Diagnostic Quiz: Season of the Itch, *Page 106*

REVIEW® OF OPTOMETRY

July 15, 2015

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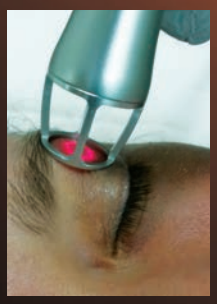

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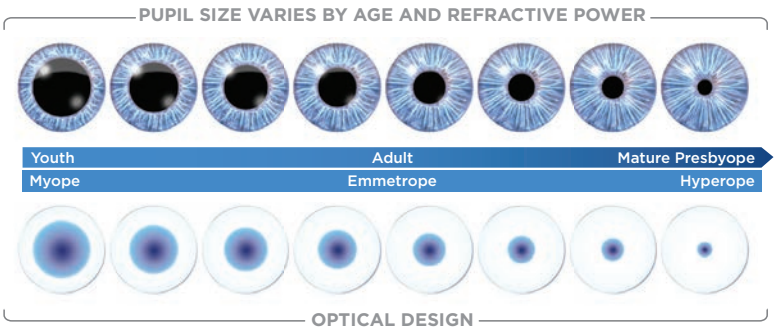

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VOL. 152 NO. 7 ■ JULY 15, 2015

IN THE NEWS

A recent **American Academy of Ophthalmology** report found that lacrimal drainage system plugs—including punctal, intracanalicular and dissolving types—**improve signs and symptoms by 50% or more** in patients with moderate dry eye and for whom topical lubrication has been ineffective. The study also noted complications from plugs were infrequent. The report, published online on May 30 in *Ophthalmology*, was based on a literature review of 27 studies.

An **FDA analysis**, published in the *British Medical Journal*, indicates drug companies don't properly inform the public of the reasons a drug is rejected. FDA complete response letters cite the specific safety and efficacy deficiencies that led to a drug's rejection; yet, as a whole, **companies only shared an average of 14% of those statements** in their press releases, the analysis found. "Press releases are incomplete substitutes for the detailed information contained in complete response letters," the study authors conclude.

Concetta Daurio, OD, MBA, died on June 3, 2015. Dr. Daurio was the **chief of Optometry at the Harvard Vanguard Medical Associates** in the Boston area, a past-chairman of the **American Optometric Association Multidisciplinary Section** and chaired various committees affiliated with the American Public Health Association.

Actavis recently announced its decision to change its name to **Allergan** after the acquisition of Allergan in March 2015. Stakeholders approved the name change June 5.

MRI Reveals EKC Inflammation

The conjunctival surface isn't the only location for inflammation in epidemic keratoconjunctivitis.

By Rebecca Hepp, Senior Associate Editor

When a 36-year-old male patient came in for a routine MRI to track the progress of bilateral optic nerve sheath meningiomas, Jonathan C. Horton, MD, PhD, and Steven Miller, MD, PhD, found more than they expected.

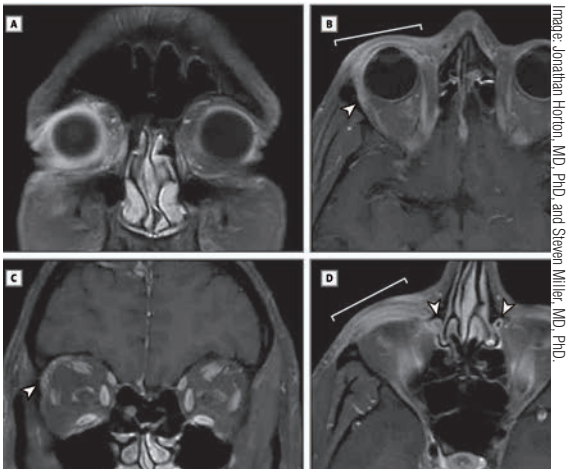

Image: Jonathan Horton, MD, PhD, and Steven Miller, MD, PhD.

A. Fat saturation, demonstrating enhancement of periocular tissues. B. Thickening of the right upper eyelid (bracket) and anterior orbital enhancement. Lacrimal gland is enlarged (arrowhead). C. Right lacrimal gland enlargement (arrowhead). D. Inflammation of the lower eyelid (bracket) and right nasolacrimal duct swelling with loss of air signal in the lumen (arrowheads).

The MRI scans revealed inflammation in the right orbit, prompting immediate follow up in the clinic. They found that the patient had developed epidemic keratoconjunctivitis (EKC) but had not complained of symptoms prior to the scheduled appointment. While inflammation is common on the conjunctival surface, this patient's MRI revealed some interesting surprises below the surface.

"The infection induces an inflammatory process that extends surprisingly deep into the orbit," the authors write. Published online May 28 in *JAMA Ophthalmology*, the primary findings based on the MRI imaging "were edema and inflammation of periocular tissue in the anterior orbit, enlargement of the lacrimal gland, and nasolacrimal duct compromise."

"Our case presented as a typical infection and the patient recovered uneventfully," the authors conclude, "indicating that deep tissue inflammation, dacryoadenitis and dacryocystitis are likely to be common manifestations of adenoviral conjunctivitis."

(Continued on page 6.)

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Deep Orbit Inflammation in EKC

(Continued from page 4.)

"Clinicians should still rule out a bacterial cellulitis or preseptal cellulitis, but the most common diagnosis of a severe conjunctivitis presentation is epidemic keratoconjunctivitis, and that is now evident with these MRI images," says Paul Karpecki, OD, head of the ocular surface disease clinic and director of clinical research at the Koffler Vision Group in Lexington, Ky.

This case study has implications for clinical practice, too. "It also shows the need for topical corticosteroids in severe cases given the level of inflammation that can be present," Dr. Karpecki says. "Finally, it helps us empathize with patients with epidemic keratoconjunctivitis who are miserable, and now we may have good evidence to understand why."

While Dr. Karpecki would love to see a larger study to confirm the findings, he notes MRI imaging can be cost prohibitive, and the data in this case can be sufficient to inform clinical decisions.

Horton J, Miller S. Research letter: Magnetic Resonance Imaging in Epidemic Adenoviral Keratoconjunctivitis. JAMA Ophthalmology. 2015 March 28. doi:10.1001/jamaophthalmol.2015.1457.

Infant's Visual Searching Associated with Later Autism Symptoms

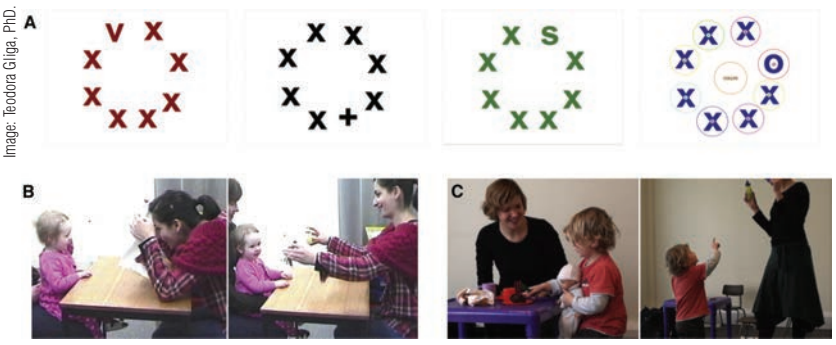
Researchers recently discovered that nine-month-old infants with increased visual searching abilities showed more autism symptoms at 15 months and two years.

Investigators from Babylab at the Centre for Brain and Cognitive Development at Birkbeck, University of London, used eye-tracking technology to follow infants' gazes when presented with letters on a screen. They studied 82 at-risk infants and 27 low-risk infants as controls, demonstrating, "for the first time, a relationship between superior visual search abilities during infancy and the severity of later autism symptoms," study author Teodora Gliga, PhD, writes.

The study supports evidence that perception and attention are more important than previously believed in autism's developmental pathway, the authors say. Investigators hope to continue research into why children with autism have improved visual perception, as well as the link between perception and social difficulties.

Gliga T, Bedford R, Charman T, et al. Enhanced visual search in infancy predicts emerging autism symptoms. Current Biology. 2015;25:1-4.

Image: Teodora Gliga, PhD.



A. Example stimuli and the areas of interest used in analysis. B. Example of behaviors assessed, such as anticipation of social contact and attention shifting. C. Example of behaviors assessed, such as pretend play and pointing.

Mascot Cleared in Hot Dog-Related Retinal Detatchment

A Jackson County, Kan., jury recently ruled against resident James Coomer, who had sought $300,000 in damages against the Kansas City Royals for injuries he sustained during a 2009 game.

According to court documents, Coomer was diagnosed with both a detached retina and a traumatic cataract eight days after being struck in the eye with a foil-wrapped hot dog thrown by the team's mascot, Sluggerrr. A 2011 ruling said Mr. Coomer was at fault. However, after an appeal, the case was sent back down for review. The Jackson County jury again handed up a decision in favor of Sluggerrr, effectively upholding the baseball rule that fans are responsible for being aware of their surroundings during a game.

