers to the appositional closure of the anterior chamber angle, resulting in aqueous obstruction. The most common underlying mechanism of primary angle-closure is pupillary block, in which the aqueous forces the pupil forward.18 The term primary means there is no detectable cause. Ninety percent of all US patients presenting with angle-closure have pupillary block.6 Pupillary block occurs when the pressure of the posterior chamber exceeds the pressure of the anterior chamber, pushing the peripheral and midperipheral iris forward and blocking the TM. The second mechanism of primary angle-closure is plateau iris, which occurs when the CB is anterior or rotated forward, displacing the peripheral iris into the TM.6

Secondary angle-closure occurs by a known pathology. An example of a secondary angle-closure is phacomorphic glaucoma, which occurs when the lens pushes the iris forward and closes the angle.19 This may also occur in subluxation. Uveitis may cause a secondary pupil block, which is characterized by iris bombe and posterior synchiae. Other secondary causes include neovascularization, malignant glaucoma, retinopathy of prematurity, posterior scleritis, acquired immunodeficiency syndrome, Vogt-Koyanagi-Harada syndrome, leukemia, orbital or carotid cavernous fistula and neuropathia epidemica.20

Clinicians should also be aware of masqueraders such as: glaucomatocyclitic crisis, steroid-induced glaucoma, phacolytic glaucoma, ghost cell glaucoma, hemorrhagic glaucoma and exfoliation glaucoma.20

Pharmacologic Causes

Numerous prescription and OTC medications may induce angle narrowing or angle closure. Such medications may cause up to 33% of all angle-closure attacks.21 Some of these drugs, including cholinergics such as Salagen (pilocarpine HCl, Pfizer) and Evoxac (cevimeline hydrochloride, Daichi Sankyo), move the lens-iris diaphragm forward. Iris dilation may occur from
Case 1
A 55-year-old white female presented with intense pain in the left eye. She reported it began the previous night shortly before bed and has progressed since. She now reports the pain as 11 on a scale of one to 10. Her ocular history is significant for an optic nerve coloboma in the left eye.

Visual acuity was 20/20 OD and light perception (LP) OS because of the coloboma. She reported seasonal allergies controlled with Claritin (loratadine, Bayer). Goldmann tonometry was 20mm Hg OD and 56mm Hg OS. Biomicroscopy OS revealed corneal edema, grade 1 cells in anterior chamber, grade 1 Van Herick and a dense, grade 4 nuclear sclerotic cataract. Gonioscopy revealed a closed angle with no view of TM and no peripheral anterior synechiae. Anterior segment angle OCT confirmed angle closure (Figure 2). We diagnosed her with phacomorphic acute angle closure.

We instilled one drop of Iopidine (0.5% apraclonidine hydrochloride, Alcon), followed a few minutes later by one drop of Cosopt (dorzolamide HCl/0.5% timolol maleate ophthalmic solution, Merck). This was repeated 20 minutes later. We also gave the patient 500mg acetazolamide PO. Approximately one and a half hours after diagnosis, the patient’s IOP was 47mm Hg. Lacking isosorbide, we performed compression gonioscopy, which lowered IOP to 34mm Hg.

A repeat OCT angle was still quite narrow, but open (Figure 3). We scheduled the patient for immediate laser PI OS with subsequent cataract surgery a few days later. Her pressure is now stable in the 15mm Hg to 17mm Hg range OS.

Diagnostic Tools
While gonioscopy remains the standard for diagnosing angle-closure, angle OCT and ultrasound biomicroscopy (UBM) are playing an increasingly important role. Both of those technologies can give an objective assessment of the angle width.22 Angle OCT is non-contact, is more tolerable to the patient and provides better resolution. UBM can image the CB more clearly because of deeper sound wave penetration. In the same way that posterior segment OCT imaging may not be optimal for visualizing characteristics such as small drance-type optic nerve hemorrhages, angle OCT and UBM may not be adequate for distinguishing between peripheral anterior synechiae and iridotrabecular contact.

While these imaging technologies may eventually become a replacement for gonioscopy, currently they are more of an adjunct.

Treatment
The first goal in the management of angle closure is the immediate reduction of IOP to prevent permanent damage. This can be accomplished with medications such as apraclonidine, 0.5% timolol or combined formulation of apraclonidine and timolol such as Cosopt. If topically administered medications do not reduce IOP sufficiently, systemic acetazolamide can be added.24 If IOP reduction is still suboptimal, laser PI can be performed.25 If angle closure is not responsive to medical management, cataract surgery with angle preserving incision is the treatment of choice.

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ACG is IOP reduction. The second goal is reversing the mechanism of angle closure.

**Topical**

Often used as initial treatment, eye drops that can quickly reduce IOP include beta blockers, alpha agonists, carbonic anhydrase inhibitors and pilocarpine. Beta blockers, alpha agonists and carbonic anhydrase inhibitors all quickly reduce aqueous production, making them ideal to use when rapid IOP reduction is desired. Pilocarpine constricts the pupil, which is helpful for subsequent laser PI. Even though pilocarpine increases the angle width in patients with narrow angles, it may actually narrow the angle in eyes with phacomorphic glaucoma, pseudoexfoliation and vitreous block glaucoma.27-29 Prostaglandins may not be as effective because of delayed onset and may increase anterior chamber inflammation.

Oral or intravenous acetazolamide or hyperosmotics can also help relieve elevated IOP. Because quick reduction is warranted, acetazolamide sequels are less effective, as they reduce pressure slowly.

Topical steroids are helpful to relieve inflammation, and topical osmotic agents such as glycerin can reduce corneal edema and clear the cornea quickly if corneal edema is present and the anterior chamber and iris structures are difficult to clinically visualize.

Optometrists should be aware of medication contraindications, including: asthma and COPD for beta blockers; severe cardiac and cerebrovascular disease for alpha agonists; and kidney disease or sulfa allergies for carbonic anhydrase inhibitors. Clinicians must always weigh the treatment risks with the risks of nontreatment with conditions such as ACG that can rapidly cause irreversible blindness.

If medical management is unsuccessful in returning IOP to a safe level, clinicians should consider indentation or compression gonioscopy. When performing indentation gonioscopy, use a small footprint goni lens and apply a significant amount of pressure. The force transferred to the angle may move the peripheral iris away from the TM, suddenly reducing IOP. This will also help determine the extent of peripheral anterior synechiae. Angles with higher amounts of peripheral anterior synechiae are more likely to fail IOP reduction attempts with medical treatment and laser PI because of the iris mechanically adhering and blocking the trabecular meshwork.31 Paracentesis may help to quickly reduce IOP and pain, but clinicians must use caution, as the anterior chamber will be shallow. Paracentesis is effective in primary angle-closure but may not be as successful in secondary angle-closure.32

**Surgical**

Laser PI is the mainstay of angle-closure treatment. Creating an alternate outflow pathway allows the aqueous

---

**Table 1. Scheie Classification System**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Open</td>
<td>All structures visible</td>
</tr>
<tr>
<td>I</td>
<td>Iris root visible, difficult to see into recess</td>
</tr>
<tr>
<td>II</td>
<td>Narrow ciliary band</td>
</tr>
<tr>
<td>III</td>
<td>Only anterior trabeculum visible, posterior trabeculum obscured</td>
</tr>
<tr>
<td>IV</td>
<td>Only Schwalbe’s line visible = closed angle</td>
</tr>
</tbody>
</table>

**Table 2. Shaffer Classification System**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Angle Width</th>
<th>Description</th>
<th>Risk of Closure</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>45-35</td>
<td>Wide open</td>
<td>Impossible</td>
</tr>
<tr>
<td>3</td>
<td>35-20</td>
<td>Wide open</td>
<td>Impossible</td>
</tr>
<tr>
<td>2</td>
<td>20</td>
<td>Narrow</td>
<td>Possible</td>
</tr>
<tr>
<td>1</td>
<td>≥10</td>
<td>Extremely narrow</td>
<td>Probable</td>
</tr>
<tr>
<td>Slt</td>
<td>Slt</td>
<td>Narrowed to silt</td>
<td>Probable</td>
</tr>
</tbody>
</table>

**Table 3. Spaeth Classification System**

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A: Anterior: iris inserts anterior to SL</td>
<td></td>
</tr>
<tr>
<td>B: Behind Schwalbe’s line: anterior to posterior limit of the TM, or between SL and SS</td>
<td></td>
</tr>
<tr>
<td>C: Sclera: posterior to SS. SS is visible</td>
<td></td>
</tr>
<tr>
<td>D: Deep: deep into the CB</td>
<td></td>
</tr>
<tr>
<td>E: Extremely deep: very deep into the CB</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Angular width</th>
<th>Estimated angle in degrees</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Iris configuration</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>B: (steep) bowing anteriorly, graded on a 1-4+ scale</td>
<td></td>
</tr>
<tr>
<td>P: plateau configuration</td>
<td></td>
</tr>
<tr>
<td>F: flat configuration</td>
<td></td>
</tr>
<tr>
<td>C: concave, posterior bowing</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pigment grading</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pigment in PTM at 12 o’clock position graded on a 0-4+ scale</td>
<td></td>
</tr>
</tbody>
</table>
to bypass the pupil, thus eliminating the pressure differential between the anterior and posterior chambers.

The iris will then return from a convex configuration in the midperipheral and peripheral area to neutral, thus opening the angle. While laser PI is often successful at reducing IOP and successfully treating angle-closure glaucoma, subsequent treatment with eye drops, surgery or both is often necessary. Clinicians should also perform a laser PI on the fellow eye, as roughly half will have an angle-closure event within five years if left untreated. The patient should be evaluated at least one day, one week and one month after a PI procedure for angle-closure.

Many clinicians recommend cataract surgery within a few weeks of a patient successfully treated for an acute angle-closure for two reasons. First, cataract surgery opens the angle more than a laser PI. Second, the patient typically requires fewer pressure-lowering medications after cataract surgery. In fact, several studies indicate clinicians should be recommending cataract surgery in lieu of laser peripheral iridotomy. Cataract surgery may be helpful at each and every stage of angle-closure treatment. Though controversial, some are advocating refractive lens exchange in angle-closure patients with clear lenses. The EAGLE study (Effectiveness in Angle-closure Glaucoma of Lens Extraction) may soon give additional insight to this alternate treatment option.

If patients are unresponsive to medical therapy or laser PI, or if the cornea prevents adequate visualization for a laser iridotomy, some clinicians recommend iridoplasty—a procedure that uses a laser to contract the peripheral iris stroma away from the angle. This underscores the importance of reducing illumination during gonioscopy to avoid artificially causing pupil constriction. We educated her on the signs and symptoms of angle closure, and she understands the need to contact us immediately if she notices any of them. We have decided against a laser PI and are monitoring her angles every six months.

Borderline Cases
The challenge is deciding whether or not to recommend laser PI for all patients with narrow angles. Clinicians may be tempted to do...
INDICATIONS AND USAGE

SIMBRINZA® (brinzolamide/brimonodine tartrate ophthalmic suspension) 1%/0.2% is a fixed combination indicated in the reduction of elevated intraocular pressure (IOP) in patients with open-angle glaucoma and/or ocular hypertension. The recommended dose is one drop of SIMBRINZA® Suspension in the affected eye(s) three times daily. Shake well before use. SIMBRINZA® Suspension may be removed during instillation of SIMBRINZA® Suspension but may be reinserted 15 minutes after instillation.

IMPORTANT SAFETY INFORMATION

Contraindications

SIMBRINZA® Suspension is contraindicated in patients who are hypersensitive to any component of this product and neonates and infants under the age of 2 years.

Warnings and Precautions

Sulfonylurea Hypoglycemia—Sulfonylurea hypoglycemia may occur. Patients should be monitored for hypoglycemia, particularly in the presence of other factors that may increase the risk of hypoglycemia (e.g., fasting, decreased caloric intake, concurrent administration of potentially hypoglycemic agents, drug interactions). Severe hypoglycemia may be an initial manifestation of sulfonylurea hypersensitivity. If sulfonylurea hypersensitivity is suspected, SIMBRINZA® Suspension should be discontinued and an alternative treatment started.

Contact Lens Wear—The preservative in SIMBRINZA® Suspension, benzalkonium chloride, may be absorbed by soft contact lenses. Contact lenses should be removed during instillation of SIMBRINZA® Suspension but may be reinserted 15 minutes after instillation.

Severe Cardiovascular Disease—Brimonidine tartrate, a component of SIMBRINZA® Suspension, has a less than 5% mean decrease in blood pressure 2 hours after dosing in clinical studies; caution should be exercised in treating patients with severe cardiovascular disease.

Adverse Reactions

SIMBRINZA® Suspension

In two clinical trials of 3 months’ duration with SIMBRINZA® Suspension, the most frequent reactions associated with its use occurring in approximately 3-5% of patients in descending order of incidence included: blurred vision, eye irritation, dysgeusia (bad taste), dry mouth, and eye allergy. Adverse reaction rates with SIMBRINZA® Suspension were comparable to those of the individual components. Treatment discontinuation, mainly due to adverse reactions, was reported in 11% of SIMBRINZA® Suspension patients.

Drug Interactions

Consider the following when prescribing SIMBRINZA® Suspension: Concomitant administration with oral carbonic anhydrase inhibitors is not recommended due to the potential additive effect. Use with high-dose salicylate may result in acid-base and electrolyte alterations. Use with CNS depressants may result in an additive or potentiating effect. Use with antihypertensives/cardiac glycosides may result in additive or potentiating effect on lowering blood pressure. Use with triyclic antidepressants may blunt the hypertensive effect of systemic clonidine and it is unknown if use with this class of drugs interfere with IOP lowering. Use with monoamine oxidase inhibitors may result in increased hypotension.

Learn more at myalcon.com/simbrinza

For additional information about SIMBRINZA® Suspension, please refer to the brief summary of the full Prescribing Information on the following page.
**Triyclic Antidepressants** - Tri cyclic antidepressants have been reported to blunt the hypotensive effect of systemic clonidine. If it is not known whether the concomitant use of these agents with the individual components in humans can lead to resulting interference with the IOP-lowering effect. Caution is advised in patients taking tri cyclic antidepressants which can affect the metabolism and uptake of circulating amine.

**Drug Interactions**

- **Oral Cinnamic Acid Derivatives** - Oral cinnamic acid derivatives can cause additive effects on blood pressure in patients receiving brimonidine tartrate or brinzolamide. It is recommended that blood pressure should be monitored closely.
- **Dopaminergic Agonists** - Caution is advised in patients taking dopaminergic agonists, which can affect the metabolism and uptake of circulating amine.

**Contraindications**

- **Children** - SIMBRINZA Suspension is contraindicated in children under 2 years of age.
- **Hypersensitivity** - SIMBRINZA Suspension is contraindicated in patients who are hypersensitive to any component of SIMBRINZA Suspension.
- **Pregnancy** - Pregnancy Category C - Developmental toxicity studies with brinzolamide in rabbits at oral doses of 1/10, 1/3, and 1/10 mg/kg (50, 15, and 5 mg/kg) respectively) resulted in an increased incidence of fetal resorption, fetal weight, and organ weight changes. There is no evidence of harm to the fetus. Administration of brinzolamide to pregnant rats resulted in an increased incidence of fetal resorption, fetal weight, and organ weight changes. There is no evidence of harm to the fetus.

**Warnings and Precautions**

- **Bronchodilators** - Bronchodilators, such as albuterol, can reduce the IOP-lowering effect of brinzolamide.
- **Beta-Adrenergic Blockers** - Beta-adrenergic blockers, such as propranolol, can reduce the IOP-lowering effect of brinzolamide.

**Adverse Reactions**

- **Clinical Studies** - In clinical studies, the majority of patients receiving SIMBRINZA Suspension experienced no adverse reactions. In the case of brinzolamide and brimonidine tartrate, the most commonly reported adverse reactions were:
  - **Gastrointestinal**:
    - Nausea
    - Abdominal pain
  - **Skin**:
    - Dermatitis
    - Pruritus
  - **Ocular**:
    - Conjunctivitis
    - Blurred vision
  - **Other**:
    - Headache

- **Postmarketing Experience** - The following reactions have been identified during postmarketing use of brinzolamide:
  - **Gastrointestinal**:
    - Nausea
    - Abdominal pain
  - **Skin**:
    - Dermatitis
    - Pruritus
  - **Ocular**:
    - Conjunctivitis
    - Blurred vision
  - **Other**:
    - Headache

**Pregnancy**

- **Category C** - Developmental toxicity studies with brinzolamide in rabbits at oral doses of 1/10, 1/3, and 1/10 mg/kg (50, 15, and 5 mg/kg) respectively) resulted in an increased incidence of fetal resorption, fetal weight, and organ weight changes. There is no evidence of harm to the fetus. Administration of brinzolamide to pregnant rats resulted in an increased incidence of fetal resorption, fetal weight, and organ weight changes. There is no evidence of harm to the fetus.