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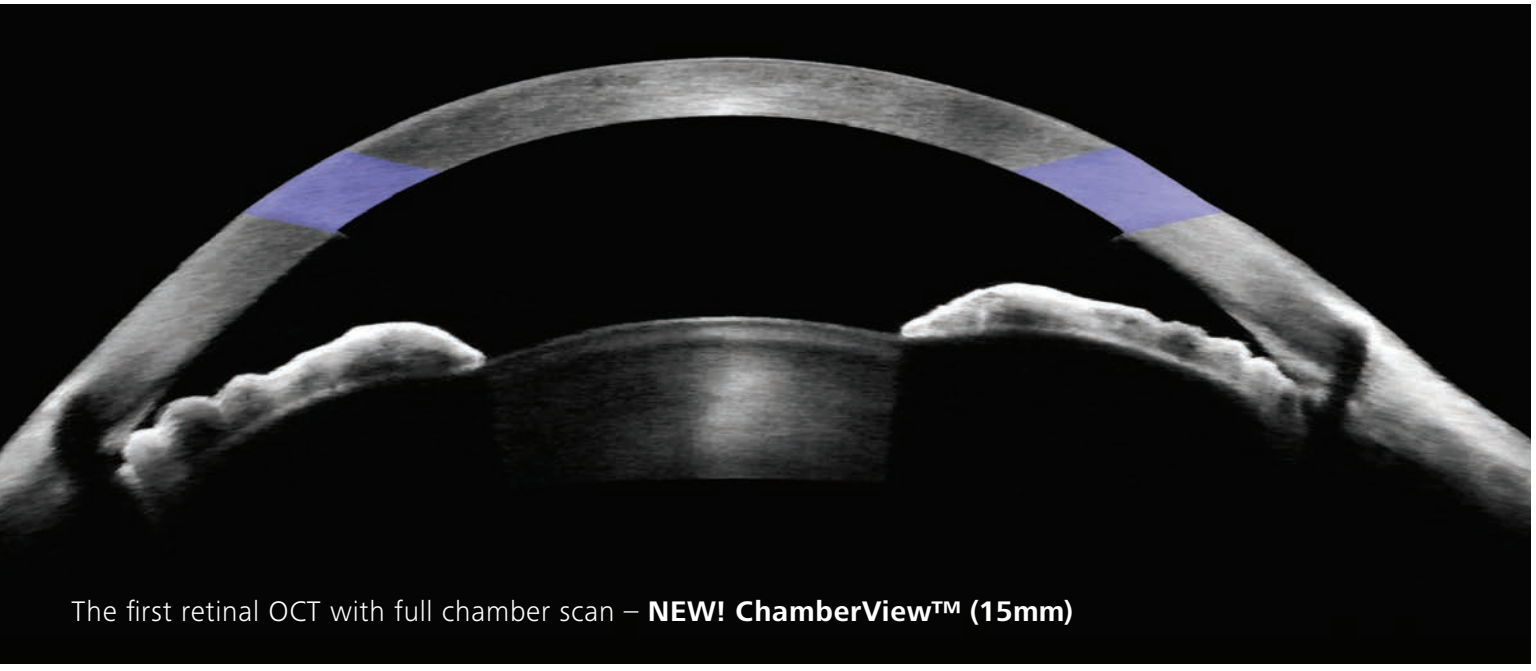

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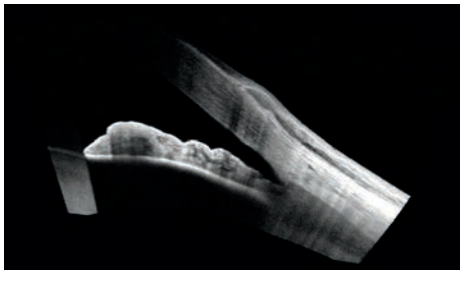

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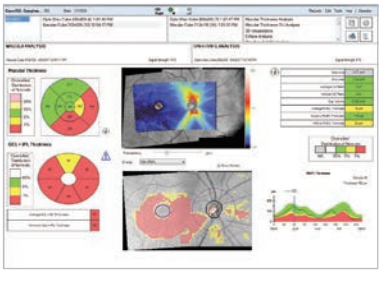

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Lymphatic System, Brain Linked

Investigators from the University of Virginia (UVA) School of Medicine have discovered that the brain is directly linked to the lymphatic system through a network of well-disguised meningeal lymphatic vessels.

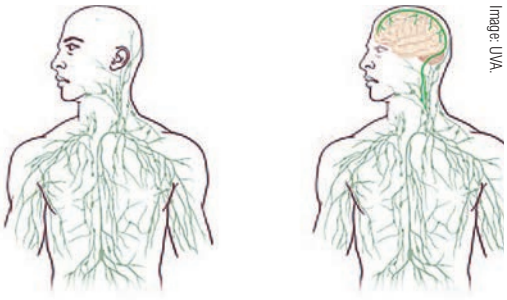

Image: UVA.

Map of the lymphatic system: traditional (left) and as redrawn by UVA's discovery.

Overturning decades of teaching, the new discovery will allow clinicians to approach neuro-immune interactions mechanistically, Jonathan Kipnis, PhD, professor in the UVA Department of Neuroscience and director of UVA's Center for Brain Immunology and Glia, said in a UVA press release.

"We believe that for every neurological disease that has an immune component to it, these vessels may play a major role," Kipnis said. "Hard to imagine that these vessels would not be involved in a [neurological] disease with an immune component."

Study author Antoine Louveau, PhD, a postdoctoral fellow, discovered the lymphatic vessels following a major blood vessel down into the sinuses, a finding that could have huge implications for diseases of the brain such as Alzheimer's, multiple sclerosis and autism, to name a few.

And considering the eye could be thought of as an extension of the brain, it could mean big changes to the future of eye care, too. "This may change our understanding of how certain inflammatory, infectious and degenerative neurologic disorders affect the optic nerves," says Michael Trottini, OD, of Outlook Eyecare in New Jersey. "It may give further insight into the mechanisms of the optic neuropathies as well as optic neuritis and papilledema."

"At this point there is no known lymphatic outflow from the eye, except for the lids and the conjunctiva," says Carlo Pelino, OD, assistant professor at Pennsylvania College of Optometry at Salus University.

He notes recent research is starting to reveal lymphatic endothelial markers in the eye, such as lymphatic vessel endothelial hyaluronan receptor one (LYVE1), transmembrane glycoproteins, VEGF R3 receptors and prox1 transcription factors.

"As eye care practitioners, we have to think, it's not just the lymphatics in the lids and conjunctiva anymore, we have to look to see if there are lymphatics elsewhere in the eye," Dr. Pelino says. "I think they are starting to come to that conclusion and this finding will really move things forward." ■

Louveau A, Smirnov I, Keyes T, et al. Structural and functional features of central nervous system lymphatic vessels. Nature. June 1, 2015. [Epub ahead of print].

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Optic nerve image on front cover courtesy of Michael Chaglasian, OD.

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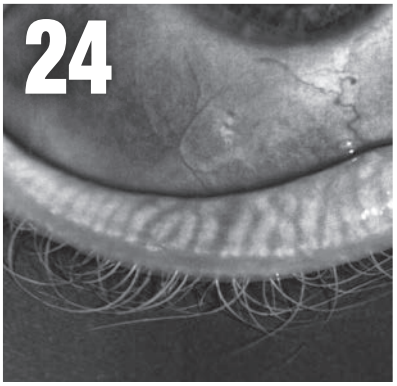

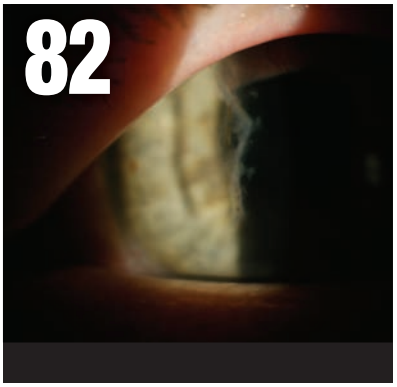

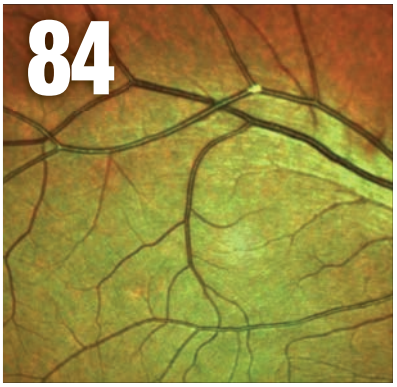

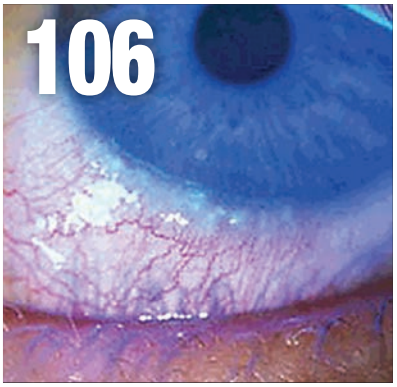

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References: 1. The big picture: eye protection is always in season. The Vision Council Website. http://www.thevisioncouncil.org/sites/default/files/VCUVReport2013FINAL.pdf. Accessed May 7, 2014. **2.** Chandler H. Ultraviolet absorption by contact lenses and the significance on the ocular anterior segment. *Eye Contact Lens*. 2011;37(4):259-266.

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Beyond the Numbers

Treat the patient, not the pressure, glaucoma experts remind practitioners. **By Jack Persico, Editor-in-Chief**

Interesting new research from Moorfields is helping the eye care community better understand the visual performance of glaucoma patients in real-world settings. After using eye tracking to monitor saccades while patients and controls watched TV, researchers identified telltale 'scanpaths' indicative of glaucoma. "Such a procedure would have the potential to provide a continuous assessment of changes either as the disease developed, or during treatment, within a more realistic visual environment," they write in the online journal *Frontiers in Aging Neuroscience*.

Seeing glaucoma as your *patients* see it humanizes the experience and might lead to better care. Many articles in our 21st annual Glaucoma Report this month highlight just such a patient-centered approach.

Since intraocular pressure is the only clinical factor in glaucoma that's amenable to intervention, it's no surprise that IOP plays such a central role. In just about all the important decisions—making the diagnosis, initiating treatment, changing meds, referring for surgery—IOP's influence is inescapable. But pressure doesn't matter to patients—vision does. They want to enjoy their normal daily activities unhindered by physical deficits that might diminish quality of life.

"Perhaps the time to start treating can't be based on numbers alone," glaucoma specialist Murray Fingeret, OD, explains in a feature on glaucoma's real-world impact (see page 54). "In fact, the patient's ability to function visually day to day may be just as integral as any diagnostic measurement."

Functional effects on vision are the focus of the Optometric Study Center CE on page 62, where Craig Thomas, OD, helps clinicians understand the relationship between structural damage and visual deficits. Can greater use of functional vision testing help you better realize the patient's experience of their condition? And what constitutes 'functional vision testing,' anyway?

"When most of us talk about functional changes in glaucoma, we are usually talking about changes in the visual field measured by an automated threshold perimeter," Dr. Thomas writes. "However, a disturbance of any test of visual function can indicate glaucoma-induced functional changes." He goes on to explain the clinical relevance of visual evoked potentials, electroretinography and other sophisticated measures, and how they improve the care of glaucoma patients.

ODs recognize that many patients who can't maintain adequate control with topical therapies or just find the daily ritual too frustrating might be better served by a surgical procedure. Richard Zimbalist, OD, and Angela Gentry, OD, provide a great overview of the latest surgical interventions on page 48. When glaucoma surgery is delayed because of over-reliance on topical drugs, patients may no longer be able to experience the desired outcome. Making the surgical referral need not be seen as relinquishing control; rather, you're ensuring the best possible intervention and quality of life. ■

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Why So Serious?

You can still have some old-fashioned fun the office—even without an Old Fashioned in your hand. **By Montgomery Vickers, OD**

Are optometrists allowed to use the "F" word anymore? You know, fun? It's easy to be serious these days. I remember when the first order of business at local association meetings had something to do with a certain beverage from Scotland. Needless to say, we had some *fun* discussions about our profession's challenges!

Plus, it was a lot easier to get doctors to agree to be officers when they were two swigs in.

It's not that we aren't fun individually or even when we mob some hapless legislator's office. I find plenty of reasons to laugh with, and at, my colleagues. To me, clip-on ties and hair gel are hilarious. Plus I think it is really, really fun to watch the aforementioned legislator try to pronounce difficult words like "glaucoma" and "latanoprost." Like I said, optometrists can be fun any time they want.

So why not have fun, not just when harassing politicians, but also in the office? I read things on Facebook, Twitter and all the other inane time wasters where optometrists are having near-death experiences just fitting a contact lens on some 50-year-old. Of course, being medically trained, we all just pop antidepressants when we can't solve problems the old fashioned way—by drinking Old Fashioneds all day.

But pill popping doesn't help. If you listen to the rapid disclaimers at the end of some miracle pill's TV ad, you should be well aware that the side effects of oral antidepressants include not wanting to put on new nose pads for free.

Cyndi Lauper hit the nail on the head when she sang, "Eye doctors just wanna have fun." Ok, a few changes were made in the final recorded version.

Here are some ideas to make your day in the office more fun:

1. When it's raining in Biblical proportions, ask your assistant to run out to your car and get the newspaper you left in the front seat.
2. When patients are no-shows, use the time to examine your lifelong invisible friend.
3. Schedule a recheck for your new invisible friend who can never seem to adapt to new glasses without a remake.
4. Read about the Texas Rangers. Not the baseball team—that will just depress you more. I mean the original Texas Rangers, the ones who made Texas what it is today, half as big as Alaska.
5. Tell your career-long patient that story about the lady who put fingernail polish remover in the same eye twice in one day. That one never gets old!
6. Take a peek inside your office refrigerator. Wonder what that green thing used to be …
7. Tell especially nice patients they have won the Patient of The Day contest, which comes with all the privileges someone would get if they didn't get a single number right on the lottery.
8. Whisper during the refraction—you get points if patients start to whisper back.
9. Tell patients you have to "puff" them 20 times and take the average. Throw out the results and do applanation tonometry because the puffer's seen better days.
10. With a new contact lens candidate, put a hard lens on one eye and a soft lens on the other and ask which seems more comfortable.
11. Visit the office attic or basement and kill anything that moves.
12. Tell your staff they are all fired and today is National Opposite Day then wait to see who shows up tomorrow.
13. Change your Snellen chart to read: "A guy walks into a bar." ■

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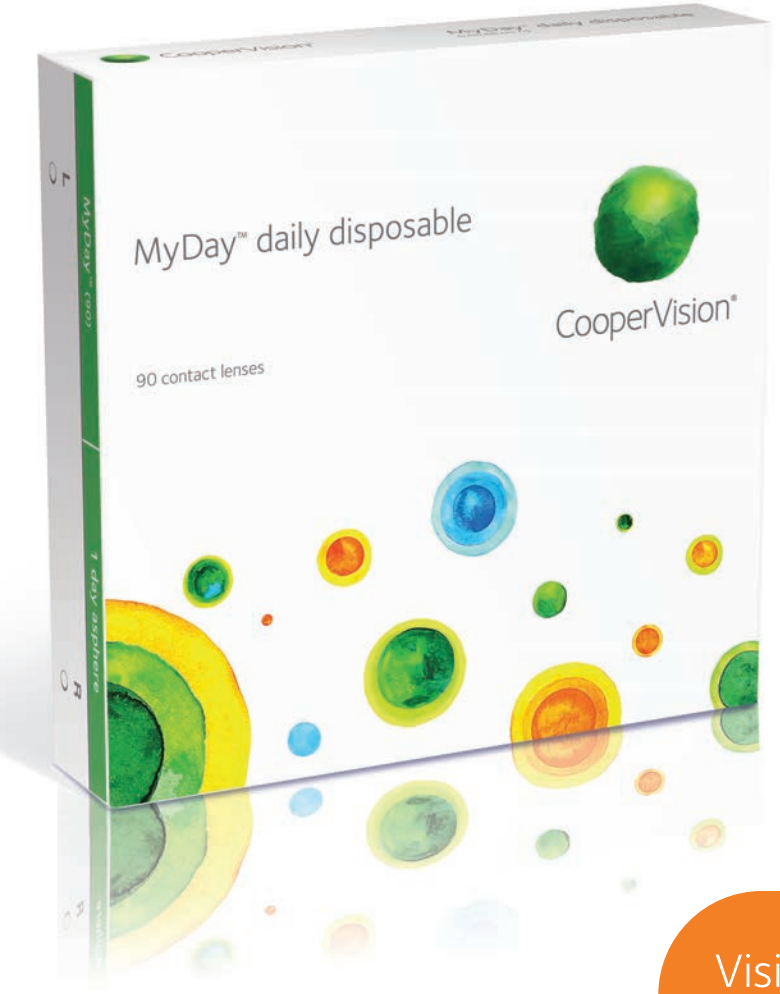

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Sixth Nerve Palsy Prompts a Surprising Diagnosis

Delayed onset of symptoms made this diagnosis tough—but maintaining a suspicion of PMR/GCA put this patient on the road to recovery.

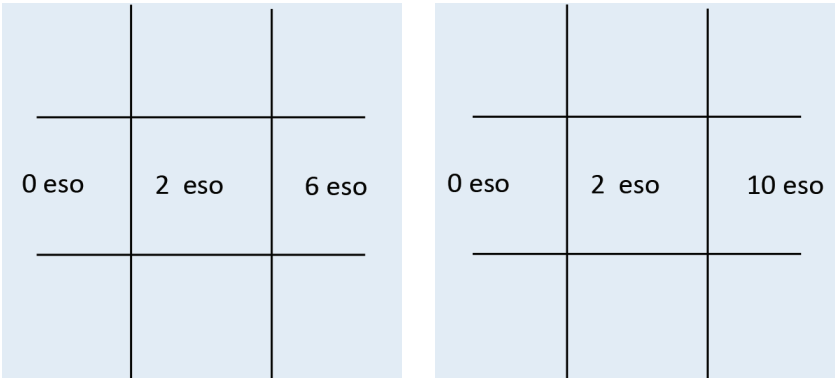
By Michael Trottini, OD, and Michael DelGiodice, OD

A 92-year-old white male presented for evaluation of acute diplopia. He described it as horizontal, worse when looking to the left and only noticeable at distance. Pertinent medical history included hypertension, high cholesterol, diabetes, depression and GERD. His current medications included lisinopril, glucophage, atorvastatin, omeprazole and mirtazapine. He denied head pain, scalp tenderness, jaw claudication, weakness or increased fatigue.