**Concomitant Use of Other Ophthalmic Drugs** - If more than one topical ophthalmic drug is being used, the drugs should be administered at least 5 minutes apart. If one of the drugs has a potential for serious adverse reactions, the treatment should be discontinued. If more than two topical ophthalmic drugs are being used, the drugs should be administered at least 5 minutes apart. If one of the drugs has a potential for serious adverse reactions, the treatment should be discontinued.

**Dosage and Administration**

- **Adults** - The recommended dose of SIMBRINZA Suspension in the adult is 1 drop (0.03 ml) twice daily, 5-10 minutes apart. Patients should be instructed to use the dropper fully to ensure delivery of the correct dose.
- **Children** - SIMBRINZA Suspension is not recommended in children under 2 years of age.

**Drug Interactions**

- **Beta-Adrenergic Blockers** - Beta-adrenergic blockers, such as propranolol, can reduce the IOP-lowering effect of brinzolamide.
- **Dopaminergic Agonists** - Dopaminergic agonists, such as amantadine, can reduce the IOP-lowering effect of brinzolamide.

**Contraindications**

- **Children** - SIMBRINZA Suspension is contraindicated in children under 2 years of age.
- **Hypersensitivity** - SIMBRINZA Suspension is contraindicated in patients who are hypersensitive to any component of SIMBRINZA Suspension.

**Warnings and Precautions**

- **Bronchodilators** - Bronchodilators, such as albuterol, can reduce the IOP-lowering effect of brinzolamide.
- **Beta-Adrenergic Blockers** - Beta-adrenergic blockers, such as propranolol, can reduce the IOP-lowering effect of brinzolamide.

**Adverse Reactions**

- **Clinical Studies** - In clinical studies, the majority of patients receiving SIMBRINZA Suspension experienced no adverse reactions. In the case of brinzolamide and brimonidine tartrate, the most commonly reported adverse reactions were:
  - **Gastrointestinal**:
    - Nausea
    - Abdominal pain
  - **Skin**:
    - Dermatitis
    - Pruritus
  - **Ocular**:
    - Conjunctivitis
    - Blurred vision
  - **Other**:
    - Headache

- **Postmarketing Experience** - The following reactions have been identified during postmarketing use of brinzolamide:
  - **Gastrointestinal**:
    - Nausea
    - Abdominal pain
  - **Skin**:
    - Dermatitis
    - Pruritus
  - **Ocular**:
    - Conjunctivitis
    - Blurred vision
  - **Other**:
    - Headache

**Pregnancy**

- **Category C** - Developmental toxicity studies with brinzolamide in rabbits at oral doses of 1/10, 1/3, and 1/10 mg/kg (50, 15, and 5 mg/kg) respectively) resulted in an increased incidence of fetal resorption, fetal weight, and organ weight changes. There is no evidence of harm to the fetus. Administration of brinzolamide to pregnant rats resulted in an increased incidence of fetal resorption, fetal weight, and organ weight changes. There is no evidence of harm to the fetus.

**Concomitant Use of Other Ophthalmic Drugs** - If more than one topical ophthalmic drug is being used, the drugs should be administered at least 5 minutes apart. If one of the drugs has a potential for serious adverse reactions, the treatment should be discontinued. If more than two topical ophthalmic drugs are being used, the drugs should be administered at least 5 minutes apart. If one of the drugs has a potential for serious adverse reactions, the treatment should be discontinued.
so, although management of these patients is not clear cut, as fewer than one in 20 gonioscopically narrow eyes will develop angle-closure. Additionally, laser PI may hasten the development of cataracts.\(^5^9\)

We don’t have better guidelines on when to prophylactically treat because we still have very little insight on who will and who won’t have an angle-closure attack. Unfortunately, clinicians must rely on gonioscopy—which is an imperfect test because of subjectivity—and inconsistent results with different testing variables such as illumination. Additionally, we still do not completely understand all the variables that lead to angle-closure, and the variables we do understand have limited predictive value. One variable that may hold promise in helping to better understand the mechanism of angle-closure lies in the fact that the iris squeezes aqueous from its stroma when the pupil dilates.\(^1^0\) The iris that holds more water upon dilatation may be at a higher risk for an angle-closure attack.\(^1^1\) This may eventually prove to be an important measurement in clinical practice.

Although angle-closure glaucoma can be challenging, optometrists are in an optimal position to manage these patients. Timely diagnosis using gonioscopy is critical, as is IOP control. The optometrist must then either perform the laser PI (in states that permit such treatment) or promptly refer the patient for laser PI or cataract surgery.

Dr. Cymbor is a partner with Nittany Eye Associates in State College, PA, and is a member of the Optometric Glaucoma Society. He is a speaker for Optos. Dr. Cymbor would like to thank Isaac Lindenmuth, fourth-year Salus student, for compiling tables 1-3 and taking figures 1, 4, 5, and 6.

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Images courtesy of The Westin Kierland Resort
A great debate seems to rage in optometry circles concerning the pronunciation of gonioscopy. Whether you say “go-knee-ah-scopy” or “gah-knee-ah-scopy” largely depends on your particular geographical location or optometry school alma mater. Regardless of which side of this great schism you find yourself, the examination technique itself remains one of the most illuminating available for optometrists.

It is essential in differentiating glaucoma subtype and determining proper medical or surgical treatment interventions. In addition to its use in the classification of glaucoma, gonioscopy aids in evaluation of iris cysts and tumors, examination of neovascularization of the anterior chamber angle, and in the search for intraocular foreign bodies.

Although the value of gonioscopy is evident, two separate studies reviewing patient records show that less than half of primary open angle glaucoma (POAG) patients had a single gonioscopy procedure during their initial glaucoma workup.1 2 Perhaps, clinicians find it difficult to obtain adequate views due to improper technique,
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poor patient cooperation or lack of practice. This could be due, in part, to increased use of newer technologies such as anterior chamber OCT, ultrasound biomicroscopy or an over-reliance on Van Herick angle estimation. New technologies enable us to evaluate patients in new ways, and even allow optometric physicians to delegate more to technicians. As beneficial as new techniques are, only gonioscopy allows us to visualize the entire anterior chamber angle. Gonioscopy is the only technique which permits clinicians to see the angle in true color, as opposed to cross-sectional images interpolated and presented on a screen.

Because light rays from the anterior chamber angle undergo total internal reflection at the cornea-air interface, it is impossible to view the angle unaided. This is why we need a gonioscopy lens. Light rays are able to pass directly into the lens because of its higher index of refraction, then continue through the lens to be viewed by the clinician. This ability to see the angle in vivo assists in evaluating angle pigmentation, recognizing blood in Schlemm's canal and quickly differentiating between apositional and synechial angle closure.

This article provides a review of proper gonioscopy techniques and strategies to be successful, even with your most apprehensive patients. So sit back, relax and enjoy your favorite carbonated beverage, be it a soda, cola or pop. No matter where you live, what school you went to or what you call it, the techniques and skills required for successful gonioscopy are the same.

The Lenses
Direct or indirect? Goldmann or Sussman? Three-mirror or four-mirror or six-mirror? Flange or no-
In interpreting the view, note how open the angle is, the most posterior structure you can see (in this case it’s the ciliary body band) and how much pigment is in the trabecular meshwork.

flange? There are many types of gonioscopy lenses to view the angle, and the one you should use depends on what you’re trying to view.

Direct lenses are typically only used by glaucoma surgeons. These are thick convex lenses used in the operating room on sedated, supine patients. Direct lenses are quite impractical in the optometric clinical setting. In contrast, indirect gonioscopy lenses have a concave contact surface and use a mirror to reflect light from the angle to the observer. This method of gonioscopy is practical and easy to perform with an upright patient seated at a slit lamp.

Indirect lenses use a mirror to reflect light rays exiting the angle towards the examiner and provide a mirror image of the angle opposite the mirror. Numerous styles of indirect lenses exist, with variable numbers of mirrors and radii of curvature of the portion which contacts the cornea. They can generally be classified as large-diameter lenses, which do not compress the cornea, and small-diameter lenses capable of compression gonioscopy.

The Goldmann three-mirror lens is a common, large-diameter indirect lens and, likely, the first one you encountered as an optometry student. The smallest and steepest mirror is used for gonioscopy, while the other two mirrors and the central lens are used for evaluation of the retina. This makes the three-mirror lens a particularly valuable multipurpose tool. Because of its larger diameter, and steeper curvature
than the human cornea, it requires a coupling substance to fill the gap between the lens and cornea. The Goldmann lens provides an excellent view of the angle and usually produces some amount of suction on the cornea, aiding in maintaining the lens on the subject’s eye. This is where gonioscopy is particularly useful. We believe LPI is useful for eyes in which TM is not visible in at least two quadrants, particularly when signs—such as patchy pigmentation, signifying likely prior intermittent closure—are present. Other factors such as age over 60, female gender, hyperopia and a family history may also bolster the decision for prophylactic treatment.

Smaller-diameter lenses, such as the Sussman or Posner four-mirror lenses, are shallow and have a curvature similar to that of the human cornea, so they do not require a coupling agent. These lenses are valuable in indentation gonioscopy—also known as compression gonioscopy—which is very useful in the differentiation of angle closure pathologies.3 Many glaucoma specialists prefer the Posner lens as the handle makes for a convenient and smooth “pivot” of the lens onto the cornea.

Both types of lenses have a place in the primary care setting for your glaucoma patients, and we would suggest you have one of each in your toolbox.

Getting Your Gonioscopy View
Before we begin a stepwise approach to successful gonioscopy, we want to stress that what you see in the slit lamp is not a static picture. Examiners must remain cognizant that they are dealing with living, responsive and deformable tissues. Normal pupil responses (and their subsequent effects on iris conformation) and ocular tissue and fluid dynamics are completely in play during the procedure.

Room illumination, slit lamp light entering the pupil, too much pressure exerted on the examination lens or, in the case of a lens requiring coupling solution, suction on the cornea accompanied by pressure away from the cornea all can change the appearance of the angle.

Step 1: Explain the procedure to the patient. Patients invariably express some degree of apprehension when having anything near their eyes. Patients are much more likely to cooperate if they understand what procedures are being performed and why. Let the patient know that the lens will touch the eye, but will not cause significant discomfort. For those of you who may struggle with how to discuss this procedure with your patients, we’ve provided a video demonstrating our technique as well as our typical conversation with patients.

Step 2: Instill one or two drops of topical ophthalmic anesthetic, such as 0.5% proparacaine, into both eyes. Even if you plan to do the procedure only on one eye, it helps slow the blink rate, which can aid in ease of the procedure.

Step 3: When using the Goldmann three-mirror lens, fill the lens half way with a coupling solution, such as 2.5% methylcellulose or 1% carboxymethylcellulose. There are advantages to each type of
medium. Methylcellulose provides a much sharper, high definition image compared with carboxymethylcellulose. However, methylcellulose is much more toxic to the cornea and must be irrigated from the patient’s eye. The Sussman lens does not require coupling solution.

**Step 4:** Situate the patient comfortably in the slit lamp and ensure they’re at an appropriate level with the lateral canthus marking on the lamp. This will allow easy movement between mirrors without having to readjust the patient after the lens is placed on the eye. Ideally, the patient’s back will be straight and they are not straining to keep their forehead against the headrest. Advise your patient to keep their forehead against the strap, chin in the chinrest and both eyes open—but always keep their teeth together.

**Step 5:** Dim the ambient lights such that the room is almost dark. Ensure that the slit beam is in click. A good starting point for your slit lamp settings is to use a magnification of 10x and a narrow and short light beam that does not enter the pupil and artificially open the iridocorneal angle.

**Step 6:** Apply the lens. For the Goldmann three-mirror, there are a couple of different ways to do this. For less experienced clinicians, a two-handed approach is in order. Tell the patient to look up. Gently pull down the patient’s lower eyelid with your left thumb while you pin the upper lid against the brow using your forefinger. With your right hand, place the gonioscopy lens slightly tilted so as to keep from spilling the coupling solution into the inferior fornix and then quickly tilt the lens onto the cornea. Have the patient look straight ahead, release the upper lid, and exchange hands. Alternatively—and somewhat more efficiently, though difficult for beginners—use your left hand alone to hold the gonioscopy lens while your third finger pulls down the lower eyelid and your second finger pins the upper lid as the patient is looking up. Tilt the lens into the lower cul-de-sac and then pivot onto the cornea. Ask the patient to look straight ahead and release the eyelids. To balance the hand, rest the fingers against the forehead rest and the heel of the hand against the patient’s cheek.

**Step 7:** Removing the lens usually requires the patient to squeeze his eyes closed. Sometimes gentle pressure from the examiner’s forefinger against the globe is required to break suction.\(^5\)\(^6\) As with all things, practice makes perfect. With the Sussman four-mirror, the procedure is less complicated. To examine the patient’s right eye,
hold the Sussman lens with your thumb and forefinger. Instruct the patient to look down. Pin the upper lid with your second finger. Next, instruct the patient to look straight ahead. Use your third finger to gently depress the patient’s lower eyelid. Next, place the lens gently against the cornea. Depending on patient cooperation, you may release the lids at this point. To balance the hand, rest your fingers against the patient’s forehead and the heel of your hand against the patient’s cheek.

Gonioscopy can be difficult for clinicians with short arms. Using the goniolens case or a tissue box on which to rest the elbow can be helpful. In addition, commercially available elbow rests can also be used.

Interpreting Your Gonioscopy View

The key to correctly interpreting and recording your view is to always perform the procedure in the same manner so you have consistent results. We recommend always starting your gonioscopy by viewing the inferior angle. This is typically the widest angle and the easiest to identify structures due to the increased pigmentation. Remember that, with indirect gonioscopy, your mirror is 180° away from the angle you are viewing.

To view the inferior angle, start with the mirror at 12 o’clock. Rotating clockwise will help you to remember the location of any abnormal findings. Begin with low magnification and increase as necessary to obtain more detail. Sometimes, especially in lightly pigmented patients, details are difficult to ascertain. In these cases, we employ a special technique called a “corneal wedge.” The corneal wedge is a very bright, razor thin slit beam, with the light source moved approximately 10 to 20 degrees off-axis. The corneal wedge will reveal Schwalbe’s line as the point at which two prominent corneal reflections come together.

Structures

The anterior limit of the trabeculum, where it meets the posterior termination of Descemet’s membrane, creates an irregular, opaque line called Schwalbe’s line. This line may be difficult to view in young people who typically have less pigmentation in the angle. The corneal wedge technique is helpful in identifying an inconspicuous Schwalbe’s line.

The trabeculum lies posterior to Schwalbe’s line and ends at the scleral spur. It has two parts, an anterior, nonfunctional part and a posterior, functional aspect. As a person ages, the posterior portion becomes increasingly pigmented due to trabecular outflow and the associated pigment debris. Pigmentation is unusual prior to puberty, and a patchy pigment distribution should raise suspicion of intermittent iris contact. Deep to the trabecular meshwork lies Schlemm’s canal, which should appear as a dark line. Blood in the canal indicates a higher than normal episcleral venous pressure.

The ciliary body presents as the most posterior angle structure, and pigmentation varies from no pigment (pink) to dark brown to slate gray.

Tips for Difficult Angles

Sometimes, the angle structures are difficult to view, either because of little-to-no pigmentation, or because the view of the angle is obscured by a forward bowing iris, as seen with iris bombe.

In the case of a lightly pigmented angle, it helps to start with the inferior angle, as it will be the widest and most pigmented. Once you’ve identified the structures here, you will be familiar with the anatomy of the particular patient and comfortable identifying structures in the other quadrants. In addition, the
corneal wedge technique comes in handy in these situations. Note that this technique can only be performed successfully in the superior and inferior quadrants, as it requires the light source to be off-axis.