Best corrected visual acuity was 20/25 OU. His extraocular motilities appeared full in all gazes. Pupils were equal, round and reactive to light with no afferent pupillary defect. Cover test was ortho in right gaze, a 2PD ET in primary gaze and a 6ET in left gaze consistent with a left sixth nerve palsy.

Anterior segment exam was unremarkable. He was pseudophakic. Cup-to-disc ratio was 0.2/0.2 OU with no disc edema, pallor or hemorrhage noted. His retinal exam was otherwise unremarkable.

Given his age, isolated sixth nerve palsy and vasculopathic risk factors, we decided to follow up in one month without any additional testing. Shortly after, the patient was hospitalized for unspecified rectal bleeding. He was in and out of the hospital and wasn't able to follow up until approximately three months later. At that follow up, he reported arm and leg weakness and increased fatigue. Upon questioning he admitted to facial pains located around his temples that started a few days prior. His sixth nerve palsy and cover test measurements were slightly worse (ortho in right gaze, 2PD ET in primary gaze and 10PD ET in left gaze). His vision was stable and there was no disc edema or signs of ischemic optic neuropathy. Because of his symptoms, we ordered a STAT ESR and CRP, which measured 62 and 8.0, respectively. The patient was referred to his internist for evaluation of polymyalgia rheumatica (PMR) and giant cell arteritis (GCA). Based on his constitutional symptoms, worsening sixth nerve palsy, elevated ESR and CRP, he was tentatively diagnosed with vasculitis-induced ocular motor palsy and treated with 40mg of prednisone. A temporal artery biopsy to confirm GCA was deferred due to his age. He returned for a follow up ophthalmic examination one week later and reported that the diplopia had resolved three days after starting the prednisone, and he was feeling significantly better. His cover test measurements were ortho in right, primary and left gazes. He has been tapering the prednisone per his internist's advice. As of our last visit, his ESR was 22 and his sixth nerve palsy was completely resolved.



The patient presented with an esotropia worse on left gaze consistent with a left sixth nerve palsy (left). On follow up exam, the sixth nerve palsy worsened (right).

Discussion

PMR is a chronic inflammatory disorder usually seen in patients over 50.[1] It primarily affects proximal

ADD SIMBRINZA® Suspension to a PGA for Even Lower IOP[1]*

INDICATIONS AND USAGE

SIMBRINZA® (brinzolamide/brimonidine 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 or ocular hypertension.

Dosage and Administration

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 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.

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

Sulfonamide Hypersensitivity Reactions—Brinzolamide is a sulfonamide, and although administered topically, is absorbed systemically. Sulfonamide attributable adverse reactions may occur. Fatalities have occurred due to severe reactions to sulfonamides. Sensitization may recur when a sulfonamide is readministered irrespective of the route of administration. If signs of serious reactions or hypersensitivity occur, discontinue the use of this preparation.

Corneal Endothelium—There is an increased potential for developing corneal edema in patients with low endothelial cell counts.

Severe Hepatic or Renal Impairment (CrCl <30 mL/min)—SIMBRINZA® Suspension has not been specifically studied in these patients and is not recommended.

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, had 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.

Prescribe SIMBRINZA® Suspension as adjunctive therapy to a PGA for appropriate patients

SIMBRINZA® Suspension should be taken at least five (5) minutes apart from other topical ophthalmic drugs

Learn more at myalcon.com/simbrinza

For additional information about SIMBRINZA® Suspension, please see Brief Summary of full Prescribing Information on adjacent page.

Reference: 1. Data on file, 2014.

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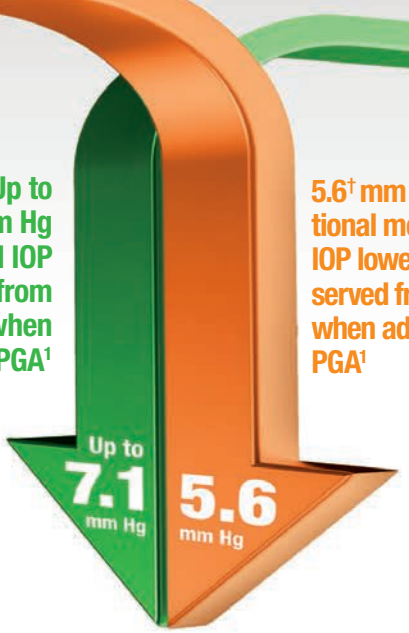
Up to 7.1 mm Hg additional IOP reduction from baseline when added to a PGA[1]

5.6† mm Hg additional mean diurnal IOP lowering observed from baseline when added to a PGA[1]



		IOP Time Points (mm Hg)[1‡]			
Treatment Arm		8 AM	10 AM	3 PM	5 PM
PGA + SIMBRINZA® Suspension (N=83)	Baseline§	24.5	22.9	21.7	21.6
	Week 6	19.4	15.8	17.2	15.6
PGA + Vehicle (N=92)	Baseline§	24.3	22.6	21.3	21.2
	Week 6	21.5	20.3	20.0	20.1

‡Least squares means at each Week 6 time point. Treatment differences (mm Hg) and *P*-values at Week 6 time points between treatment groups were: -2.14, *P*=0.0002; -4.56, *P*<0.0001; -2.84, *P*<0.0001; -4.42, *P*<0.0001.
§Baseline (PGA Monotherapy).

		Mean Diurnal IOP (mm Hg)[1‖]
Treatment Arm		
PGA + SIMBRINZA® Suspension (N=83)	Baseline¶	22.7
	Week 6	17.1
PGA + Vehicle (N=92)	Baseline¶	22.4
	Week 6	20.5

‖Treatment difference (mm Hg) and *P*-value at Week 6 was -3.4, *P*<0.0001.
¶Baseline (PGA Monotherapy).

Study Design: A prospective, randomized, multicenter, double-blind, parallel-group study of 189 patients with open-angle glaucoma and/or ocular hypertension receiving treatment with a PGA. PGA treatment consisted of either travoprost, latanoprost, or bimatoprost. Patients in the study were randomized to adjunctive treatment with SIMBRINZA® Suspension (N=88) or vehicle (N=94). The primary efficacy endpoint was mean diurnal IOP (IOP averaged over all daily time points) at Week 6 between treatment groups. Key secondary endpoints included IOP at Week 6 for each daily time point (8 AM, 10 AM, 3 PM, and 5 PM) and mean diurnal IOP change from baseline to Week 6 between treatment groups.[1]

*PGA study-group treatment consisted of either travoprost, latanoprost, or bimatoprost.
†Treatment difference (mm Hg) and *P*-value at Week 6 was -3.7, *P*<0.0001.

SIMBRINZA®
(brinzolamide/brimonidine tartrate ophthalmic suspension) 1%/0.2%

BRIEF SUMMARY OF PRESCRIBING INFORMATION
INDICATIONS AND USAGE

SIMBRINZA® (brinzolamide/brimonidine tartrate ophthalmic suspension) 1%/0.2% is a fixed combination of a carbonic anhydrase inhibitor and an alpha 2 adrenergic receptor agonist indicated for the reduction of elevated intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension.

DOSAGE AND ADMINISTRATION

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 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.

DOSAGE FORMS AND STRENGTHS

Suspension containing 10 mg/mL brinzolamide and 2 mg/mL brimonidine tartrate.

CONTRAINDICATIONS

Hypersensitivity - SIMBRINZA® Suspension is contraindicated in patients who are hypersensitive to any component of this product.

Neonates and Infants (under the age of 2 years) - SIMBRINZA® Suspension is contraindicated in neonates and infants (under the age of 2 years*) see Use in Specific Populations*

WARNINGS AND PRECAUTIONS

Sulfonamide Hypersensitivity Reactions - SIMBRINZA® Suspension contains brinzolamide, a sulfonamide, and although administered topically is absorbed systemically. Therefore, the same types of adverse reactions that are attributable to sulfonamides may occur with topical administration of SIMBRINZA® Suspension. Fatalities have occurred due to severe reactions to sulfonamides including Stevens-Johnson syndrome, toxic epidermal necrolysis, fulminant hepatic necrosis, agranulocytosis, aplastic anemia, and other blood dyscrasias. Sensitization may recur when a sulfonamide is re-administered irrespective of the route of administration. If signs of serious reactions or hypersensitivity occur, discontinue the use of this preparation *[see Patient Counseling Information]*

Corneal Endothelium - Carbonic anhydrase activity has been observed in both the cytoplasm and around the plasma membranes of the corneal endothelium. There is an increased potential for developing corneal edema in patients with low endothelial cell counts. Caution should be used when prescribing SIMBRINZA® Suspension to this group of patients.

Severe Renal Impairment - SIMBRINZA® Suspension has not been specifically studied in patients with severe renal impairment (CrCl < 30 mL/min). Since brinzolamide and its metabolite are excreted predominantly by the kidney, SIMBRINZA® Suspension is not recommended in such patients.

Acute Angle-Closure Glaucoma - The management of patients with acute angle-closure glaucoma requires therapeutic interventions in addition to ocular hypotensive agents. SIMBRINZA® Suspension has not been studied in patients with acute angle-closure glaucoma.

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 *[see Patient Counseling Information]*.

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.

Severe Hepatic Impairment - Because brimonidine tartrate, a component of SIMBRINZA® Suspension, has not been studied in patients with hepatic impairment, caution should be exercised in such patients.

Potentiation of Vascular Insufficiency - Brimonidine tartrate, a component of SIMBRINZA® Suspension, may potentiate syndromes associated with vascular insufficiency. SIMBRINZA® Suspension should be used with caution in patients with depression, cerebral or coronary insufficiency, Raynaud's phenomenon, orthostatic hypotension, or thromboangiitis obliterans.

Contamination of Topical Ophthalmic Products After Use - There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products. These containers have been inadvertently contaminated by patients who, in most cases, had a concurrent corneal disease or a disruption of the ocular epithelial surface *[see Patient Counseling Information]*.

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 the rates in the clinical studies of another drug and may not reflect the rates observed in practice.

SIMBRINZA® Suspension - In two clinical trials of 3 months duration 435 patients were treated with SIMBRINZA® Suspension, and 915 were treated with the two individual components. The most frequently reported adverse reactions in patients treated with SIMBRINZA® Suspension occurring in approximately 3 to 5% of patients in descending order of incidence were blurred vision, eye irritation, dysgeusia (bad taste), dry mouth, and eye allergy. Rates of adverse reactions reported with the individual components were comparable. Treatment discontinuation, mainly due to adverse reactions, was reported in 11% of SIMBRINZA® Suspension patients.

Other adverse reactions that have been reported with the individual components during clinical trials are listed below.

Brinzolamide 1% - In clinical studies of brinzolamide ophthalmic suspension 1%, the most frequently reported adverse reactions reported in 5 to 10% of patients were blurred vision and bitter, sour or unusual taste. Adverse reactions occurring in 1 to 5% of patients were blepharitis, dermatitis, dry eye, foreign body sensation, headache, hyperemia, ocular discharge, ocular discomfort, ocular keratitis, ocular pain, ocular pruritus and rhinitis.

The following adverse reactions were reported at an incidence below 1%: allergic reactions, alopecia, chest pain, conjunctivitis, diarrhea, diplopia, dizziness, dry mouth, dyspnea, dyspepsia, eye fatigue, hypertonia, keratoconjunctivitis, keratopathy, kidney pain, lid margin crusting or sticky sensation, nausea, pharyngitis, tearing and urticaria.

Brimonidine Tartrate 0.2% - In clinical studies of brimonidine tartrate 0.2%, adverse reactions occurring in approximately 10 to 30% of the subjects, in descending order of incidence, included oral dryness, ocular hyperemia, burning and stinging, headache, blurring, foreign body sensation, fatigue/drowsiness, conjunctival follicles, ocular allergic reactions, and ocular pruritus.

Reactions occurring in approximately 3 to 9% of the subjects, in descending order included corneal staining/erosion, photophobia, eyelid erythema, ocular ache/pain, ocular dryness, tearing, upper respiratory symptoms, eyelid edema, conjunctival edema, dizziness, blepharitis, ocular irritation, gastrointestinal symptoms, asthenia, conjunctival blanching, abnormal vision and muscular pain.

The following adverse reactions were reported in less than 3% of the patients: lid crusting, conjunctival hemorrhage, abnormal taste, insomnia, conjunctival discharge, depression, hypertension, anxiety, palpitations/arrhythmias, nasal dryness and syncope.

Postmarketing Experience - The following reactions have been identified during postmarketing use of brimonidine tartrate ophthalmic solutions in clinical practice. Because they are reported voluntarily from a population of unknown size, estimates of frequency cannot be made. The reactions, which have been chosen for inclusion due to either their seriousness, frequency of reporting, possible causal connection to brimonidine tartrate ophthalmic solutions, or a combination of these factors, include: bradycardia, hypersensitivity, iritis, keratoconjunctivitis sicca, miosis, nausea, skin reactions (including erythema, eyelid pruritus, rash, and vasodilation), and tachycardia.

Apnea, bradycardia, coma, hypotension, hypothermia, hypotonia, lethargy, pallor, respiratory depression, and somnolence have been reported in infants receiving brimonidine tartrate ophthalmic solutions *[see Contraindications]*.

DRUG INTERACTIONS

Oral Carbonic Anhydrase Inhibitors - There is a potential for an additive effect on the known systemic effects of carbonic anhydrase inhibition in patients receiving an oral carbonic anhydrase inhibitor and brinzolamide ophthalmic suspension 1%, a component of SIMBRINZA® Suspension. The concomitant administration of SIMBRINZA® Suspension and oral carbonic anhydrase inhibitors is not recommended.

High-Dose Salicylate Therapy - Carbonic anhydrase inhibitors may produce acid-base and electrolyte alterations. These alterations were not reported in the clinical trials with brinzolamide ophthalmic suspension 1%. However, in patients treated with oral carbonic anhydrase inhibitors, rare instances of acid-base alterations have occurred with high-dose salicylate therapy. Therefore, the potential for such drug interactions should be considered in patients receiving SIMBRINZA® Suspension.

CNS Depressants - Although specific drug interaction studies have not been conducted with SIMBRINZA® Suspension, the possibility of an additive or potentiating effect with CNS depressants (alcohol, opiates, barbiturates, sedatives, or anesthetics) should be considered.

Antihypertensives/Cardiac Glycosides - Because brimonidine tartrate, a component of SIMBRINZA® Suspension, may reduce blood pressure, caution in using drugs such as antihypertensives and/or cardiac glycosides with SIMBRINZA® Suspension is advised.

Tricyclic Antidepressants - Tricyclic antidepressants have been reported to blunt the hypotensive effect of systemic clonidine. It is not known whether the concurrent use of these agents with SIMBRINZA® Suspension in humans can lead to resulting interference with the IOP lowering effect. Caution is advised in patients taking tricyclic antidepressants which can affect the metabolism and uptake of circulating amines.

Monoamine Oxidase Inhibitors - Monoamine oxidase (MAO) inhibitors may theoretically interfere with the metabolism of brimonidine tartrate and potentially result in an increased systemic side-effect such as hypotension. Caution is advised in patients taking MAO inhibitors which can affect the metabolism and uptake of circulating amines.

USE IN SPECIFIC POPULATIONS

Pregnancy - *Pregnancy Category C:* Developmental toxicity studies with brinzolamide in rabbits at oral doses of 1, 3, and 6 mg/kg/day (20, 60, and 120 times the recommended human ophthalmic dose) produced maternal toxicity at 6 mg/kg/day and a significant increase in the number of fetal variations, such as accessory skull bones, which was only slightly higher than the historic value at 1 and 6 mg/kg. In rats, statistically decreased body weights of fetuses from dams receiving oral doses of 18 mg/kg/day (180 times the recommended human ophthalmic dose) during gestation were proportional to the reduced maternal weight gain, with no statistically significant effects on organ or tissue development. Increases in unossified sternebrae, reduced ossification of the skull, and unossified hyoid that occurred at 6 and 18 mg/kg were not statistically significant. No treatment-related malformations were seen. Following oral administration of ^{14}C-brinzolamide to pregnant rats, radioactivity was found to cross the placenta and was present in the fetal tissues and blood.

Developmental toxicity studies performed in rats with oral doses of 0.66 mg brimonidine base/kg revealed no evidence of harm to the fetus. Dosing at this level resulted in a plasma drug concentration approximately 100 times higher than that seen in humans at the recommended human ophthalmic dose. In animal studies, brimonidine crossed the placenta and entered into the fetal circulation to a limited extent.

There are no adequate and well-controlled studies in pregnant women. SIMBRINZA® Suspension should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers - In a study of brinzolamide in lactating rats, decreases in body weight gain in offspring at an oral dose of 15 mg/kg/day (150 times the recommended human ophthalmic dose) were observed during lactation. No other effects were observed. However, following oral administration of ^{14}C-brinzolamide to lactating rats, radioactivity was found in milk at concentrations below those in the blood and plasma. In animal studies, brimonidine was excreted in breast milk.