When the angle is obscured by a steep midperipheral iris, tilting the lens in the direction of the angle you want to view or having the patient look slightly in the direction of the observation mirror will allow the light rays to pass over the obstructing iris and into the angle, allowing a view.

To distinguish between synechial and appositional angle closure, use the small-diameter goniolens to apply gentle pressure against the patient’s cornea. There should be enough pressure to cause wrinkling of the cornea. This pressure should push anterior chamber aqueous against the iris/lens diaphragm, and widen an appositional angle closure. In the case of a synechial closure or plateau iris, the angle will not widen with pressure. This compression gonioscopy technique is helpful in considering whether a patient would benefit from a laser peripheral iridotomy (LPI). If there is no significant improvement/opening of the angle with compression, then an LPI probably wouldn’t help in a narrow angle patient.

With an aging population, the incidence of glaucoma is likely to increase. As primary eye care providers, we want to make sure we employ all the tools available to provide the highest quality of care possible.

Gonioscopy is an easy to perform—and invaluable—procedure. Don’t let the angle get the best of you.

Managing the Post-op Glaucoma Patient

As minimally invasive surgeries and laser procedures become more commonplace, learn the basics to stay ahead of the comanagement curve.

By Anthony Van Alstine, OD, MS, and James M. Caruso, OD

Glaucoma is the second leading cause of blindness worldwide, projected to affect nearly 80 million people by 2020. While several different forms of treatment are available for this debilitating disease, all modalities share the same goal: to preserve vision by lowering intraocular pressure (IOP). The remarkable advancements in surgical techniques, particularly in the areas of glaucoma laser procedures and minimally invasive surgeries, means glaucoma specialists increasingly rely upon the primary care optometrist for pre- and postoperative support.

This article reviews important concepts to better equip optometrists in appropriately managing patients who require glaucoma surgery. We will discuss considerations for surgical intervention, introduce common and emerging surgical procedures and provide expectations of postoperative management. The objective: solidify the optometrist’s understanding to better comanage post-op glaucoma patients.

Surgical Indications

Once considered a last resort, surgery is increasingly viable, even preferable, earlier in the course of the disease. Here are a few reasons to consider surgery for your glaucoma patients.

- **Therapeutic failure.** Reaching an appropriate target IOP for a glaucoma patient may be unobtainable with medical therapy alone, especially if the patient first presents with more advanced disease. The Ocular Hypertension Treatment Study (OHTS) determined that 39% of eyes required two or more medications to achieve a 20% reduction from baseline IOP. Since most patients are initially treated with prostaglandin analogs, practitioners conventionally use β-blockers, α-agonists, carbonic anhydrase inhibitors or a combination of these to further lower IOP. However, most of these second-line drugs show reduced efficacy when used as additive treatments compared with their use as monotherapies.

  **Bottom line:** If target IOP is not achieved, or your patient is showing progressive structural or visual field loss on maximum drug therapy, surgical intervention is required.

- **Poor compliance.** Achieving medication compliance can be a major hurdle for glaucoma patients. Noncompliance with glaucoma regimens is reported to be quite high, ranging anywhere from 25% to 80%. In addition, as the dosing schedule becomes more complex, compliance wanes. In an effort to determine how compliance is affected when an additional medication is added to the regimen, researchers investigated the refill intervals of 4,930 patients using latanoprost before and after adding a second medication. Once the second drug was added, these patients refilled their latanoprost less frequently compared with monotherapy alone.

  From a pragmatic standpoint, a glaucomatous eye can be treated with three different drug classes, using as few as three drops per day if a prostaglandin and one of the combination drugs available on the market are used (i.e., brimonidine-timolol, dorzolamide-timolol and brinzolamide-brimonidine). However, adding a fourth drug can complicate the dosing schedule too much, affecting compliance.

  **Bottom line:** If your patient has a protracted history of noncompliance with drops or if you face the prospect of adding a fourth drug class, it may be time to consider surgery.
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Expense. The cost of medications can complicate, and often limit, how we treat our patients. With glaucoma affecting more than three million Americans—overwhelmingly in the senior citizen cohort, who mostly rely on a fixed income—the United States spends an estimated $2.9 billion annually on glaucoma drugs. The annual cost of care per patient is projected to range from $623 to $2,511, depending on the severity of the disease. Pharmaceutical treatments for glaucoma can be a serious financial burden to our patients—drug cost is among the top barriers to medication adherence. Adding to the problem, several glaucoma drugs remain unavailable as generics. Insurance plans may require evidence of therapeutic failure or intolerance to cheaper medications before approving coverage for brand-name drugs. Bottom line: If your patient is having difficulty affording their glaucoma medications, surgical options may provide a long-term cost savings, depending on their medical insurance coverage.

Adverse effects. Intolerance to topical medications can limit our ability to medically manage glaucoma patients. Optometrists should always be mindful of the side effects associated with the staple glaucoma drugs, such as the cardio and pulmonary effects of β-blockers and the cosmetic changes associated with prostaglandins that include pigmentary darkening around the eyelids, eyelash growth, iris color darkening and periorbital fat loss.

Carbonic anhydrase inhibitors are sulfa drugs and, though they differ in composition from sulfonamide antimicrobials, may cause adverse effects in patients with sulfa allergies. Researchers found that up to 25.7% of patients using 0.2% brimonidine develop an allergic conjunctivitis in response to their drops. Furthermore, repeated exposure to high amounts of preservatives, such as benzalkonium chloride (BAK), has been associated with ocular surface changes, especially for patients with pre-existing ocular surface disease.

Bottom line: If patients are intolerant to one or more glaucoma medications, surgery to lower IOP may be necessary.

Narrow angles. It's essential to assess the iridocorneal angle of all glaucoma patients with gonioscopy. A recent epidemiological study estimates angle closure glaucoma affects 16 million people worldwide, rendering up to 25% of these patients bilaterally blind. If left untreated, an angle-closure attack in one eye means a 40% to 80% chance of developing an attack in the fellow eye in 5 to 10 years.

Bottom line: If your patient is at risk for angle closure or has developed one, surgical intervention is indicated.

Minimally Invasive Surgeries

After decades of stagnation, the options for surgical management have diversified in recent years. Minimally invasive glaucoma surgery (MIGS) options are the hot topic in interventional glaucoma, as these less effective but more patient-friendly alternatives find an appropriate place in the glaucoma management hierarchy.

The more invasive surgeries, such as trabeculectomy and ab-externo glaucoma drainage implants, are associated with significant risk of postoperative complications. In contrast, MIGS procedures are performed through small incisions, often during cataract surgery using the same incision, and demonstrate excellent safety profiles. Optometrists will find themselves referring for and comanaging these procedures more frequently in the coming years; therefore, it is essential to be familiar with these options. Here, we cover a few of the more popular MIGS procedures.

Trabectome (Neomedix). FDA approved in 2004, this procedure is performed with a handheld device inserted into the anterior chamber through a small corneal incision. The Trabectome is positioned through the trabecular meshwork (TM) into Schlemm's canal, where it employs...
electro-cautery and aspiration functions to remove strips of tissue within the angle. This mechanism reduces resistance and aids aqueous drainage. Surgeons can use the Trabectome in isolation or during cataract surgery. It should be noted that adding the Trabectome to cataract surgery has not been reported to increase complications compared to cataract surgery alone.

Does it work? In 2015, researchers followed 82 treated eyes and found a 23% reduction from pre-treatment IOP at the two-year mark. Other studies estimate the IOP reduction can range from 16% to 44% during the one- to two-year period after treatment.

Although the postoperative medication schedule can vary by surgeon, patients are typically prescribed topical fluoroquinolones QID for one week and topical steroids QID tapered over the next one to two months. Additionally, pilocarpine 1% to 2% is applied BID to QID and tapered along with the steroid drops; this prevents peripheral anterior synechiae and concomitantly lowers IOP.

The Trabectome procedure comes with the risk for postoperative IOP spike and hyphema; however, these adverse events are rare and usually resolve quickly. Preoperative glaucoma medications should be continued immediately following the Trabectome procedure and adjusted appropriately as the IOP profile stabilizes in six to eight weeks. The preoperative glaucoma medications will not alter the probability of a post-op IOP spike, but will reduce its severity if it occurs.

Pros: The hallmark of MIGS is the high safety profiles. These procedures possess minimal risk of adverse effects and complications. They may be a safe means of reducing or eliminating glaucoma medications in many of our patients.

Cons: Since the IOP-reducing effect with MIGS is not as substantial as the more invasive glaucoma surgeries, such as trabeculectomy and ab-externo glaucoma drainage implants, MIGS is not indicated for patients with severe disease requiring very low (often in the single-digit) IOP profiles.
The heparin-coated titanium device measures 1mm by 0.3mm, making it the smallest FDA-approved device for implantation in the human body. During cataract surgery, the device is implanted into Schlemm’s canal where it remains permanently to improve aqueous outflow. The iStent is non-magnetic and thus compatible with magnetic resonance imaging (MRI).15

Does it work? In December 2015, researchers published exciting results after following 41 eyes implanted with a single iStent over three years. The average preoperative IOP for the subjects was 24.1mm Hg; on average, the patients were taking 1.8 glaucoma medications. At the three-year post-op interval, the average IOP was 14.9mm Hg with medications being eliminated in 74% of eyes.16

However, not all studies have found such optimistic results. A meta-analysis published in July 2015, which evaluated 37 studies reporting on 2,495 patients, concluded that cataract surgery alone reduced IOP by 4% while the combined phacoemulsification/iStent procedure reduced IOP by 9%.17

A second generation of the iStent is currently available in Europe. With a modified design, it comes pre-loaded with two iStents. When researchers conducted a meta-analysis of cases involving two iStents implanted in a single eye, they showed IOP was reduced by 27% from baseline.17 With the possibility of approval in the United States for the dual iStent design, there is hope for even greater IOP reduction for American glaucoma patients.

It is recommended to wait six to eight weeks before observing the new postoperative IOP state. Patients should therefore continue their glaucoma medications immediately after surgery, and clinicians can discontinue meds as the IOP improves. Similar to the postoperative course in cataract surgery, patients are prescribed a topical fluoroquinolone QID for seven days. Steroids starting at QID after surgery should be tapered over the next one to two months, and a topical NSAID QID should be given to supplement the steroid. Some clinicians may choose to taper the steroids more quickly than with the average postoperative cataract patient, as glaucoma patients can be more vulnerable to an IOP steroid response.

Cypass (Transcend Medical/Alcon). Cypass is the newest MIGS device to hit the market, having just recently achieved its FDA approval for use in conjunction with cataract surgery on July 29th, 2016. Unique from the other devices we have discussed, Cypass targets the suprachoroidal space to increase aqueous outflow.

The device is a tube shunt made of polyamide material that measures 6.35mm long and a mere 510 microns in diameter. It is carefully placed in the angle between the ciliary body and the sclera and terminates in the suprachoroidal space. The Cypass has openings at each end and micro-holes along its exterior to allow aqueous to flow into the shunt from the anterior chamber and exit the shunt into the suprachoroidal space.

Does it work? One study in particular is responsible for the device’s FDA approval. The COMPASS Trial, a multicenter, randomized clinical trial, was published in August 2016.18 COMPASS trial followed 505 subjects. One hundred and thirty-one subjects were randomized into the control group to receive phacoemulsification alone; the remaining 347 received phacoemulsification and Cypass placement. All subjects had primary open-angle glaucoma with entering unmedicated IOP ranging from 21mm Hg to 33mm Hg. The study found that mean IOP was reduced by 7.4mm Hg for the Cypass group and 5.4mm Hg for the control group at the two-year postoperative checkpoint.18 Although the difference between groups was only 2.0mm Hg, this was statistically significant enough for the study to conclude that Cypass provided long-term IOP reduction.18

At the two-year mark, medications were completely eliminated in 85% of the Cypass group, vs. 59% of the control.18 No severe or visually threatening events occurred throughout the study. The mild adverse events reported were iritis, corneal edema, hypotony and IOP elevation. However, these events were quite rare.

The COMPASS trial followed patients in the immediate post-op period at day one, week one, month one and month three. Patients were put on topical antibiotic drops for one week, topical NSAID drops for three weeks, and topical steroid drops, tapered over one month. During the clinical trial, patients were left off of their glaucoma medications postoperatively; they were restarted on glaucoma medications being eliminated in 85% of the Cypass group, vs. 59% of the control.18 No severe or visually threatening events occurred throughout the study. The mild adverse events reported were iritis, corneal edema, hypotony and IOP elevation. However, these events were quite rare.

The COMPASS trial followed patients in the immediate post-op period at day one, week one, month one and month three. Patients were put on topical antibiotic drops for one week, topical NSAID drops for three weeks, and topical steroid drops, tapered over one month. During the clinical trial, patients were left off of their glaucoma medications postoperatively; they were restarted on glaucoma.
medications on a case-by-case basis if IOP remained elevated for two consecutive visits.

- **Endoscopic cyclophotocoagulation** (ECP) aims to lower IOP by diminishing production of the aqueous by the epithelium of the ciliary processes. ECP is performed in conjunction with cataract surgery and uses a curved endoscopic laser probe. The surgeon applies laser energy to between 270 and 360 degrees of the ciliary processes, thereby reducing aqueous production.

  *Does it work?* The results vary between studies. In 2016, researchers followed a cohort of 91 eyes for one year and report an IOP reduction of 19% from baseline.\(^1\)

Two years prior, scientists studied 80 eyes over the course of two years and report an average IOP reduction of only 10%.\(^2\) Then, in 2015 another set of researchers published a retrospective study of 261 eyes with results three years postoperatively; their research shows a 14.5% average reduction in IOP.\(^3\)

In each of these studies, no increase exists in the rate of complications for combined ECP and phacoemulsification compared with phacoemulsification alone. Although the results do not always yield a dramatic reduction in IOP, the Early Manifest Glaucoma Trial shows the risk of progression decreases by 10% for every 1mm Hg reduction of IOP.\(^4\) With minimal risks involved, ECP is a procedure worth considering for glaucoma patients concomitantly in need of cataract surgery.

### Glaucoma Laser Procedures

These mainstays are familiar to practicing optometrists and remain viable in our long-term glaucoma management efforts.

- **Laser trabeculoplasty** using an argon laser (ALT) was first introduced in 1979. In this procedure, thermal energy is applied to the trabecular meshwork (TM), which induces contracture of the affected tissue. These focal alterations allow adjacent areas of the TM to expand, decreasing outflow resistance.\(^5\) Due to its limited repeatability over time, ALT’s role has not historically been found to be superior to medical therapy.

Since its approval in 2001, selective laser trabeculoplasty (SLT) has taken over as the preferred means of laser trabeculoplasty. SLT uses a frequency-doubled (532nm), Q-switched Nd:YAG laser to deliver laser pulses over 180
to 360 degrees of TM. These pulses selectively target the pigmented cells of the TM, which increase photolysis and cytokinetic activity, causing a healthy restructuring of the TM.

The limited structural damage seen in SLT allows for SLT to be repeated in the same eye if efficacy wanes or if further IOP reduction is needed. This has ultimately allowed clinicians to use this procedure as an initial treatment option in mild to moderate cases of primary and secondary open-angle glaucoma. Selective laser trabeculoplasty’s success rate—defined as 20% IOP reduction—was found to be between 55% and 82% in certain studies. SLT typically shows greater efficacy in patients who have higher pre-treatment IOP values.

Alpha-agonist drops, such as brimonidine or apraclonidine, are usually instilled immediately before and after SLT. IOP should be checked one hour after SLT is performed to rule out a transient postoperative spike, which has been reported to occur in 4.5% to 27% of patients. Patients should continue their glaucoma regimen; topical anti-inflammatory drops are typically applied for five to seven days.

In a recent study that compared artificial tears, prednisolone acetate and ketorolac, each dose daily QID following SLT, there was no significant difference in the IOP-lowering outcome or effect on failure rates of the procedure after one year. It is common for patients to report varying levels of postoperative discomfort, and up to 50% can present with postoperative anterior chamber reactions at the standard one-week followup. Therefore, the post-op management should focus not only on evaluating IOP, but also maximizing patient comfort with appropriate anti-inflammatory drops. Patients should remain on their glaucoma medications after the procedure and be re-evaluated at one and three months. It’s recommended to wait at least six to eight weeks before adjusting a patient’s glaucoma medications, as SLT reaches its steady-state at this time.