It is not known whether brinzolamide and brimonidine tartrate are excreted in human milk following topical ocular administration. Because many drugs are excreted in human milk and because of the potential for serious adverse reactions in nursing infants from SIMBRINZA® (brinzolamide/brimonidine tartrate ophthalmic suspension) 1%/0.2%, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use - The individual component, brinzolamide, has been studied in pediatric glaucoma patients 4 weeks to 5 years of age. The individual component, brimonidine tartrate, has been studied in pediatric patients 2 to 7 years old. Somnolence (50-83%) and decreased alertness was seen in patients 2 to 6 years old. SIMBRINZA® Suspension is contraindicated in children under the age of 2 years *[see Contraindications]*.

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

OVERDOSAGE

Although no human data are available, electrolyte imbalance, development of an acidotic state, and possible nervous system effects may occur following an oral overdose of brinzolamide. Serum electrolyte levels (particularly potassium) and blood pH levels should be monitored.

Very limited information exists on accidental ingestion of brimonidine in adults; the only adverse event reported to date has been hypotension. Symptoms of brimonidine overdose have been reported in neonates, infants, and children receiving brimonidine as part of medical treatment of congenital glaucoma or by accidental oral ingestion. Treatment of an oral overdose includes supportive and symptomatic therapy; a patent airway should be maintained.

PATIENT COUNSELING INFORMATION

Sulfonamide Reactions - Advise patients that if serious or unusual ocular or systemic reactions or signs of hypersensitivity occur, they should discontinue the use of the product and consult their physician.

Temporary Blurred Vision - Vision may be temporarily blurred following dosing with SIMBRINZA® Suspension. Care should be exercised in operating machinery or driving a motor vehicle.

Effect on Ability to Drive and Use Machinery - As with other drugs in this class, SIMBRINZA® Suspension may cause fatigue and/or drowsiness in some patients. Caution patients who engage in hazardous activities of the potential for a decrease in mental alertness.

Avoiding Contamination of the Product - Instruct patients that ocular solutions, if handled improperly or if the tip of the dispensing container contacts the eye or surrounding structures, can become contaminated by common bacteria known to cause ocular infections. Serious damage to the eye and subsequent loss of vision may result from using contaminated solutions *[see Warnings and Precautions]*. Always replace the cap after using. If solution changes color or becomes cloudy, do not use. Do not use the product after the expiration date marked on the bottle.

Intercurrent Ocular Conditions - Advise patients that if they have ocular surgery or develop an intercurrent ocular condition (e.g., trauma or infection), they should immediately seek their physician's advice concerning the continued use of the present multidose container.

Concomitant Topical Ocular Therapy - If more than one topical ophthalmic drug is being used, the drugs should be administered at least five minutes apart.

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.

U.S. Patent No:
6,316,441

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muscles and joints and is associated with GCA in 18% to 26% of cases.[2]

The hallmark symptoms of GCA are headache, jaw claudication, scalp tenderness, neck and shoulder pain, as well as fatigue and weakness. Although PMR often begins prior to the onset of GCA, it can be difficult to predict its conversion since up to 50% of PMR patients, even without headache, have been reported to have a positive temporal artery biopsy.[2] While symptoms of transient ischemia have been described in PMR patients, they suggest progression to GCA.

With exclusion of patients receiving low-dose systemic steroids for PMR, symptoms of isolated transient ischemia without concurrent headache or other systemic complaints in a patient with GCA are uncommon. However, atypical cases have been reported. The most unusual scenarios involve patients with a normal ESR, no systemic complaints and a positive cranial neuropathy, or patients with ophthalmologic or neurologic findings in the absence of systemic complaints and a normal ESR.[1] Other rare presentations of GCA include acute internal carotid artery, middle cerebral artery, posterior cerebral artery and basilar artery transient ischemic attacks.[1] Although rare, cranial nerve palsies, especially the oculomotor nerve and abducens nerve, have been associated with GCA.[3-7] Researchers believe this is a result of arteritis affecting the blood supply to the nerve.[8] GCA prevalence in individuals over 50 is 133 in 100,000; however, the prevalence increases to 843 in 100,000 in individuals older than 80.[1]

Our patient's presentation was atypical because he didn't develop symptoms of PMR/GCA until three months after presenting with the sixth nerve palsy. Because of the immediate resolution of his diplopia and nerve palsy after starting prednisone, we feel this was related to vasculitis and not microvascular. It was most likely a lower grade inflammation exacerbated by his medical issues and hospitalizations.

Although most isolated nerve palsies are microvascular, the clinician should maintain a suspicion for other etiologies. No general consensus exists regarding management of isolated sixth nerve palsies in individuals over 50. Some authors recommend monthly observation for up to three months before considering neuroimaging and laboratory studies (assuming there are no other neurologic deficits or symptoms suggestive of a process other than microvascular disease).[9] Others recommend neuroimaging and laboratory studies at initial examination.[3,5]

We typically do not order an ESR or CRP in individuals with isolated third or sixth nerve palsies unless symptoms to suggest GCA. Apply good clinical judgment when managing these cases. Test ESR and CRP in patients over 50 presenting with sixth nerve palsies when suspicion of GCA exists or if the nerve palsy worsens or fails to resolve.

After managing this patient and because the prevalence of GCA increases exponentially after age 80, we believe this increased prevalence is high enough to warrant early serology, including an urgent ESR and CRP despite the isolated nature of the palsy or any pre-existing vascular risk factors.[10] We recommend obtaining these tests as a precaution in this subset population to avoid a delay in diagnosis since ophthalmologic involvement, including ocular motor paresis, unilateral visual loss, or both, will develop in 17% to 55% of untreated patients.[1] ■

Table 2. EULAR-ACR Classification Criteria for Polymyalgia Rheumatica*

Giant Cell Arteritis (GCA)	Polymyalgia Rheumatica (PMR)
• Age over 50 years • New onset of localized headache • Abnormality of temporal artery (temporal artery tenderness, reduced pulsation) • Raised ESR (≥ 50mm/1st hour) • Abnormal arterial biopsy (vasculitis with predominantly monocular cell infiltration or granulomatous inflammation or evidence of giant cells)	• Morning stiffness >45 minutes (2 points) • Rheumatic factor and/or anti-CCP-antibodies negative (2 points) • Pelvic girdle pain or reduced hip mobility (1 point) • No other painful joint (1 point) • Ultrasound: inflammatory changes in both shoulders (including subdeltoid bursitis) (1 point) • Ultrasound: inflammatory changes in at least one shoulder and hip joint (1 point)

*Classification as PMR requires 4 points; for giant cell arteritis (GCA), three criteria for the classification of GCA must be fulfilled.[1]
Modified from EULAR, European League Against Rheumatism; ACR, American College of Rheumatology; ESR, erythrocyte sedimentation rate; CCP, anti-cyclic citrullinated peptide antibodies.[2]

1. Kupersmith MJ, Berenstein A. Neuro-vascular neuro-ophthalmology. Springer-Verlag Berline; Heidelberg, 1993.
2. Dasgupta B, Cimmino MA, Maradit-Kremers H, et al. 2012 provisional classification criteria for polymyalgia rheumatica: a European League Against Rheumatism/American College of Rheumatology collaborative initiative. Ann Rheum Dis. 2012;71:484-92.
3. Caylor T, Perkins A. Recognition and management of polymyalgia rheumatica and giant cell arteritis. Am Fam Physician. 2013 Nov;88(10):676-84.
4. Tamhankar M, et al. Isolated third, fourth, and sixth cranial nerve palsies from presumed microvascular versus other causes. Ophthalmology. 2013 Nov;120(11):2264-9.
5. Arai M, Katsumata R. Temporal arteritis presenting with headache and abducens nerve palsy. Rinsho Shinkeigaku. 2007 Jul;47(7):444-6.
6. Thurtell M, et al. Third nerve palsy as the initial manifestation of giant cell arteritis. J Neuro-Ophthalmol. 2014;34:243-5.
7. Davies GE, Shakir RA. Giant cell arteritis presenting as oculomotor nerve palsy with pupillary dilation. Postgrad Med J. 1994 Apr;70(822):298–9.
8. Asensio-Sanchez VM, et al. Third nerve palsy as the only manifestation of occult temporal arteritis. Arch Soc Esp Oftalmol. 2009;84:395-8.
9. Fisher CM. Ocular palsy in temporal arteritis. Minn Med. 1959;42:1258-68.
10. Goodwin D. Differential diagnosis and management of acquired sixth cranial nerve palsy. Optometry. 2006 Nov;77(11):534-9.

Finding MGD in DED

Are you specifically looking for meibomian gland dysfunction? You should be, and here's why. **By Paul M. Karpecki, OD**

A better understanding of meibomian gland dysfunction (MGD) has been key to our ability to more effectively manage dry eye disease in the last decade.[1]

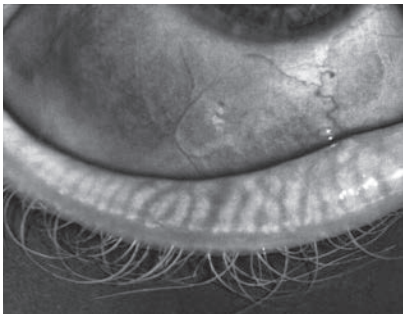
When I started my first dedicated dry eye clinic, it wasn't identified regularly, and perhaps that is why patients weren't achieving high success with their dry eye disease management. And even if we knew to look for and grade MGD, few effective treatments existed then. Screening for and uncovering the presence of MGD in patients, looking at the components of the disease and then choosing effective treatments are integral to today's success in the management of this common cause of dry eye disease.

MGD can result in significant and progressive dry eye disease that can, in turn, lead to severe atrophy of the meibomian glands. It also may be a leading cause of contact lens dropout, whether by affecting blink rates or adding stress to the required meibomian gland output.[2,3]

If clinicians are not looking for MGD and associated complications, they can't take measures to prevent patients from abandoning contact lens use or seeking another eye doctor. MGD can contribute to inaccurate biometry measurements for IOL calculations and unexpected outcomes following refractive surgery.

Identifying MGD

Every exam should include a close look at the meibomian glands and their expression characteristics.



A 28-year-old female patient showing early signs of MGD.

When examining the lower eyelid in particular—which is responsible for the majority of oil in the tear film—look for notching, which is a sign of gland atrophy; froth in the tear film, which is an indication of meibomian gland dysfunction; telangiectasia; tylosis or thickening of the eyelids; and hyperemia. Next, use a wet cotton-tip applicator, the Meibomian Gland Evaluator (TearScience) or an expression paddle such as the Mastrota Paddle (OcuSoft) to express the area of the nasal to central lower eyelid glands. Note how many glands express and the quality of the meibum. Healthy meibum is clear like olive oil and easily expresses, barely noticeable when rolling off the eyelid. A turbid expression or paste-like or non-expressive glands are indicative of further progression of the disease.[4]

Other tests that help with a diagnosis include osmolarity testing, patient questionnaires such as the SPEED or OSDI questionnaire, blink analysis and especially meibography. Meibography can reveal the effects on the structure of the gland and show areas of atrophy, aiding in the diagnosis and severity of the disease.[5]

MGD Components and Effective Management

Although the mechanism underlying MGD is likely multifactorial and complex ranging from androgen deficiency to evaporative stress and low humidity environments, MGD itself begins with obstruction of the meibomian glands that leads to further sequelae which may need to be managed.[6,7] Although not every case has all four components, the other sequelae include inflammation, biofilm development and tear film disruption or instability.[8] Here are each of the four components and their respective treatment options:

Obstruction. When obstruction occurs, it may cause the other glands to up-regulate or over-work to make up for the ones that are not functioning. This leads to further stress and, eventually, gland atrophy. If a gland is dysfunctional for a period of time, it may also get a keratin covering that further prevents its ability to produce oils for the tear film.[9]

While treatment options may include scaling the lower eyelid to remove keratin, the key to obstruction removal is thermal treatments that can soften the meibum over time and promote proper gland expression. One technology that combines both is the LipiFlow thermal-pulsation system (TearScience). You can get a significant effect in a relatively short treatment time by avoiding the

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INDICATION

LUMIGAN® (bimatoprost ophthalmic solution) 0.01% is indicated for the reduction of elevated intraocular pressure in patients with open angle glaucoma or ocular hypertension.

IMPORTANT SAFETY INFORMATION

WARNINGS AND PRECAUTIONS

LUMIGAN® 0.01% causes changes to pigmented tissues, mostly increased pigmentation of the iris, eyelid, and eyelashes as long as LUMIGAN® 0.01% is administered. Iris color change may not be noticeable for several months to years. After discontinuation of bimatoprost, iris pigmentation is likely to be permanent, while eyelid and eyelash changes have been reported to be reversible in some patients. Patients should be informed of the possibility of increased pigmentation. The long term effects of increased pigmentation are not known.

Prostaglandin analogs, including bimatoprost, have been reported to cause intraocular inflammation. These products may also exacerbate inflammation, so use with caution in patients with active intraocular inflammation (e.g., uveitis).

Macular edema, including cystoid macular edema, has been reported with LUMIGAN® 0.01%. LUMIGAN® 0.01% should be used with caution in aphakic patients, in pseudophakic patients with a torn posterior lens capsule, or in patients with known risk factors for macular edema. Remove contact lenses prior to instillation of LUMIGAN® 0.01% and reinsert after 15 minutes.

ADVERSE REACTIONS

The most common adverse reaction was conjunctival hyperemia (31%). Approximately 1.6% of patients discontinued therapy due to conjunctival hyperemia. Other adverse drug reactions (reported in 1 to 4% of patients) with LUMIGAN® 0.01% included conjunctival edema, conjunctival hemorrhage, eye irritation, eye pain, eye pruritus, erythema of eyelid, eyelids pruritus, growth of eyelashes, hypertrichosis, instillation site irritation, punctate keratitis, skin hyperpigmentation, vision blurred, and visual acuity reduced.

Please see Brief Summary of the full Prescribing Information on adjacent page.

1. US Food and Drug Administration. Drugs@FDA. Drug details: LUMIGAN® 0.01%. FDA website: www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm. Accessed May 19, 2015. **2.** Katz LJ, Cohen JS, Batoosingh AL, Felix C, Shu V, Schiffman RM. Twelve-month, randomized, controlled trial of bimatoprost 0.01%, 0.0125%, and 0.03% in patients with glaucoma or ocular hypertension. *Am J Ophthalmol.* 2010;149(4):661-671. **3.** IMS Health, Inc. *Vector One®: National* (VONA). April 2015.

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Brief Summary—Please see the LUMIGAN® 0.01% package insert for full Prescribing Information.

INDICATIONS AND USAGE

LUMIGAN® (bimatoprost ophthalmic solution) 0.01% is indicated for the reduction of elevated intraocular pressure in patients with open angle glaucoma or ocular hypertension.

CONTRAINDICATIONS

None

WARNINGS AND PRECAUTIONS

Pigmentation: Bimatoprost ophthalmic solution has been reported to cause changes to pigmented tissues. The most frequently reported changes have been increased pigmentation of the iris, periorbital tissue (eyelid) and eyelashes. Pigmentation is expected to increase as long as bimatoprost is administered. The pigmentation change is due to increased melanin content in the melanocytes rather than to an increase in the number of melanocytes. After discontinuation of bimatoprost, 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. 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 nevi nor freckles of the iris appear to be affected by treatment. While treatment with **LUMIGAN®** (bimatoprost ophthalmic solution) 0.01% can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly *[see Patient Counseling Information (17.1)]*.

Eyelash Changes: LUMIGAN® 0.01% 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: Prostaglandin analogs, including bimatoprost, have been reported to cause intraocular inflammation. In addition, because these products may exacerbate inflammation, caution should be used in patients with active intraocular inflammation (e.g., uveitis).

Macular Edema: Macular edema, including cystoid macular edema, has been reported during treatment with bimatoprost ophthalmic solution. **LUMIGAN®** 0.01% should be used with caution in aphakic patients, in pseudophakic patients with a torn posterior lens capsule, or in patients with known risk factors for macular edema.

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 *[see Patient Counseling Information (17.3)]*.

Use with Contact Lenses: Contact lenses should be removed prior to instillation of **LUMIGAN®** 0.01% 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.

In a 12-month clinical study with bimatoprost ophthalmic solutions 0.01%, the most common adverse reaction was conjunctival hyperemia (31%). Approximately 1.6% of patients discontinued therapy due to conjunctival hyperemia. Other adverse drug reactions (reported in 1 to 4% of patients) with **LUMIGAN®** 0.01% in this study included conjunctival edema, conjunctival hemorrhage, eye irritation, eye pain, eye pruritus, erythema of eyelid, eyelids pruritus, growth of eyelashes, hypertrichosis, instillation site irritation, punctate keratitis, skin hyperpigmentation, vision blurred, and visual acuity reduced.

Postmarketing Experience: The following reaction has been identified during postmarketing use of **LUMIGAN®** 0.01% in clinical practice. Because it was reported voluntarily from a population of unknown size, estimates of frequency cannot be made. The reaction, which has been chosen for inclusion due to either its seriousness, frequency of reporting, possible causal connection to **LUMIGAN®** 0.01%, or a combination of these factors, includes headache.

In postmarketing use with prostaglandin analogs, periorbital and lid changes including deepening of the eyelid sulcus have been observed.

USE IN SPECIFIC POPULATIONS

Pregnancy: Pregnancy Category C

Teratogenic effects: In embryo/fetal developmental studies in pregnant mice and rats, abortion was observed at oral doses of bimatoprost which achieved at least 33 or 97 times, respectively, the maximum intended human exposure based on blood AUC levels.

At doses at least 41 times the maximum intended human exposure based on blood AUC levels, the gestation length was reduced in the dams, the incidence of dead fetuses, late resorptions, peri- and postnatal pup mortality was increased, and pup body weights were reduced.

There are no adequate and well-controlled studies of **LUMIGAN®** (bimatoprost ophthalmic solution) 0.01% administration in pregnant women. Because animal reproductive studies are not always predictive of human response **LUMIGAN®** 0.01% should be administered during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers: It is not known whether **LUMIGAN®** 0.01% is excreted in human milk, although in animal studies, bimatoprost has been shown to be excreted in breast milk. Because many drugs are excreted in human milk, caution should be exercised when **LUMIGAN®** 0.01% 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 chronic use.