Laser peripheral iridotomy (LPI) is indicated for the treatment of (1) angle-closure glaucoma (ACG) associated with relative or absolute pupillary block and (2) prophylactic management of patients with narrow angle anterior chamber angles who may be at risk for ACG. This procedure uses an argon or Nd:YAG laser to create a full-thickness opening of the peripheral iris. This allows the aqueous humor to bypass its normal course through the pupil, and gives the fluid a direct pathway from the posterior chamber into the anterior chamber and ultimately to the trabecular meshwork. It effectively eliminates iridolenticular obstruction to aqueous flow.

IOP should always be checked approximately one hour after the applied laser to rule out a transient IOP spike. Patients should be given a topical steroid QID for five to seven days and should continue any glaucoma medications they are taking. A one-week postoperative visit should entail an IOP evaluation, a check for patency of the iridotomy with direct and retroillumination, and should address the presence of any post-procedural inflammation. Patients should be re-examined at one month to ensure stable IOP; a further evaluation of the anterior chamber angle should be conducted via gonioscopy and, if available, anterior segment optical coherence tomography (AS-OCT). If a dilated fundus exam is indicated, post-dilation IOP should be documented to help provide evidence of a properly functioning LPI.

Although the risk is low, there are potential adverse effects associated with laser iridotomies. A marked increase in IOP and mild iritis following the procedure may occur in up to 30% to 35% of cases. Intraocular inflammation is usually observed within the first 24 hours and resolves either spontaneously or with topical anti-inflammatory drops. Structural damage to the cornea and lens is possible, along with the later development of peripheral anterior synchiae and hyphema. Less common, but more visually threatening, complications such as retinal and choroidal detachments, focal retinal burns and macular edema can occur.

Summary

Although most of our glaucoma patients can be medically managed using topical IOP-lowering drugs,
MIGS and glaucoma laser procedures offer numerous benefits for those battling progressive disease, medication cost, difficulty with compliance or intolerance to eye drops. These procedures will become ever more popular for our mild to moderate stage glaucoma patients in the years to come. Familiarity with the key principles of appropriate comanagement will help you provide the best care to patients who have undergone these surgical techniques.

Dr. Van Alstine practices at the WJB Dorn VA Medical Center in Columbia, SC. He also serves as the president of the South Carolina chapter of the American Academy of Optometry.

Dr. Caruso practices within the Ralph H. Johnson VA Medical Center at the outpatient clinic in Myrtle Beach, SC.


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Controversies in Glaucoma Management

In optometry, it’s always debate season. This article looks at some hot topic issues and challenges conventional wisdom. By Bruce Onofrey, OD

The rational management of ocular disease is simple. First, know the disease. Get acquainted with the pathophysiology—or altered physiology—that leads to functional or structural loss. Second, know the patient. Consider the risk factors for, and consequences of, the disease, as well as the patient’s individual considerations for drug therapy. Finally, know the drug. Get familiar with the pharmacology of the many therapies available. Understand the mechanism of action, the drug’s indications, relative and absolute contraindications, proper dosages, dosage forms and proper warnings for counseling the patient, including the likelihood of adherence. This three-pronged approach ensures both safe and effective use of therapeutic agents. While all physicians can agree upon these principles, many of the specifics associated with glaucoma management remain up for debate. With today’s evolving understand-

Fig. 1. Correlation between retinal nerve fiber layer and ganglion cell loss and visual field loss in glaucoma. Preperimetric glaucoma demonstrates retinal nerve fiber layer and ganglion cell loss with normal visual fields.
worldwide, 2.8 million of whom are
As of 2010, 44.7 million people
elevated intraocular pressure (IOP).

The primary risk factor for devel-

carried optic neuropathy that, if left untreated,
can produce profound vision loss. The primary risk factor for de-
veloping damage from glaucoma is
intraocular pressure (IOP).
As of 2010, 44.7 million people
worldwide, 2.8 million of whom are
in the United States, are diagnosed
with POAG.1

When we apply our first rule of
disease management—know the
disease—to glaucoma, we unfortu-
nately come up short. In spite of the
work of many dedicated scientists over
the decades, understanding the cause
of POAG eludes us, as does a cure.

Without a complete understand-
ing of how glaucoma works, some
wonder if fundus images are as
valuable as we once believed and
what metrics we should use to target
medical treatment. Optometrists also
face challenges when individualizing
drug therapy in a way that maxi-
mizes efficacy and minimizes adverse
effects. Questions concerning wheth-
er unilateral drug trials are worth the
effort also persist.

This article explores the current
management of primary open-angle
glaucoma (POAG) and unpacks
some of the controversies within
optometry concerning glaucoma
management.

Controversy 1: How Do You
Target a Stealthy Disease?
POAG is a progressive anterior optic
neuropathy that, if left untreated,
can produce profound vision loss. The primary risk factor for de-
veloping damage from glaucoma is
elevated intraocular pressure (IOP).
As of 2010, 44.7 million people
worldwide, 2.8 million of whom are

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**Table 1. Major Glaucoma Clinical Studies and Select Results**

<table>
<thead>
<tr>
<th>STUDY</th>
<th>SIGNIFICANT RESULTS</th>
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<tbody>
<tr>
<td>Ocular Hypertensive Treatment Study(^1)</td>
<td>• Quantification of risk of conversion from ocular hypertension to glaucoma over a five-year period.</td>
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<tr>
<td></td>
<td>• Importance of central corneal thickness, IOP and vertical cup-to-disc ratio in assessing risk of conversion from ocular hypertension to POAG.</td>
</tr>
<tr>
<td>Early Manifest Glaucoma Trial(^2)</td>
<td>• Lowering IOP reduces risk of glaucoma progression.</td>
</tr>
<tr>
<td></td>
<td>• Visual field more sensitive than evaluation of disc changes in identifying progression.</td>
</tr>
<tr>
<td>Advanced Glaucoma Intervention Study(^3)</td>
<td>• A treated IOP of less than 18mm Hg, at all visits, significantly reduces the statistical risk of progression.</td>
</tr>
<tr>
<td>Diurnal Fluctuations in IOP(^4)</td>
<td>• Large diurnal fluctuations in IOP are an independent risk factor in POAG.</td>
</tr>
<tr>
<td>Collaborative Normal Tension Glaucoma Study(^5)</td>
<td>• Initial IOP must be lowered significantly (30%) to reduce risk of progression in normal tension glaucoma.</td>
</tr>
<tr>
<td>Los Angeles Latino Eye Study(^6)</td>
<td>• Large vertical cup-to-disc ratio greater than 0.6 is strongly associated with risk of POAG in this ethnic group.</td>
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</tbody>
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insight into which patients can be observed and which patients require treatment.2,4

A fascinating subset of POAG patients present with no clinical evidence of disease—at least, no evidence observable with standard observation.\(^5\) Their visual fields (VF) are normal, and clinical obser-
vation of the optic nerve may not show evidence of structural dam-
age. However, when their retinal nerve fiber layer (RNFL) or gangli-
on cells are evaluated using optical coherence tomography (OCT), loss of nerve fiber layer, ganglion cell damage or both, is observable. I
refer to this group as preperimetric glaucoma (Figure 1). At initial eval-
uation, it is impossible to determine if the tissue alteration is pro-
gressive. This form of glaucoma was predicted in a 1979 study indicat-
ing that structural loss (nerve fiber layer damage) precedes functional loss (VF defects), and nerve fiber layer loss occurs prior to detection of field defects.\(^6\)
Controversy 2: Do You Still Need Disc Photos and Visual Fields?
Quick answer: Yes and no. Not everyone will agree, but in my estimation, it’s a big “no” to disc photos. Why not, you may ask? Because high-resolution OCT has revolutionized our ability to detect structural nerve/RNFL damage and progression in a way that no individual, even a glaucoma specialist, could hope to detect by evaluating disc photos (Figure 2). Most clinicians think they are much better at evaluating optic nerves than they really are. This is particularly important in diagnosing preperimetric glaucoma. The goal of therapy is to minimize RNFL loss. Since functional changes present themselves after structural changes, waiting to treat until patients experience a VF defect sacrifices a significant amount of RNFL. Furthermore, research shows imaging the macular ganglion cell layer can help physicians detect POAG and its progression. Temporal hemifield defects of the ganglion cell layer can be detected quite early in the disease and represent a dependable sign of progression (Figure 1). Visual fields are a source of major frustration. They take time, are highly subjective and patients, for good reason, don’t like to do them. However, they are necessary in assessing the stage of disease and, if performed properly, are extremely sensitive in predicting disease progression. In the Early Manifest Glaucoma Trial 86% of progression was detected by VF changes, as opposed to 1% by disc changes detected using flicker choroscopy.

Controversy 3: Can Glaucoma Treatment Wait?
Glucomatous damage is currently classified under the broad categories of mild, moderate and severe. The purpose of these identifiers is to encourage consistency in documentation. It enables us to make a more accurate prognosis and inform patients of their relative risk of vision loss. Certainly, it should encourage compliance in patients who show evidence of progression. It can also justify the frequency of visits and tests performed. We generally consider glaucoma a slow process. However, patients in any of these broad categories have the potential to experience significant progression of their disease if they fail to use their medications as directed.

The degree of glaucomatous damage can be quantified by using optic nerve damage (structural) and VF loss (perimetric). The most accepted methods use perimetric testing. This recognizes that disability is most directly related to VF loss, whereas structural loss is generally used by clinicians to assess disease progression.

Perimetric staging of POAG.
Automated static perimetry is the benchmark for evaluating visual loss from POAG. It detects and quantifies damage, identifies the pattern loss associated specifically with POAG and helps to determine the success of therapy. Patients with perimetric glaucoma may be staged on their VF sensitivities as measured by standard automated perimetry (SAP) based on the number and depth of defective points, mean deviation (MD) or, most recently, the visual field index. While these parameters are all Humphrey perimeter based, other perimeter manufacturers have software that offers similar information.

An ideal method to classify functional damage in glaucoma should: be objective, reproducible and user-friendly; supply useful information on the characteristics of VF defects (shape, type, location and depth);
provide a classification which is consistent with structural damage data; be widely accepted and used; and able to monitor even relatively small changes in functional loss over time.

The most common criteria used to stage glaucoma is that of Hodapp, Parish and Anderson (HPA).13 The HPA classification system is a clinically useful method that considers two criteria: the overall extent of damage using the MD value and the number of defective points in the Humphrey Statpac-2 pattern deviation probability map of the 24-2, SITA-standard test. In addition, the method takes into consideration the proximity of the defect or defects to fixation (Table 2).

Despite its popularity, this classification has some disadvantages. Staging the VF defect requires time-consuming analysis of every visual field test, reducing its day-to-day clinical usefulness—not to mention it provides no information about the location and depth of the defects.13

Simplified optic nerve/perimetric staging. The American Academy of Optometry and the American Glaucoma Society work group recently developed a new glaucoma staging system to evaluate and test the accuracy of the severity levels, using real-world cases (Table 3).12 This system’s strengths are its simplicity and incorporation of both structural and perimetric data. The most important component is location of the VF defect. The system is particularly biased toward VF defects that approach fixation. One factor it does not take into account is superior vs. inferior VF loss. Studies show inferior hemifield defects are more disabling than superior defects due to the effect inferior field loss has on a person’s ability to read.15

Table 3. AAO Glaucoma Severity Staging Descriptions

<table>
<thead>
<tr>
<th>STAGE</th>
<th>DESCRIPTION</th>
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<tbody>
<tr>
<td>Mild/early</td>
<td>Structural optic nerve changes consistent with glaucoma with no evidence of visual field changes with standard automated perimetry (preperimetric glaucoma).</td>
</tr>
<tr>
<td>Moderate</td>
<td>Optic nerve changes consistent with glaucoma and glaucomatous visual field changes in one hemifield and not within five degrees of fixation.</td>
</tr>
<tr>
<td>Severe</td>
<td>Optic nerve changes consistent with glaucoma and glaucomatous visual field changes in both hemifields or loss within five degrees of fixation in at least one hemifield, or both.</td>
</tr>
<tr>
<td>Indeterminate</td>
<td>Field not done, or patient unable to perform visual field testing.</td>
</tr>
<tr>
<td>Unspecified</td>
<td>Stage not recorded in chart.</td>
</tr>
</tbody>
</table>

Figs 2a and 2b. Above, optic nerve images can help identify glaucomatous damage, but the high-resolution OCT images of the same patient below provide a greater level of detail that can help identify structural changes resulting from glaucoma earlier.
Controversy 4: Should You Set IOP Goals?

The ultimate target IOP is the one that prevents development or progression of the disease; however, our initial treatment goal is to lower IOP to a number or by a percentage that significantly lowers the statistical risk of progression. The Advanced Glaucoma Intervention Study demonstrated that a treated IOP below 18mm Hg lowered the risk of progression to just more than 20%, and an additional decrease in IOP further reduces the risk of glaucoma.4 The Early Manifest Glaucoma Trial determined that the risk of progression of POAG dropped by 10% for every 1mm Hg of IOP reduction, and a 25% drop in IOP halved the risk of progression.10 Any clinician involved in the management of glaucoma must set IOP targets that are, in part, based on these important evidence-based studies.

Controversy 5: Which Targeted Medical Management Should You Use?

Reduction of IOP remains the cornerstone of the management of POAG. Commonly used topical IOP-lowering drugs can be divided into four major classes: prostaglandin analogs (PGAs), beta-blockers, alpha-agonists and carbonic anhydrase inhibitors (CAIs). Also available are fixed combinations of topical IOP-lowering drugs. In the United States, we typically see three drug combinations: one that includes a beta-blocker and a CAI, one that includes a beta-blocker with an alpha-agonist and an alpha-agonist with a CAI. The FDA has not approved any prostaglandin combinations. A benefit of fixed-combination formulations is the intent to maximize efficacy and improve adherence.

The introduction of PGAs 20 years ago shifted the management of glaucoma; at the time, only filtering surgery could reliably produce the same degree of IOP lowering. PGAs quickly became the drugs of choice for glaucoma, while the use of other medications, as well as surgery, dropped dramatically.16

PGAs are the most popular first-line agents for glaucoma treatment for good reason. In addition to their efficacy, PGAs are notable for the paucity of significant systemic (cardiovascular or pulmonary) side effects associated with their use. Mild ocular side effects, however, are not uncommon, including reversible darkening of the iris and periocular skin, growth of lashes, stinging and conjunctival hyperemia. Although most of these effects are cosmetic, some patients find them worrisome or unacceptable, making it important to counsel patients about the potential ocular effects of PGAs beforehand.17

Beta-blockers were once the mainstay medical treatment for glaucoma. These agents lower IOP by decreasing aqueous production; the effect—at least a 25% pressure reduction—occurs primarily during the day.18 Although highly effective and generally well tolerated, in susceptible individuals beta-blockers can produce severe and sometimes life-threatening cardiovascular and respiratory side effects, including bradycardia, arrhythmia, heart block and bronchiolar constriction.19 Adverse central nervous system effects are also common, ranging from weakness and depression to hallucinations.18 In patients with diabetes, use of beta-blockers can mask hypoglycemic signs and symptoms, sometimes resulting in dangerously low blood sugar. Additionally, beta-blockers have the potential to raise serum triglycerides and thereby increase the risk of cardiovascular disease.18 If used in patients who are highly allergic to substances such as peanuts or insect venom, beta-blockers can reduce the efficacy of injected epinephrine.11

Clinically, it is vital to identify patients who may be susceptible to these potential dangers. Contradictions to beta-blocker use include asthma, chronic obstructive pulmonary disease, bradycardia and congestive heart failure. A careful clinical history is often helpful in recognizing patients at risk. When a topical beta-blocker is prescribed, patients should be told of its potential systemic side effects and instructed to measure blood pressure and pulse regularly. It is not uncommon to prescribe beta-blockers once daily in the morning to maximize efficacy and avoid issues such as nocturnal hypotensive events.19

In spite of systemic side effects, beta-blocker use has remained strong, possibly due to cost, predictability of side effects and excellent efficacy. Additionally, the drug works extremely well in combination with all other agents.

Selective alpha-agonists, such as alpha-2 agonists, lower IOP by about 20% to 25%, although the dosing schedule for monotherapy—three times daily—is inconvenient. These agents can, however, be used in combination with other drugs, allowing for twice daily dosing. Alpha-2 agonists are generally well tolerated but may stimulate alpha-2 receptors of the central nervous system and produce adverse systemic reactions such as low blood pressure and orthostatic hypotension. Alpha-2 agonists can also cause allergic responses at rates ranging from 12% to 25%.20

The two selective alpha-agonists available today are apraclonidine and brimonidine. Apraclonidine, the first relatively selective alpha-2 ago-
nont available, was initially used to treat open-angle glaucoma. Allergy and diminution of therapeutic effect with repeated use (tachyphylaxis) have limited its usefulness to short-term applications, such as preventing pressure spikes after anterior segment laser procedures. Brimonidine, which is more alpha-2 selective than apraclonidine, is more appropriate for chronic therapy.21

Carbonic anhydride inhibitors reduce IOP by about 20%—less IOP-lowering efficacy than PGAs.22 Because they reduce IOP by decreasing aqueous production, these sulfonamide agents are often used adjunctively with PGAs, which lower IOP by increasing non-trabecular aqueous outflow. Like PGAs, topical CAIs have no effect on blood pressure, heart rate or pulmonary function.

Because they are sulfonamides, CAIs can cause allergic reactions in sensitive patients. Oral CAIs, such as acetazolamide, are also associated with a number of serious systemic side effects, including metabolic acidosis, renal calculus formation, hematologic abnormalities and sickle cell crisis.23 Since topical CAIs (dorzolamide and brinzolamide) have become available, the use of oral CAIs is generally limited to angle-closure glaucoma and secondary forms of glaucoma such as uveitic glaucoma.

There are two key factors in selecting any medication: efficacy and safety. The PGAs are today’s preferred choice for initial therapy owing to their greater IOP-lowering efficacy and systemic safety. Before initiating treatment, clinicians should obtain a thorough history and determine whether the drug of choice is safe for that particular patient. In the case of a PGA, the side effects are, as noted, mainly local and cosmetic. But when an alternative or a second agent is warranted, systemic risks such as cardiovascular or pulmonary disease or allergy become important considerations.

A review of clinical evidence and expert opinions suggests that a PGA coupled with a topical CAI may be the best combination to lower IOP.24,25 The pair synergistically reduces IOP with minimal systemic risk. My own primary choice is a PGA followed by a topical CAI. After that, I add either an alpha-agonist or a beta-blocker. If three medications cannot bring the patient to target IOP, the patient should be referred for laser or surgical intervention.26 Because IOP reduction from monotherapy and multi-drug therapy can vary greatly among individuals, some clinicians prefer to use individual monotherapy regimens to detect the most effective treatment combination for their patients.

Patient compliance is critical to the success of chronic medical therapy for glaucoma. Patients must understand that glaucoma is a lifetime disease, and the success of therapy requires commitment to the medication regimen and continued assessment. In addition to teaching the importance of adherence, clinicians can help patients by selecting agents that are safe and comfortable to use on a regular basis. One study demonstrates that, over a period of approximately 18 months, the compliance level of glaucoma medications dropped by approximately 60% to 80%.27 Therapy with latanoprost had the least reduction in compliance; the use of all other classes dropping by 80%.27 The most significant find was that the most dramatic decline in compliance occurs approximately six to seven months after initiating therapy.27 Most glaucoma eye drops, especially preserved ones, have a deleterious effect on the ocular surface that can exacerbate dry eye signs and symptoms. Treating preexisting dry eye and other ocular surface conditions may help improve tolerability and reduce noncompliance.

Controversy 6: Are Unilateral Drug Trials Worth the Effort?

Murray Fingeret, OD, noted in 2009 the positive aspects of unilateral drug trials, stating that “IOP, while often different between the two eyes, will rise and fall over the day to a similar degree. Also, the response to a medication should be similar in both eyes. Since non-responder rates vary from 8% to 25% depending on the class of medication, a monocular trial is one way to ensure the medication is effective, as well as determine if side effects are occurring.”28 This point of view is countered by a 2014 study that suggests the pressure lowering effect crosses over to the other eye with a large degree of variability, thereby underestimating the actual efficacy of the drug. This lack of reliability and accurate predictability put the value of monocural drug trials in doubt.29

New medications. Bausch + Lomb’s prostaglandin analog latanoprostene bunod recently completed phase III trials. The results indicate that the efficacy was “non-inferior” to timolol with an IOP reduction range of 7.5mm Hg to 9.1mm Hg. The study included 800 patients and was conducted over a period of 12 weeks. The suggested benefit of this new agent is a dual action. Latanoprostene bunod decreases IOP, like other prostaglandin analogs, by increasing uveoscleral outflow. It’s designed to increase optic nerve blood flow via nitric oxide donation.30 Rho-kinase inhibitors are another option currently under development.
These pharmacological agents are designed to target the cells of the trabecular meshwork to facilitate aqueous outflow. Their mechanism inhibits Rho GTPase proteins—in particular, RhoA, which may be associated with glaucoma’s pathophysiology since RhoA is significantly elevated in glaucomatous optic nerve heads.11,32

Generics, sampling and assistance programs. As a pharmacist, I am commonly asked, “How can the pharmacist dispense a generic when I wrote the prescription for the brand-name product?” The simple answer is, they can’t. Writing for a branded product does not ensure that a generic will not be dispensed. You must check the box or write on the prescription “no generic substitution allowed.” When this statement is included, the pharmacist cannot legally dispense a generic version of your prescription.

The real reason substitution occurs is based on health plan limits and the high cost of branded products. Some plans will only pay for the generic, if available. Insurance companies may also only pay for the generic of a class of drug. For example, if you write an Rx for Travatan Z (travoprost, Alcon), the patient’s drug plan may only cover generic Xalatan (latanoprost). The patient then is faced with accepting the covered generic or paying out-of-pocket for the non-covered brand product. The final scenario is the patient who has no drug coverage and is asked to pay, sometimes hundreds of dollars, for the branded product, something they may be unwilling or unable to afford. This certainly can affect compliance. The patient may not fill the prescription, or even worse, may not use the drug as often as directed. When it comes to generics, a patient cannot use a drug they cannot afford. I always start with a generic and upgrade to a brand only if the generic fails to meet my treatment goals.

Drug assistance programs are something every clinician should consider for patients who have limited resources. Most of the major companies have a fairly direct process of evaluating the financial eligibility for these programs. The patient must be willing and able to submit proof of their income as well as possess proper identification—usually a social security card. Once approved, the medication is usually shipped to the prescribing clinician to be dispensed to the patient.

Drug companies hate sampling, period, and I agree up to a point. I don’t sample acute care medications such as antibiotics or steroids. However, when it comes to chronic medications for glaucoma or allergy, for example, I thoroughly believe in a sample trial to evaluate the efficacy of the drug. I generally give a one-month supply and write an Rx if, after the first follow up, the drug has shown adequate efficacy without evidence of ocular or systemic side effects.

Invest in the Future

The modern, rational management of glaucoma requires a major investment on our part. We must invest financially in new technologies, which is not cheap. SD-OCT, SAP, Goldmann tonometry, pachymetry, gonioscopy and posterior segment lenses with a good slit lamp are basic tools needed to ethically and professionally manage glaucoma.

Aside from the monetary investment, we must also invest time in reviewing and understanding the pharmacology of treatment agents to safely and effectively prescribe them to our glaucoma patients. Finally, we must be familiar with the major clinical studies that are necessary to guide our clinical decisions.

Dr. O’Nofrey is a clinical professor and the executive director of continuing education programs at the University of Houston.

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1. Preperimetric glaucoma is characterized by structural damage to the optic nerve and a normal:
   a. Visual field.
   b. Ganglion cell layer.
   c. Nerve fiber layer.
   d. IOP.

2. Rational management of disease requires an understanding of all these except:
   a. The drug.
   b. The patient.
   c. The disease.
   d. The patient’s hobbies.

3. According to the HPA staging of glaucoma, early visual field defects are characterized by all of the following except:
   a. MD less than 12dB.
   b. Central 5° has a sensitivity of at least 15dBs.
   c. Fewer than 25% of points are depressed below 5% level on the pattern deviation plot.
   d. Fewer than 10 points are depressed below the 1% level on the pattern deviation plot.

4. The Ocular Hypertension Treatment Study determined that all of the following factors were important in predicting that the patient would convert from ocular hypertension to glaucoma except:
   a. Family history of glaucoma.
   b. Corneal thickness.
   c. IOP.
   d. Vertical cup-to-disc ratio.

5. Latino patients with cup-to-disc ratios greater than ___ vertically have a greater than 90% chance of having POAG. (a. 0.1.  b. 0.6.  c. 0.4.  d. None of the above.)

6. Perimetry is useful for all of the following reasons except:
   a. To determine the degree of functional loss.
   b. As a subjective test.
   c. To determine severity classification.
   d. To demonstrate loss in an effort to help motivate patient compliance.

7. Monocular glaucoma drug trials may lack reliability due to the drug’s effect crossing over to the other eye, causing ___ of the drug’s impact.
   a. Overestimation.
   b. No impact.
   c. Underestimation.
   d. A false sense of security.

8. The major problem with interpretation of disc photos is:
   a. Underestimation of cup size.
   b. Overestimation of cup size.
   c. Variability among observers.
   d. All of the above.

9. The AAO system of the staging of glaucoma severity is biased towards scotomas that:
   a. Touch on fixation.
   b. Are in the superior hemifield.
   c. Are in the inferior hemifield.
   d. Are in the temporal visual field.

10. Beta blockers have maintained their popularity due to all of the following except:
    a. 25% efficacy.
    b. Very low risk of systemic side effects.
    c. Low cost generics.
    d. Ease of use.

11. Which statement regarding prostaglandin analogs is false?
    a. They have a very high efficacy.
    b. They have a significant effect on heart rate and blood pressure.
    c. They can produce irreversible darkening of the iris.
    d. They work well in combination with all other classes of glaucoma medications.

12. The new prostaglandin analog latanoprostene bunod increases extra-trabecular outflow and:
    a. Decreases aqueous production by parasympathetic activity.
    b. Has intrinsic sympathomimetic activity.
    c. They can produce irreversible darkening of the iris.
    d. Decreases aqueous production by parasympatholytic activity.

13. Which patients should avoid oral carbonic anhydrase inhibitors?
    a. Sulfonamide sensitive patients.
    b. Sulfonamide sensitive patients.
    c. Patients prone to kidney stones.
    d. All of the above.
Examination Answer Sheet
Valid for credit through October 1, 2019

14. The most common use of oral acetazolamide today is for:
a. Acute angle-closure glaucoma.
b. POAG.
c. Normal tension glaucoma.
d. In sickle cell patients with POAG.

15. Apraclonidine use in treating POAG has been largely discontinued due to a risk of:
a. Drug hypersensitivity.
b. Drug toxicity.
c. Tachyphylaxis.
d. a and c.

16. Which drug combination has very few systemic side-effects?
a. PGA and timolol.
b. PGA and CAI.
c. CAI and beta blocker.
d. Beta-blocker and alpha agonist.

17. Which agent can produce bradycardia and heart block?
a. Brimodine.
b. Brimtoprost.
c. Timolol.
d. Brinzolamide.

18. Which is classified as a sulfonamide?
a. Brinzolamide.
b. Dorzolamide.
c. Apraclonidine.
d. a and b.

19. One study of glaucoma drug compliance found that after six months of treatment, the prescribed use of the drugs:
a. Remained stable.
b. Improved dramatically.
c. Fell sharply for all agents.
d. Fell only for latanoprost.

20. The pharmacist can only substitute a generic drug for a brand drug:
a. If the patient asks them to.
b. If the doctor gives their permission.
c. When insurers will only pay for generics.
d. At their own discretion.

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Demodex management may be dismissed by some as a mere “fad” in optometric practice, but with more and more patients reporting refractory dry eye symptoms, it’s time to consider it an actual ocular concern that can impact any patient, especially those older than 50. Demodex is typically not difficult to manage, but its treatment in optometric offices remains uncommon. The majority of Demodex patients have mild to moderate symptoms that include itching and burning of the eyes and lids, foreign body sensation and fluctuating blurry vision. These symptoms can be swiftly managed with artificial tears, but if Demodex is the culprit, artificial tears will offer only temporary relief. As symptoms are similar to ocular surface disease, it is easy to assume dry eye syndrome and prescribe over-thecounter artificial tears rather than evaluate for the contributing factor, Demodex.

This article urges doctors to consider this diagnosis while explaining how to differentiate between Demodex and other causes of dry eye symptoms.

Meet the Mites
More than 100 species of Demodex mites have been identified. Demodex folliculorum and Demodex brevis are the two main species that inhabit the human skin. D. folliculorum typically range from 0.3mm to 0.4mm long and tend to reside within the hair follicles while D. brevis mites are about half the length (0.186mm) and reside within the sebaceous and meibomian glands.

Both parasites are half the diameter of a grain of table salt and worm-shaped with four legs. D. folliculorum consume and damage epithelial cells at the hair follicle, resulting in a weakened lash root and the eventual loosening and

Cuffing, seen here, is a classic symptom of Demodex overpopulation, which can lead to dry eye symptoms that artificial tears simply can’t address.
## The Pros and Cons of Demodex Therapies

<table>
<thead>
<tr>
<th>Product</th>
<th>Pros</th>
<th>Cons</th>
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| **Avenova** (Novabay) | • No stinging sensation  
• Proven effective against Demodex despite not containing active tea tree oil  
• Three year shelf life | • Only available through prescription  
• Not covered by all insurers |
| **BlephEx** (RySurg) | • Disposable, rotating microspunge  
• Mechanical removal of lid debris via a handheld device  
• Performed in clinic by OD or staff | • Requires patient compliance  
• Frequent return visits to office  
• Patients currently need to pay out of pocket |
| **Cliradex, Cliradex Complete, (Bio-Tissue)** | • Comes with a microexfoliator for in-office use for cases with severe debris to allow better penetration of T4O  
• High concentration Cliradex Advanced Care gel (50% T4O) in Cliradex Complete Kit for in-office use allows more effective initial dose  
• Lower concentration (10% T4O) for home maintenance between visits  
• Lid scrubs available OTC for mild to moderate cases | • Strong menthol-like sensation (can be described as a stinging sensation) |
| **Eye Eco Tea Tree Oil Eyelid & Facial Cleanser (Eye Eco)** | • 2-in-1 face and lid wash  
• An effective make-up remover  
• Foam dispensing bottle  
• Available OTC | • Strong menthol-like sensation (can be described as a stinging sensation)  
• Removal of accumulated debris depends on patient’s attention to lids while applying |
| **Ocusoft Lid Scrub (Ocusoft)** | • Available in lid scrub pads or a foam to be used with a cotton applicator  
• Contains active tea tree oil ingredients  
• Removes debris and excessive oils  
• Available OTC | • Strong menthol-like sensation (can be described as a stinging sensation) |
| **SteriLid (Theratears)** | • Contains tea tree oil ingredients  
• Removes accumulated debris and excessive oils from lid margin  
• Available OTC | • Detergent-based product |
| **Blephadex lid scrubs (Lunovus)** | • Available in lid scrub pads or a foam to be used with a cotton applicator  
• Contains tea tree oil ingredients  
• Removes accumulated debris and excessive oils from lid margin  
• Available OTC | • Strong menthol-like sensation (can be described as a stinging sensation) |
| **Baby Shampoo** | • Cost efficient  
• Will address any bacterial component of blepharitis  
• No stinging sensation | • Typically no tea tree oil |
misdirection of the lash.\textsuperscript{3,5,7} Their appendages create microabrasions that cause epithelial hyperplasia and reactive hyperkeratinization, which we see as cylindrical dandruff. \textit{D. brevis}, on the other hand, tend to burrow deep into the sebaceous glands, physically blocking the orifice.\textsuperscript{5,7} In addition, its chitinous exoskeleton acts as a foreign body, inducing granulomatous reactions that can lead to hordeola or chalazia.\textsuperscript{5,7} Due to the \textit{D. brevis}' proximity to the ocular surface, it is more likely the cause of refractory corneal lesions than \textit{D. folliculorum}.\textsuperscript{6}

Under scrutiny of a slit lamp, \textit{D. folliculorum} can sometimes be seen orienting themselves face down toward the follicle bed with a small portion of their tail protruding from the opening. Though more difficult, rotating the lash (without epilation) has also been effective in encouraging mites to travel to the follicle opening for easier observation behind a slit lamp.\textsuperscript{8}

For those with easy access to a microscope, the presence of \textit{Demodex} can also be enhanced with the addition of fluorescein or alcohol on the glass slide; they cause the cylindrical dandruff to become semitransparent to reveal mites hiding within the debris.\textsuperscript{3} Due to their photosensitivity, \textit{Demodex} tend to avoid the bright lights and can be difficult to observe under plain biomicroscopy. For the same reason, most mating and movement occurs at night. Their lifespan is about one to two weeks for adults.\textsuperscript{3,6}

Patients may note increased lid irritation first thing in the morning as a result of the increased activity at night.

**Demodex and Rosacea**

\textit{Demodex} has been implicated as a causative agent in rosacea since 1932.\textsuperscript{6} The affliction affects about 16 million Americans, mostly those older than age 30 who have fair skin.\textsuperscript{15} Those affected present with erythema, flushing and transient papules along the cheeks, chin, nose and central forehead.\textsuperscript{15,21} Considered an inflammatory condition, patients suffering from rosacea have increased levels of interleukin-1a and -1b, as well as a greater activity of metalloproteinases (MMP-9 and MMP-8) in the tear film.\textsuperscript{15}

These markers support the benefits of doxycycline, which acts to decrease both MMP-8 and MMP-9 expression.\textsuperscript{15} There is a large discrepancy (6% to 72%) in the prevalence of ocular involvement in those with rosacea.\textsuperscript{15,21} Investigators have found \textit{Demodex} presence in 60% of patients clinically and up to 80% when detected via skin biopsy.\textsuperscript{21} Since not all rosacea patients presented with \textit{Demodex} infestation, researchers believe these mites are aggravators or symptomatic, but not the causative agent. Rosacea patients had an average of 12.8 mites per square centimeter of skin, significantly greater than the 0.7 mites per square centimeter in nonrosacea subjects.\textsuperscript{3}

Increased UV exposure and warmer seasons are correlated with increased rosacea flare-ups.\textsuperscript{6,12,13} In addition, \textit{Demodex} colonization increases in the spring and summer and, as a result, UV treatments may be contraindicated as a treatment option against \textit{Demodex} mites.\textsuperscript{6} Other triggers for rosacea include: spicy foods, alcohol consumption, extreme temperatures, physical exercise, emotional distress and menopause.\textsuperscript{15}

**Invasion of the Body Snackers**

Because these ectoparasites are susceptible to desiccation, they require a host to survive.\textsuperscript{8} In addition, the mite population increases with age.\textsuperscript{1,3,4,6,7,9} An overpopulation of mites can lead to an imbalance of tear cytokine levels, particularly an influx of interleukin 17, which is proinflammatory, leading to blepharitis.\textsuperscript{8,10} A recent study found that 84% of those older than age 60 have \textit{Demodex}, and 100% of those older than 70 are infested.\textsuperscript{4} Several studies found that the average healthy patient will likely have a small population of \textit{Demodex}, but also confirmed a marked increase (two to six mites infesting each follicle) in mite population in patients older than 50.\textsuperscript{4} One study found

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\textsuperscript{3} Photo credit: Hunter, O.D.
an average count of 12.9 mites per patient seen with cylindrical dandruff, whereas those without had an average of 0.35 mites—that’s a considerable difference.7 Individuals with compromised immune systems, such as those with poor systemic health and those taking immunosuppressive agents, have a higher level of Demodex, although contradicting research postulates that mites may thrive better in healthy, normal tears as opposed to tear deficient states.3

Interestingly, a 2014 study based the presence of Demodex on amplification of Demodex DNA rather than visual observation.11 They found that, although only 14% of subjects older than 18 years visually presented with Demodex, they were able to detect Demodex 16D rDNA in up to 70% of those subjects.11 We can presume that Demodex may actually just be part of the natural human bioflora and does not become a problem until they are allowed to overpopulate and tip the natural equilibrium of the skin flora.

**Patient Presentation**

In addition to dry eye symptoms, Demodex patients often complain of irritation along the lid margin. Blepharitis can be caused by bacterial or parasitic etiologies. Numerous studies, including one meta-analysis, have found a strong correlation between bacterial blepharitis and Demodex blepharitis.12-14 Demodex mites act as vectors, carrying bacteria such as Staphylococci and Streptococci as they travel between lashes.6,7,10 The superantigens produced by these bacteria are implicated in anterior blepharitis and the induction of stubborn skin conditions such as rosacea, pityriasis folliculorum, pustular folliculitis, perioral granulomatous dermatitis and permanently hyperpigmented patches on the skin.1,4-6,10
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Cylindrical cuffing along the base of lashes is a pathognomonic finding for *Demodex* infestation. Researchers believe this conical buildup is the combination of the saprophytes’ chitinous exoskeleton, in addition to other keratin and lipid debris naturally sloughed from the skin’s epithelial layer. If the materials are not removed from the lid margin, irritation results in lid inflammation, demodicosis (blocked follicles and follicular distention) and madarosis.

Since the presentation of cylindrical dandruff indicates a significant increase in mites along the lash line, proper lid hygiene and stabilization of the *Demodex* population should be initiated upon the first signs of cuffing to prevent additional sequelae. If the patient has already been using lid scrubs in treating misdiagnosed dry eye syndrome and bacterial blepharitis, it's fairly common to not observe any conical cuffing. However, even after proper lid hygiene and complete resolution of lash cuffing, mites can still be hidden within the hair follicle. In fact, one study found that *Demodex* can be left in the follicle even upon epilation of lashes, which would greatly underestimate the true severity of parasitic infestation under microscopic observation.

Still, a strong indicator of *Demodex* infestation is past unresponsiveness to conventional dry eye treatment. As most dry eye cases are typically due to meibomian gland dysfunction (MGD), careful observation of the eyelid and tear film under slit lamp examination are the key to differentiating between *Demodex* and decreased meibum secretion. Ineffective treatments for removing *Demodex* include ATs, cyclosporine, antihistamines, doxycycline, lid hygiene and baby shampoo—though these treatments may be added to the management plan against bacterial agents that are also contributing to lid inflammation.

Delayed proper management can also lead to the progression of more chronic symptoms, such as trichiasis, MGD, chalazia, conjunctivitis and corneal pathologies such as corneal neovascularization, marginal corneal infiltration, phlyctenule-like lesions, superficial corneal opacities and nodular scars.
Treatments
By understanding the natural life cycle of the Demodex mites, treatments aimed at eradicating the infestation will be more efficient. Because these ectoparasites are more nocturnal, it is important to limit their ability to reproduce and migrate when they are most active. Ophthalmic ointments, such as erythromycin or Tobradex (tobramycin-dexamethasone, Alcon), along the lid margin, therefore, have been used as a solution to keep Demodex from reaching the surface to reproduce or migrate at night. In addition, practitioners often advise patients to start washing their bed-sheets and pillowcases regularly in hopes of preventing reinoculation. In severe cases, patients may even need to consider replacing their pillows altogether.

Combined with a tea tree oil therapy along the lash line, the treatment not only hinders proliferation of mites, but also actively eradicates Demodex adults, larvae and eggs.16,18,19

Tea tree oil has been a widely accepted treatment for mite infestation; it offers anti-inflammatory, antimicrobial and antifungal properties to effectively manage not only parasitic but also bacterial blepharitis.16,18,19 If Demodex truly only acts as a mode for bacterial migration, tea tree oil should still be considered to directly destroy the symbiotic bacteria and parasitic relationship.5,6 Investigators found that D. folliculorum was resistant to not only antiseptic treatments, including 75% alcohol and 10% povidone-iodine, but also some antimicrobials such as erythromycin and metronidazole.4,20

Tea tree oil, specifically the active ingredient terpinen-4-ol (T4O), was found to be the most effective in both cleaning cylindrical debris from the roots of lashes and stimulating the migration of hidden mites to the surface for eradication.4,20 The current option with the highest concentration of terpinen-4-ol is Cliradex (Bio-Tissue).4,20 In mild cases, at-home treatments such as Cliradex, Steri-Lid (Thera Tears), Blephadex (Lunovus) and Ocusoft scrubs BID are sufficient to decrease the Demodex population. These products are available over the counter.

For more stubborn cases, Cliradex Complete (Bio-Tissue) offers a higher concentration (50% T4O) solution for in-office use two to three times in 10-minute intervals. The patient then uses the lower concentration wipes (10% T4O) twice a day at home.9 If you note considerable debris and cylindrical dandruff, it may be prudent to mechanically remove the debris prior to treating with tea tree oil. Although the Cliradex Complete pack comes with a microblepharoexfoliator, the BlephEx (Rys Surg) handheld device may provide a more thorough and efficient removal process in severe cases.

Newer products, such as Avenova (Novabay), for which the main ingredient is Neutroxx (pure 0.01% HOCl) rather than tea tree oil, also effectively decrease symptoms, according to a company study.22

Simply put, the diagnosis of Demodex associated eye disease is clinical and relies on observation and the correct interpretation of both ocular surface and accompanying skin manifestations. Though the focus of optometrists is primarily on the health of the eyes and adnexa, it is becoming more evident that we should also be prudent in taking a step back to observe and learn more about the patient’s overall condition to collect all pertinent information for proper diagnosis and treatment. ■

Dr. Roan is a staff optometrist at the Pacific Cataract and Laser Institute in western Washington.

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REVIEW OF OPTOMETRY OCTOBER 15, 2016

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My associate contracted a bad adenoviral infection from a patient last week. He’s into the second week with multiple subepithelial infiltrates (SEIs) over the visual axis and is symptomatic with blur and discomfort. He also has a conjunctival membrane. How might we have avoided this response and, now that he has it, what treatment do you recommend?

“The somewhat flippant answer,” says Daniel G. Fuller, OD, of Southern College of Optometry, “is by practicing safety precautions and appropriate infection control.” Aaron Bronner, OD, of Pacific Cataract and Laser Institute agrees, stating that, “clean examination, proper hand washing and disinfection protocol are the keystones for preventing clinic-based transmission.”

Adenoviral Dominos

When doctor becomes patient, how do we mitigate infectious interactions?

Edited by Joseph P. Shovlin, OD

A Pound of Cure

With EKC, you can treat with supportive therapy (i.e., cool compresses, preservative-free tears and time), or “you could employ one of several off-label approaches in hopes that it may hasten resolution of the infection and reduce late sequelae,” says Dr. Bronner.

Adenoviruses are medium-sized (90nm to 100nm), non-enveloped icosahedral viruses with double-stranded DNA, notes Dr. Fuller. “There are more than 50 known serotypes responsible for causing conjunctivitis and upper respiratory, gastrointestinal, cystic and neurological illness, while only a limited number have proven responsible for EKC, pharyngoconjunctival fever and acute hemorrhagic conjunctivitis.”

Serotypes 8, 19 and 37 are more prevalent in severe presentations of EKC. EKC presents in adults as watery discharge, hyperemia, chemosis, follicles and ipsilateral lymphadenopathy. The more severe forms, says Dr. Fuller, “include SEIs (50%) with concomitant decrease in vision, petechial hemorrhages, pseudomembrane formation and symblepharon with true membrane formation. The infection is considered biphasic with symptoms of inflammation appearing seven to 10 days after infection and a usual course of two to three weeks,” he explains.

There is a dearth of FDA-approved interventions, notes Dr. Fuller. “Treatment is supportive for mild forms, and infection control is critical.” Multiple off-label interventions have been tried with limited success, including steroids, antiseptics, antivirals, immunosuppressants and interferon.

Dr. Bronner credits Mark Maraman, OD, for suggesting hypochlorous acid may be an avenue worth considering. Dr. Bronner explains that a group of 14 cases treated with ultrapure hypochlorous acid (0.008%) QID had rapid clearing of signs and symptoms. Of all the off-label treatments for EKC, “hypochlorous acid has the least research,” he notes, “though I feel it may be a compelling option because, as with povidone-iodine (PI), hypochlorous acid is a potent disinfectant and, as with PI, would only theoretically be effective in the...
extracellular phase. But hypochlorous acid is available by prescription and can be dosed at home.”

Both doctors caution against the use of steroids without weighing the risks against the benefits. Several studies show that routine use of steroids for symmetric relief should be avoided, as they can prolong the minimizing spread to the other eye and indeed a severe case, and manage:

“Since your associate has multiple SEIs, any steroid that effectively penetrates the corneal epithelium is a good way to diminish the lesion,” Dr. Bronner suggests, citing prednisolone acetate 1%, diflupred-nate and loteprednol etabonate as options. Start steroid treatment “at the first sign of SEIs rather than when multiple lesions are present and the visual axis is heavily involved,” says Dr. Bronner, “to reduce their downstream severity and hasten resolution.”

As a doctor, he should refrain from patient care for 10 to 12 days, and if his eye remains red or there is active tearing, discharge or both, he should stay home. While at home, he should also avoid sharing anything that could transmit the virus, such as towels and washcloths, to avoid intra-familial spread.

2. Jhanji V, Chan TCY, Li JEA, et al. Adenoviral keratoconjuncti-
7. Gonzalez-Lopez JJ, Miccio-Lait R, Muñoz-Neira FJ. Quanti-
10. Kauflman HE. Adenovirus advances: new diagnostic and thera-
14. Ladson PR, Drill S, Dooner J, Orphan G. Corneal infiltrates in epidemic keratoconjunctivitis. Response to double-blind cortico-

THE 2016 AAO SHOW DAILY!

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Show copies will also be available at Review of Optometry booth #915.
Out, Damned Spot

A 26-year-old female presented with a chief complaint of missing spots in her vision in the right eye, which she first noticed while watching television. She had a two-year history of one to two headaches a week that was unchanged. She had no ocular pain and no headache at the time of her vision loss. Her vitals at the time of exam included a blood pressure of 151/90 and a pulse of 95 BPM. Her general health and previous ocular history was noncontributory.

On examination, her visual acuity measured 20/200 OD and 20/20 OS. Her pupils were equally round and reactive to light. An afferent pupillary defect was seen in the right eye. On color vision testing, she had red desaturation in the right eye by 50%, compared with her left eye, and had decreased light brightness compared with the left eye. She was unable to decipher the test plate on Ishihara with the right eye and had normal color vision in

Figs. 1a and 1b. Fundus photos of our 26-year-old female patient’s right and left eyes at clinical presentation.

Figs. 2a and 2b. SD-OCT images of our 26-year-old female patient’s right and left eyes at clinical presentation.

Can you identify any significant findings in either the patient’s right (at left) or left macula using these SD-OCT images and data?

By Leslie Small, OD, and Mark Dunbar, OD
the left eye. Her extraocular motility was normal, and confrontation visual fields in the right eye were significant for a central scotoma with preserved peripheral vision. The left confrontation visual field was full. The slit lamp exam was unremarkable and intraocular pressure was normal.

On dilated fundus exam, the optic nerves appeared normal. There was no disc edema in either eye, and the macula also looked normal, although there was a reduced fovea light reflex in the right eye (Figure 1).

An OCT (Figure 2), visual field (Figure 3) and multifocal electroretinography (ERG) (Figure 4) and fundus autofluorescence (FAF) (Figure 5) are available for review.

**Take the Retina Quiz**

1. What is the significant OCT finding?
   a. Loss of foveal contour.
   b. Thickening of the nerve fiber layer in the macula and optic nerve.
   c. Disruption of inner segment/outer segment junction and thinning of outer nuclear layer.
   d. Accumulation of fluid.

2. What does the multifocal ERG show?
   a. It is normal.
   b. A choroidal excavation.
   c. A central depression in the right and normal foveal peak in the left.
   d. A normal right eye and hypersensitivity of the left.

3. What is the likely diagnosis?
   a. Malingerer.
   b. Cone-rod dystrophy.
   c. Acute zonal outer occult retinopathy.
   d. Stargardt’s macular dystrophy.

4. What is the expected prognosis?
Fig. 5. Note that the right eye (at left) shows patchy hyperautofluorescence around the optic nerve and macula, while the left shows the normal uniformly diffuse autofluorescence in these FAF images.

a. Stabilization of the visual field by six months.
b. Some improvement of the inner segment/outer segment integrity.
c. Persistent visual defect, including scotoma, usually persists.
d. All of the above.

**Diagnosis**

Based on the patient’s symptoms and testing, we suspected she had acute zonal occult outer retinopathy (AZOOR). This condition has an unconfirmed etiology, but investigators suspect either a viral or autoimmune cause.1 AZOOR, first described in 1992, shows a predominance in young women and is characterized by acute photopsias, scotomas and ERG abnormalities with minimal or no fundus findings, minimal or no vitreous cell and normal fluorescein angiography (FA).1,2 Investigators found 20% of patients had a viral prodrome.1 A majority of these patients have vision 20/40 or better, with only 5% of eyes presenting with 20/200 or worse.1 Changes can be detected on FAF and on OCT that correlate with the field loss and diminished ERG. Due to our suspicion, we acquired mfERG, FAF and FA. Our patient had significant visual field loss in the right eye that correlated with a central depression on her mfERG (Figure 3). The FAF also showed corresponding increased hyperautofluorescence around the optic nerve and macula (Figure 4). This is common in AZOOR, indicating damage to the RPE.3 The OCT showed marked disruption of the inner segment/outer segment junction in the right eye, suggesting photoreceptor involvement, a consistent finding in AZOOR (Figure 5).3 These abnormalities were all in the setting of a normal fundus exam (Figure 1) and FA. This testing confirmed the diagnosis of AZOOR. As in this case, multimodal imaging is critical for establishing diagnosis.

**Management**

There is no established treatment for AZOOR, and the natural course of AZOOR is highly variable. Only 26% of cases show visual improvement, and 13% had visual deterioration, according to researchers.1 Most cases reported had visual field defects that stabilized by six months.1,4 Treatment options include steroid therapy, antiviral therapy and noncorticosteroid immunosuppression. All therapies show limited success.1,5 The most commonly used treatment that has shown possible benefits is steroid therapy.1,5 Some studies show that early initiation is critical.5 These studies suggest that AZOOR has an inflammatory component involving the photoreceptors and that early initiation of steroid therapy may have a better potential to reverse the natural course.1,4

The difficulty with steroid therapy is that confirmation of the diagnosis often takes significant time and the results become less beneficial with delayed treatment.5

With our patient, steroid treatment (40mg oral prednisolone daily) and antiviral therapy of 1g Valtrex (valacyclovir HCL, GlaxoSmithKline) daily was initiated seven days after the start of symptoms. At her six month follow-up from presentation, her exam findings were stable with the exception of visual acuity improvement in the right eye to 20/30 from 20/200, and resolution of her RAPD. She still has a persistent central scotoma, but the patient appreciated subjective significant field improvement. Her FAF showed improvement as well with a decrease in hyperautofluorescence around the macula and nerve. On OCT of the macula, the integrity of her inner segment/outer segment junction had improved. ■

Leslie Small, OD, practices at the Bascom Palmer Eye Institute.

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In July, Review of Optometry celebrated its 125th anniversary. Upon the occasion, I took a look back at decades of published articles on glaucoma and evaluated their clinical relevance today. Now, I’m taking some time to reflect upon the 30 years that I personally have spent in practice. In that era, we’ve witnessed tremendous changes in glaucoma care—and it’s not over yet.

Here, I discuss how glaucoma care has changed in 30 years and what developments currently in the pipeline will become the norm in monitoring these patients in the future.

The Way We Were
When I began practice in 1985, the diagnosis and management of glaucoma was simple. In hindsight, this was probably due to lack of knowledge about the disease’s nuances and risk factors complicating matters. Back then, we initiated treatment on patients who had elevated IOPs, suspicious disc findings and visual field defects. In cases where field defects were not detected, we often deferred treatment until an IOP threshold—usually 30mm Hg—was reached. Research already showed black patients generally had larger optic nerves and, therefore, larger optic cups. We also thought that normal tension glaucoma was relatively rare. We didn’t yet know the role that corneal thickness played, especially in black patients, nor were we fully on board with the concept of preperimetric glaucoma (glaucomatous damage that was present before visual field aberrations). In fact, the earlier definition of glaucoma as preperimetric was provided by the American Academy of Ophthalmology included references to elevated IOP, optic nerve damage and visual field defects.

Treatment options were limited at that time. Timolol was the workhorse of topical therapy, and Propine (dipivefrin, Allergan) had only recently been approved. Of course, pilocarpine and other cholinergics were available, although accompanied by associated side effects. Often, patients we treated ended up undertreated, and those we only monitored eventually developed field defects detectable with standard automated perimetry.

Eventually, we recognized that glaucomatous optic neuropathy did occur in the early stages without identifiable visual field loss and, subsequently, the Academy of Ophthalmology changed its definition of glaucoma by removing the recommendation that field defects be present. Given that field testing was subjective and variable in reliability, we tended to rely more on structural appreciation of optic nerve nuances and IOP in determining the presence or absence of glaucoma. Stereoscopic optic nerve photography was considered the gold standard in optic nerve imaging, and it served us well.

We Have the Technology
The early 90s saw a movement to develop more precise instruments to measure optic nerve characteristics. Visual field testing modifications remained relatively stagnant. Also, when these newer imaging instruments were becoming more common, the results of several well-designed glaucoma risk factor studies began to trickle in. These are now commonly referred to as the “alphabet soup” studies.
of glaucoma, due to their names—"The Ocular Hypertension Study (OHTS)," "Advanced Glaucoma Intervention Study (AGIS)," and the "Collaborative Initial Glaucoma Treatment Study (CIGTS)," for example. Further studies on the data they presented have left us with good guidelines on how to evaluate a patient with glaucoma and predict their likelihood of progression.

While the imaging technology began to take on a more precise, objective nature, we as clinicians tended to describe the optic nerve in simple terms related to the relationship between the optic disc and the optic cup; the all too familiar cup-to-disc ratio. While the use of the cup-to-disc ratio was a simple tool to describe some characteristics of the optic disc, we relied too heavily on its documentation. We've all had patients for whom we document a cup-to-disc ratio widely different from one visit to another. We may see a nerve and call it 2x2 then, the next time we see the same nerve, call it 1x1. Of course, inter observer variation is even greater. So while we've realized that the cup-to-disc ratio has limited practical value, we continue to use it. And even objective instrumentation, whether it's scanning laser tomography or optical coherence tomography (OCT), usually has some reference to a 'cup' and 'disc' in one form or another.

With the advent of high resolution OCT technology, some researchers have suggested modifying our approach to evaluating the optic nerve by including different markers of optic nerve structure, such as Bruch’s membrane opening.

**The World of Tomorrow**

At the end of the day, anatomy is anatomy is anatomy. Knowing normal anatomy, and the finer nuances thereof, makes identifying abnormal anatomy much easier.

When we look at a cross section of the optic nerve, all individual ganglion cells (whether there are a million of them or only 100,000), pass from the retinal ganglion cell layer into the optic nerve by passing medial to Bruch’s membrane opening.

So where does this lead the clinician in a glaucoma practice? One study shows rather exquisitely the relationship between the Bruch’s membrane opening (BMO), the internal limiting membrane and the neuroretinal rim of ganglion cells that lies between these two readily identifiable structures. By identifying the BMO, and the adjacent neuroretinal rim in a series of cross sections through the optic nerve, a viable picture begins to emerge of the volumetric characteristics of the very ganglion cells we are trying to salvage.

Furthermore, retinal nerve fiber layer (RNFL) circle scans centered around the optic nerve can identify areas of nerve fiber layer thinning corresponding to preferential areas affected in glaucoma. Likewise, studies show defects in the ganglion cell layer in the macula, indicating early glaucomatous structural loss.

But the central question remains unanswered: where does the structural loss initially occur? In the macula? In the periopic RNFL? How far away from the center of the optic disc should we measure the RNFL? Does it occur first in the neuroretinal rim in the optic disc adjacent to Bruch’s membrane opening? We don’t know the precise answer yet—my guess is that it can occur in any of these locations first, probably governed by individual patient characteristics. With that being the case, we need to image all these areas.

Functionally, some newer perimetry strategies and instruments better correlating with early detection are evolving. We will be seeing more developments in the next few years, as more data comes in pertaining to new, more reliable scanning techniques. Eventually, we will begin easing away from the cup-to-disc terminology that we’ve held on to for decades.

Infectious keratitis is a common condition that carries a high risk of visual morbidity. Often, these patients present with a painful red eye and varying vision loss following an injury to the cornea or, perhaps, a contact lens related mishap. Epithelial disruption and focal stromal infiltration with edema are hallmark findings of infectious keratitis.

Optometrists and comprehensive ophthalmologists have grown accustomed to success with fluoroquinolone antibiotics in managing mild and moderate cases. Unfortunately, things don’t always go according to plan, even with these superior antibiotics. Patterns of bacterial resistance have begun to develop.

While bacteria are the most common cause of infectious keratitis, fungal and protozoan infections do occur, and delays in diagnosis often result in a poorer visual outcome, especially if topical corticosteroids are used adjunctively with an ineffective antibiotic. In the past several years, one of the most common causes of malpractice litigation against optometrists that we have seen is alleged mismanagement of infectious keratitis.

There are various approaches to managing suspected bacterial keratitis. Some use fluoroquinolones, while others use fortified antibiotics. Some rely on topical steroid adjuncts, while others opt for no steroids. While some use microbiologic studies, others employ empiric therapy.

Of course, few can argue against a good outcome. When the outcome is poor, such as in the case of resistant bacteria, or a fungal or protozoan cause, litigation becomes a possibility.

In cases where we have defended our colleagues in these situations, invariably the plaintiff’s attorney will ask at some point in the deposition, “where are the culture results?”

In this column, we look at practical management of infectious keratitis.

**Where’s the Culture?**

Practical recommendations for managing keratitis.

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

**Treatment Methods**

Historically, bacterial keratitis management involved the combined use of two fortified antibiotics, usually an alternating regimen of cefazolin 10% and an aminoglycoside, such as tobramycin 1.5%. These medications are developed from parenteral forms in a compounding pharmacy and used off-label. They can be challenging to obtain, have a limited shelf life and can be quite corneotoxic.

Unquestionably we have become comfortable with the success of the newer fluoroquinolone antibiotics. Many have replaced fortified antibiotic use with later generation fluoroquinolones due to their availability, tolerability and effectiveness. In a study comparing gatifloxacin 0.3% with fortified tobramycin and cefazolin in treating bacterial keratitis, investigators report that fluoroquinolone monotherapy was equivalent to fortified combination therapy. A similar study individually comparing both moxifloxacin 0.5% and gatifloxacin 0.3% with combined fortified cefazolin and tobramycin in bacterial corneal ulcers ranging in size from 2mm to 8mm found
no difference in clinical cure rates, with fluoroquinolone monotherapy performing as successfully as fortified polytherapy.\textsuperscript{2} Moxifloxacin 0.5% was also seen in another report to have the same healing success as fortified polytherapy.\textsuperscript{1}

While no data from prospective, controlled, human clinical trials is available regarding the specific use of besifloxacin for bacterial keratitis, several publications advocate for this agent as a safe and effective therapy.\textsuperscript{4,5}

**In the Literature**

The success of later generation fluoroquinolone monotherapy has changed how practitioners approach cases of suspected bacterial keratitis. A report surveying ophthalmologists in a four-state area found that most respondents initiate empiric therapy with the newer fluoroquinolone antibiotics for corneal ulcers, forgoing Gram staining and culturing.\textsuperscript{6}

In another survey, a minority of corneal ulcers were Gram stained or cultured, though cornea specialists were more likely to perform both. The most popular antibiotic for the treatment of less severe ulcers was moxifloxacin, while the most popular treatment of more severe ulcers was a fortified broad-spectrum antibiotic. Cornea specialists were more likely than noncornea specialists to prescribe fortified antibiotics instead of later generation fluoroquinolones for more severe corneal ulcers.\textsuperscript{7}

Despite longstanding recommendations to stain and culture cases of presumed infectious keratitis, approximately half of all comprehensive ophthalmologists have long forgone microbiologic study in favor of empiric treatment.\textsuperscript{8}

Formal microbiologic evaluation of microbial keratitis involves multiple corneal scrapings for samples to culture on various growth media such as chocolate agar, 5% sheep blood agar with Columbia agar base (SBA), Gram stain, Sabouraud agar, thioglycolate broth and brain heart infusion broth.\textsuperscript{9} This culturing method increases the probability of recovering a responsible pathogen from corneal tissue, which has a relatively low microbial load. It is not, however, cost effective for most eye care practitioners.

Obtaining and maintaining fresh, unexpired media, storing and transporting it properly is costly and can be a deterrent for many.

**Table 1. Culture Testing Options**

<table>
<thead>
<tr>
<th>CPT Code</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>87070</td>
<td>Culture, bacterial; any other source except urine, blood or stool, with isolation and presumptive identification of isolates.</td>
</tr>
<tr>
<td>87076</td>
<td>Anaerobic isolate, additional methods required for definitive identification of isolates.</td>
</tr>
<tr>
<td>87081</td>
<td>Culture, presumptive, pathogenic organisms, screening only.</td>
</tr>
<tr>
<td>87106</td>
<td>Culture, fungi, definitive identification, each organism; yeast.</td>
</tr>
<tr>
<td>87107</td>
<td>Culture, fungi, definitive identification, each organism; mold.</td>
</tr>
<tr>
<td>87118</td>
<td>Culture, mycobacterial, definitive identification, each isolate.</td>
</tr>
<tr>
<td>87184</td>
<td>Susceptibility studies, disk method, per plate (12 or fewer agents).</td>
</tr>
<tr>
<td>87205</td>
<td>Smear, primary source with interpretation; Gram or Giemsa stain for bacteria, fungi or cell types.</td>
</tr>
</tbody>
</table>

**Other Methods**

An alternative to directly plating corneal scrapings involves commercially available swab systems that employ a transport medium designed to keep organisms viable until a lab can inoculate appropriate media. With this method, a rayon swab is rolled or rubbed across the infiltrate in order to collect organisms. It is then placed into a tube containing transport media such as Amies agar gel or modified Stuart’s medium. The transport medium presumably now carrying the organism is then sent to a lab and plated onto the appropriate substrates.

One such commercially available device is the BD CultureSwab (Becton-Dickinson).\textsuperscript{10} Another is the ESwab (Copan Diagnostics). Whichever brand you choose, the nylon-tipped swab uses spray-on flocked fiber technology, improving sample collection and specimen release, with less entrapment than dacryon, rayon and cotton tips. The swab uses modified Amies medium, which maintains sample viability for 48 hours and has a shelf-life of 18 months.\textsuperscript{9} These swab-based transport media are inexpensive and an account with a local lab can easily be set up to facilitate pick up of specimens with subsequent analysis.

Of course, it is incumbent upon the practitioner to know which cultures or other tests are standard for their given lab, and which additional tests may be pertinent to the case, such as a fungal culture or antimicrobial sensitivities. Table 1 lists some of the more common options for corneal specimens.

Despite their ease of use, these devices have been historically avoided due to a perception of ineffectiveness and low recovery or organisms in a condition that typically yields extremely few inocula.
even when using corneal scrapings. However, these fears may be unfounded. Investigators report transport media using these commercially available kits are quite successful in identifying infectious organisms in cases of keratitis, in some cases matching that seen with traditional culturing methods.9,11

Even with the ease and effectiveness of the simplified microbiological specimen collection devices, many cases of suspected infectious keratitis will still be treated empirically. Most do not advocate microbiologic study on every case of suspected infectious keratitis, but those with the greatest potential for vision loss should be considered for evaluation. A guide to identifying ulcers at risk of vision loss is the “1, 2, 3” rule. To identify potentially sight-threatening ulcers, any one of the following characteristics must be present:

1. > Cells 1+ in the anterior chamber (10 cells or greater in 1mm beam);
2. Dense infiltrate greater than 2mm in greatest linear dimension (by slit-lamp light measurement);
3. Edge of infiltrate smaller than 3mm from the center of cornea.12

Infectious keratitis is a potentially sight-threatening issue with several diagnostic and therapeutic approaches. Commercially available later generation fluoroquinolones and one-step culturing devices can help obtain a successful outcome for patients.

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The Right Patient Population

The ideal candidate is emmetropic in the dominant eye and mildly myopic in the non-dominant. Around -0.50D to -0.75D is ideal, according to FDA data.1 Patients may be naturally emmetropic, post-refractive, pseudophakic with a monofocal implant or have refractive surgery to achieve the ideal target.

A Kamra patient needs to have a clear lens, healthy cornea and a good tear film. Patients who are not good candidates for any other type of refractive surgery are likely not good candidates for this procedure. As with any type of refractive surgery, stabilizing the tear film prior to surgery is crucial, as it affects visual outcome and patient satisfaction.

Surgical Process

Prior to surgery, the patient will be measured on the Acutarget HD (AcuFocus) to check for optical scatter (lens dysfunction), provide a dynamic tear film assessment and determine proper placement.

Using a surgical microscope, the surgeon will mark the Purkinje, create the pocket using a femtosecond laser and insert the inlay centering around the marking.

Postop

After the procedure, patients are placed on an antibiotic, a three-month minimum course of steroids and regular use of artificial tears. At three months if the patient is healing well and vision is good, the drops may be discontinued. If any issues persist, drops may be continued for an extended period of time. Every surgeon has a different drop regime, so comanaging optometrists should check with the surgery center on its protocol. We have found putting patients on a cyclosporine drop preoperatively and postoperatively helps speed up visual recovery.

Patients should expect a slow visual recovery, with the majority of patients comfortably reading and seeing well at distance by one month. Roughly 20% of our patients notice immediate near vision improvement, while the remaining 80% take a few weeks to a few months to read comfortably.

Advantages

With the Kamra inlay, the patient is fully binocular at distance. Even though the average patient is mildly myopic, the pinhole effect improves vision, giving them distance vision, as well as near. This eliminates the imbalance monovision patients often complain about. Additionally, as the inlay is based on small-aperture optics, the reading will maintain as presbyopia progresses.

The Kamra inlay is an excellent option for well-selected presbyopic patients who are not ready for a lens-based procedure. I’ve had it for more than three years and have yet to use reading glasses.

Dr. Black is vice president and clinical director at Crystal Clear Vision in Toronto, where she examines and counsels patients seeking refractive surgery, including laser, corneal and lenticular procedures.

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or fax (718) 405-3808
Diagnostic Technology

OCTA, Better Scleral Lens Fitting

Optovue’s iSeries OCT devices can help with scleral lens fitting now that the company offers a new software package called Vault Mapping. Traditional corneal clearance imaging uses a fluorescein solution and a slit lamp, which yields one cross-sectional view. With this new software, Optovue says, a clearance assessment map is created to help determine if the lens is tilting or fitting improperly.

The company also recently brought its Angiovue imaging system to optometry, giving ODs a more precise way to view the retinal vasculature, Optovue says. The system provides conventional OCT as well as OCT angiography (OCTA) to quickly visualize the retinal microvasculature without dye. Optometrists can integrate vascular structure assessment with other imaging to form a more complete picture of retinal health.

Visit optovue.com.

Confocal Scanner Autofluorescence

Centervue now offers fundus autofluorescence capabilities on its Eidon line of confocal scanners. The new model, the Eidon AF, obtains the same range of information from multiple imaging modalities as the original while adding fundus autofluorescence, allowing you to assess the retinal pigment epithelial layer, according to the company.

The new model captures a 60° autofluorescence image with a single flash of light. The Eidon AF also offers wide-field views of the retina up to 110°, according to Centervue.

Visit centervue.com.

Eyeefficient, Mediworks Go Digital

Optometrists interested in going high tech with their slit lamp imaging and vision testing charts can look forward to checking out a new line of equipment from Mediworks that’s now being distributed by Eyeefficient.

Eyeefficient is also working to provide installation and service to equipment within the continental US.

Visit eyefficient.com.

Local Eye Site Assessment

Hiring? Use an assessment tool that is scientifically proven to predict performance in eye care jobs.

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BRIEF SUMMARY OF PRESCRIBING INFORMATION

INDICATIONS AND USAGE
TRAVATAN Z® (travoprost ophthalmic solution) 0.004% is indicated for the reduction of elevated intraocular pressure in patients with open-angle glaucoma or ocular hypertension.

DOSEAGE AND ADMINISTRATION
The recommended dosage is one drop in the affected eye(s) once daily in the evening.

TRAVATAN Z® (travoprost ophthalmic solution) should not be administered more than once daily since it has been shown that more frequent administration of prostaglandin analogs may decrease the intraocular pressure lowering effect.

Reduction of the intraocular pressure starts approximately 2 hours after the first administration with a maximum effect reached after 12 hours.

TRAVATAN Z® (travoprost ophthalmic solution) may be used concomitantly with other topical ophthalmic drug products to lower intraocular pressure if more than one topical ophthalmic drug is being used, the drugs should be administered at least five (5) minutes apart.

CONTRAINDICATIONS
None

WARNINGS AND PRECAUTIONS

Pigmentation
i. travoprost ophthalmic solution has been reported to cause changes to pigmented tissues. The most frequently reported changes have been increased pigmentation of the iris, perilimbal tissue (eyelid) and eyelashes. Pigmentation is expected to increase as long as travoprost is administered. The pigmentation change is due to increased melanin content in the melanocytes rather than an increase in the number of melanocytes. After discontinuation of travoprost, pigmentation of the iris is likely to be permanent, while pigmentation of the perilimbal tissue and eyelash changes have been reported to be reversible in some patients. Patients who receive treatment should be informed of the possibility of increased pigmentation. 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 near nor farsighted of the iris appear to be affected by treatment. While treatment with TRAVATAN Z® (travoprost ophthalmic solution) 0.004% can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly.

Eyelash Changes
TRAVATAN Z® Solution may gradually change eyelashes and vellus hair in the treated eye. These changes include increased length, thickness, and number of lashes. Eyelash changes are usually reversible upon discontinuation of treatment.

Intraocular Inflammation
TRAVATAN Z® Solution should be used with caution in patients with active intraocular inflammation (e.g., uveitis) because the inflammation may be exacerbated.

Muscular Edema
Muscular edema, including cutaneous muscular edema, has been reported during treatment with travoprost ophthalmic solution. TRAVATAN Z® Solution should be used with caution in aphakic patients, in pseudophakic patients with a torn posterior lens capsuless, or in patients with known risk factors for muscular edema.

Angle-closure, Inflammatory or Neovascular Glaucoma
TRAVATAN Z® Solution has not been evaluated for the treatment of angle-closure, inflammatory or neovascular glaucoma.

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.

Use with Contact Lenses
Contact lenses should be removed prior to instillation of TRAVATAN Z® Solution and may be reinserted 15 minutes following its administration.

ADVERSE REACTIONS

Clinical Studies Experience
Because clinical studies are conducted under widely varying conditions, adverse reaction rates observed in the clinical studies of a drug cannot be directly compared to rates in the clinical studies of another drug and may not reflect the rates observed in practice. The most common adverse reaction observed in controlled clinical studies with TRAVATAN Z® (travoprost ophthalmic solution) 0.004% and travoprost 0.005% (travoprost ophthalmic solution 0.004% was replaced with 0.005%) was reported in 30% to 50% of patients: up to 3% of patients discontinued therapy due to conjunctival hyperemia. Ocular adverse reactions reported at an incidence of 5% or more are included in this table. Some of these clinical studies included decreased visual acuity, eye discomfort, foreign body sensation, pain and pruritus. Ocular adverse reactions reported at an incidence of 1 to 4% in clinical studies with TRAVATAN Z® 0.004% include: ocular irritation, decreased visual acuity, abnormal vision, blurred vision, dryness, eye irritation, keratitis, lid margin crusting, ocular inflammation, photosensitivity, subconjunctival hemorrhage and tearing.

Nonocular adverse reactions reported at an incidence of 1% or more in these clinical studies were: artralgia, anxiety, arthritis, back pain, basal cell carcinoma, behavioral disorder, depression, dyspepsia, gastrointestinal disorder, headache, hypertension, mental depression, myalgia, back pain, pruritus, skin irritation, conjunctivitis, photophobia, rash, sneezing, rhinitis, urticaria, upper respiratory disease, and vertigo. In postmarketing use with prostaglandin analogs, peripheral and lid changes including deepening of the eyelid sulcus have been observed.

USE IN SPECIFIC POPULATIONS

Pregnancy
Pregnancy Category C

i. travoprost was teratogenic in rats, all of which died up to 10 mcg/kg/day (250 times the maximally recommended human dose (MRHD), evidenced by an increase in the incidence of skeletal malformations as well as external and visceral malformations, such as fused sternebrae, dorumed head and hydrocephaly. ii. travoprost was not teratogenic in rats at 4 times up to 5 mcg/kg/day (70 times the MRHD, or in mice at subcutaneous doses up to 1 mcg/kg/day (25 times the MRHD), and was toxic in subcutaneous doses greater than 0.3 mcg/kg/day (7.5 times the MRHD).

In the offspring of female rats that received travoprost subcutaneously from Day 7 of pregnancy to lactation Day 21 at doses of 0.12 mg/kg/day (3 times the MRHD), the incidence of postnatal mortality was increased, and neonatal body weight was decreased. Neonatal development was also affected, evidenced by delayed eye opening, gena detachment and propellargulp separation, and by decreased motor activity.

There are no adequate and well controlled studies of TRAVATAN Z® (travoprost ophthalmic solution) 0.04% administration in pregnant women. Because animal reproductive studies are not always predictive of human response, TRAVATAN Z® Solution should be administered during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers
A study in lactating rats demonstrated that radioiodinated travoprost and/or its metabolites were excreted in milk. It is not known whether this drug or its metabolites are excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when TRAVATAN Z® Solution is administered to a nursing woman.

Pediatric Use
Use in pediatric patients below the age of 16 years is not recommended because of potential safety concerns related to increased pigmentation following long-term use.

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

Hepatic and Renal Impairment
TRAVATAN Z® solution 0.004% has been studied in patients with hepatic impairment and also in patients with renal impairment. No clinically relevant changes in hematology, blood chemistry, or urinalysis laboratory data were observed in these patients.

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment of Fertility
Two-year carcinogenicity studies in mice and rats at subcutaneous doses of 10, 30, and 100 mcg/kg/day did not show any evidence of carcinogenic potential. However, at 100 mcg/kg/day, male rats were only treated for 82 weeks, and the maximum tolerated dose (MTD) was not reached in the mouse study. The high dose (100 mcg/kg) corresponds to exposure levels over 400 times the human exposure at the maximum recommended human ocular dose (MRHD) of 0.04 mcg/kg, based on plasma active drug levels. Travoprost was not mutagenic in the Ames test, mouse micronucleus test or rat chromosome aberration assay.

A slight increase in the mutant frequency was observed in one of two mouse lymphoma assays in the presence of rat S-9 activation enzymes.

Travoprost did not affect mating or fertility indices in male or female rats at subcutaneous doses up to 10 mcg/kg/day [250 times the maximum recommended human dose (MRHD), 0.04 mcg/kg/day], or 10 mcg/kg/day, the mean number of corpora lutea was reduced, and the post-implantation losses were increased. These effects were not observed at 3 mcg/kg/day (7.5 times the MRHD).

PILOT UZANUSIL UV-IPROTON IM

Potential for Pigmentation
Patients should be advised of the potential for increased brown pigmentation of the iris, which may be permanent. Patients should also be informed about the possibility of eyelid skin darkening, which may be reversible after discontinuation of TRAVATAN Z® (travoprost ophthalmic solution) 0.004%.

Potential for Eyelash Changes
Patients should also be informed of the possibility of eyelash and vellus hair changes in the treated eye during treatment with TRAVATAN Z® Solution. These changes may result in a disparity between eyes in length, thickness, pigmentation, number of eyelashes or vellus hairs, and/or direction of eyelashes growth. Eyelash changes are usually reversible upon discontinuation of treatment.

Handling the Container
Patients should be instructed not to allow the tip of the dispensing container to contact the eye, surrounding structures, fingers, or any other surface in order to avoid contamination of the solution by common bacteria known to cause ocular infections. Serious damage to the eye and subsequent loss of vision may result from using contaminated solutions.

When to Seek Physician Advice
Patients should also be advised that if they develop an intercurrent ocular condition (e.g., trauma or infection), have ocular surgery, or develop any ocular reactions, particularly conjunctivitis and eyelid reactions, they should immediately seek their physician’s advice concerning the continued use of TRAVATAN Z® Solution.

Use with Contact Lenses
Contact lenses should be removed prior to instillation of TRAVATAN Z® Solution and may be reinserted 15 minutes following its administration.

Use with Other Ophthalmic Drugs
If more than one topical ophthalmic drug is being used, the drugs should be administered at least five (5) minutes between applications.

Rx Only
U.S. Patent Nos. 5,631,287; 5,889,052; 6,011,062; 6,235,781; 6,503,497; and 6,849,253

Alcon Laboratories, Inc.
Fort Worth, Texas 76134 USA
10/15 US-212-15-E-0279

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Vision Going Down the Drain

By Andrew S. Gurwood, OD

History
A 57-year-old black female reported to the office with a chief complaint of dimmed vision in her left eye for a week. She noticed the change in vision after a procedure to treat her glaucoma. A phone call to that practice revealed that the patient had a drainage valve implanted in her left eye to augment a failing trabeculectomy.

She was placed on topical antibiotics, topical steroids and topical nonsteroidal anti-inflammatory medications QID and removed from all topical glaucoma medications.

Her systemic history was remarkable for hypertension, for which she was properly medicated. She denied allergies of any kind.

Diagnostic Data
Her best corrected entering visual acuities were 20/30 OD and 20/20 OS at distance and near with no improvement upon pinhole. Her external examination was normal with no evidence of afferent pupil defect. The biomicroscopic examination of the anterior segment was normal with a well placed drainage device and no evidence of complications. Goldmann applanation tonometry measured 15mm Hg in both eyes. Studies included stereobiomicroscopic examination of the fundus, photodocumentation and B-scan ultrasonography (Figure 1). Pertinent clinical findings in the posterior segment of the left eye are demonstrated (Figure 2).

Your Diagnosis
Does this case require additional tests? How would you manage this patient? What is the likely prognosis?

To find out, please visit www.reviewofoptometry.com.

Retina Quiz Answers (from page 88): 1) c; 2) c; 3) c; 4) d.
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AN INITIATIVE OF CooperVision
INDICATIONS AND USAGE
TRAVATAN Z® (travoprost ophthalmic solution) 0.004% is indicated for the reduction of elevated intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension.

Dosage and Administration
The recommended dosage is 1 drop in the affected eye(s) once daily in the evening. TRAVATAN Z® Solution should not be administered more than once daily since it has been shown that more frequent administration of prostaglandin analogs may decrease the IOP-lowering effect.

TRAVATAN Z® Solution may be used concomitantly with other topical ophthalmic drug products to lower IOP. If more than 1 topical ophthalmic drug is being used, the drugs should be administered at least 5 minutes apart.

IMPORTANT SAFETY INFORMATION
Warnings and Precautions
Pigmentation—Travoprost ophthalmic solution has been reported to increase the pigmentation of the iris, periorbital tissue (eyelid), and eyelashes. Pigmentation is expected to increase as long as travoprost is administered. After discontinuation of travoprost, pigmentation of the iris is likely to be permanent, while pigmentation of the periorbital tissue and eyelash changes have been reported to be reversible in some patients. The long-term effects of increased pigmentation are not known. While treatment with TRAVATAN Z® Solution can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly.

Eyelash Changes—TRAVATAN Z® Solution may gradually change eyelashes and vellus hair in the treated eye. These changes include increased length, thickness, and number of lashes. Eyelash changes are usually reversible upon discontinuation of treatment.

Use With Contact Lenses—Contact lenses should be removed prior to instillation of TRAVATAN Z® Solution and may be reinserted 15 minutes following its administration.

Adverse Reactions
The most common adverse reaction observed in controlled clinical studies with TRAVATAN Z® Solution was ocular hyperemia, which was reported in 30 to 50% of patients. Up to 3% of patients discontinued therapy due to conjunctival hyperemia. Ocular adverse reactions reported at an incidence of 5 to 10% in these clinical studies included decreased visual acuity, eye discomfort, foreign body sensation, pain, and pruritus. In postmarketing use with prostaglandin analogs, periorbital and lid changes including deepening of the eyelid sulcus have been observed.

Use in Specifi c Populations
Use in pediatric patients below the age of 16 years is not recommended because of potential safety concerns related to increased pigmentation following long-term chronic use.

For additional information about TRAVATAN Z® Solution, please see the brief summary of Prescribing Information on the adjacent page.


TRAVATAN Z® Solution has no FDA-approved therapeutic equivalent available

Help patients start strong and stay on track with Openings®

TRAVATAN Z® (travoprost ophthalmic solution) 0.004%