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

Hepatic Impairment: In patients with a history of liver disease or abnormal ALT, AST and/or bilirubin at baseline, bimatoprost 0.03% had no adverse effect on liver function over 48 months.

OVERDOSAGE

No information is available on overdosage in humans. If overdose with **LUMIGAN®** (bimatoprost ophthalmic solution) 0.01% occurs, treatment should be symptomatic.

In oral (by gavage) mouse and rat studies, doses up to 100 mg/kg/day did not produce any toxicity. This dose expressed as mg/m^2 is at least 210 times higher than the accidental dose of one bottle of **LUMIGAN®** 0.01% for a 10 kg child.

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment of Fertility: Bimatoprost was not carcinogenic in either mice or rats when administered by oral gavage at doses of up to 2 mg/kg/day and 1 mg/kg/day respectively (at least 192 and 291 times the recommended human exposure based on blood AUC levels respectively) for 104 weeks.

Bimatoprost was not mutagenic or clastogenic in the Ames test, in the mouse lymphoma test, or in the *in vivo* mouse micronucleus tests.

Bimatoprost did not impair fertility in male or female rats up to doses of 0.6 mg/kg/day (at least 103 times the recommended human exposure based on blood AUC levels).

PATIENT COUNSELING INFORMATION

Potential for Pigmentation: Advise patients about the potential for increased brown pigmentation of the iris, which may be permanent. Also inform patients about the possibility of eyelid skin darkening, which may be reversible after discontinuation of **LUMIGAN®** (bimatoprost ophthalmic solution) 0.01%.

Potential for Eyelash Changes: Inform patients of the possibility of eyelash and vellus hair changes in the treated eye during treatment with **LUMIGAN®** 0.01%. These changes may result in a disparity between eyes in length, thickness, pigmentation, number of eyelashes or vellus hairs, and/or direction of eyelash growth. Eyelash changes are usually reversible upon discontinuation of treatment.

Handling the Container: Instruct patients to avoid allowing 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: Advise patients 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 **LUMIGAN®** 0.01%.

Use with Contact Lenses: Advise patients that **LUMIGAN®** 0.01% contains benzalkonium chloride, which may be absorbed by soft contact lenses. Contact lenses should be removed prior to instillation of **LUMIGAN®** 0.01% and may be reinserted 15 minutes following its administration.

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

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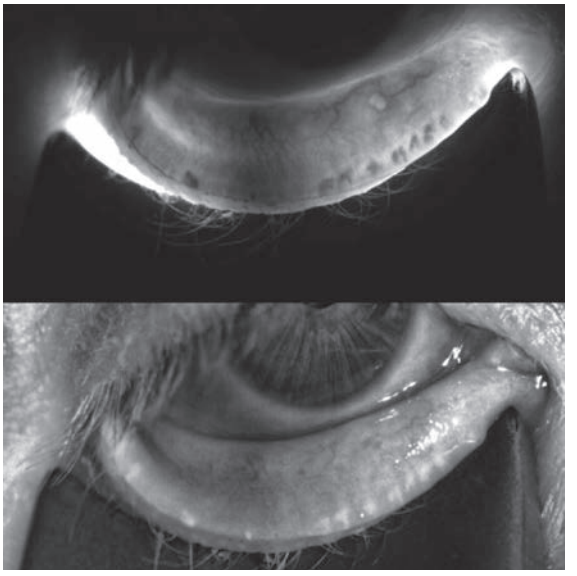
external eyelid and instead providing heat through the back surface of the eyelid.[10] Adding pulsation may further improve the meibum consistency. Other thermal systems that may benefit patients with MGD obstruction include MiboFlo (Mibo Medical Group) and intense pulsed light (IPL) devices. Studies also show potential for infrared heat treatments.[11]

Keep in mind that previous MGD treatments such as 'rice-in-a-sock' simply do not allow adequate penetration of heat because of the limits through the external skin. There are also limitations to a wet washcloth compress, as patients must exchange the washcloths while at a sink for eight to 10 minutes or longer to maintain adequate heat levels. But warm compresses increase the tear film lipid layer if penetrating heat and compliance is in place.[12,13] A new daily compress, the Bruder Eye Hydrating Compress (Bruder Healthcare Company), uses unique beads with a small angstrom opening that, upon microwaving for 15 to 20 seconds, releases hydration that is obtained from the environment. It is reusable, durable, can be washed and provides adequate heat and hydration for approximately 12 to 14 minutes. The hydration aids in transfer of heat to the glands. Other commercial compresses include ThermoEyes (OcuSoft), TheraPearls (Bausch + Lomb) and TranquilEyes (Eye Eco). Note that patients are no longer instructed to massage their eyelids after using compresses, due to potential changes to the cornea and because thermal pulsation systems are now used for that purpose.[14]



A 56-year-old female with endstage MGD manifesting significant gland atrophy/drop out.

Inflammation. Studies show that inflammation can be present in cases of both MGD (meibomitis) and dry eye disease.[15] Inflammation plays a key role in further damage and progression of both diseases because of the constant stress of a poor tear film, friction from the eyelid moving across the ocular surface and obstruction of glands; thus, it is crucial to treat the inflammation. Anti-inflammatory medications known to work include cyclosporine, corticosteroids and corticosteroid-combination agents, oral doxycycline, topical azithromcyin and oral azithromycin and omega fatty acids.[15-21]

Biofilm Formation. Lid hygiene may also play a role in the management of MGD, as biofilms have been known to develop in this disease. A biofilm is an aggregate of bacterial microorganisms that coat a particular living or non-living structure. Biofilms can cause infections or affect structural physiology and may actually become more resistant to treatment than individual bacterial colonies not in a biofilm formation.[22] Studies show that repetitive lid hygiene products such as eyelid cleansers and mechanical cleaning devices such as BlephEx (BlephEx) help minimize the negative effects of biofilms.[23-25]

Tear Film Instability. Without an adequate lipid component to the tear film, evaporation ensues. Evaporation causes increased meibocyte production that is greater than the oil production, further obstructing the glands and increasing tear film instability.[26] Patients with tear film alterations have dry eye and require artificial tears to supplement the tear film. Cyclosporine is a possible treatment option for tear film instability.

MGD is a critical disease that results in chronic dry eye, and the progression of the disease ultimately results in complete MG atrophy. It likely plays a key role in contact lens drop out, unexpected postsurgical complications and overall quality of vision. By looking at the components of the disease—including obstruction, inflammation, biofilm formation and tear film instability—clinicians can effectively manage and provide patients with adequate treatment, symptomatic relief and an improved quality of life.[26] ■

Dr. Karpecki has a financial relationship with AcuFocus, AMO, Alcon Labs, Allergan, Akorn, Bausch + Lomb/Valeant, BioTissue, Bruder Healthcare, Cambium Pharmaceuticals, Eleven Biotherapeutics, Eyemaginations, Essilor, Fera Pharmaceuticals, Focus Laboratories, iCare USA, Ocusoft, Freedom Meditech, Konan Medical, Beaver-Visitech, Eye Solutions, Reichert, Shire Pharmaceuticals, RySurg, Science Based Health, SightRisk, TearLab, TearScience, TLC Vision, Topcon and Vmax.

1. Lemp MA, Nichols KK. Blepharitis in the United States 2009: a survey- based perspective on prevalence and treatment. Ocul Surf. 2009;7(2 suppl):S1-14.
2. Foulks GN, Borchman D. Meibomian gland dysfunction: the past, present, and future. Eye Contact Lens. 2010;36(5):249-53.

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3. Jansen ME, Begley CG, Himebaugh NH. Effect of contact lens wear and a near task on tear film break-up. Optom Vis Sci. 2010 May;87(5):350-7.
4. Horn MM, Silverman MW. Displacement technique and meibomian gland expression. J Am Optom Assoc. 1987 Mar;58(3):223-6.
5. Srinivasan S, Menzies K, Sorbara L, et al. Infrared imaging of meibomian gland structure using a novel keratograph. Optom Vis Sci. 2012 May;89(5):788-94.
6. Sullivan DA, Sullivan BD, Evans JE, et al. Androgen deficiency, meibomian gland dysfunction and evaporative dry eye. Ann N Y Acad Sci. 1999 June;876:312-24.
7. Suhalim JL, Parfitt GJ, Xie Y, et al. Effect of desiccating stress on mouse meibomian gland function. Ocul Surf. 2014 Jan;12(1):59-68.
8. Pflugfelder SC, Karpecki PM, Perez VL. Treatment of blepharitis: recent clinical trials. The Ocular Surface. 2014 October;12(4).
9. Ong BL, HOdson SA, Wigham T, et al. Evidence for keratin proteins in normal and abnormal human meibomian fluids. Curr Eye Res. 1991 Dec;10(12):1113-9.
10. Finis D, Hayajneh J, König C, et al. Evaluation of an automated thermodynamic treatment (LipiFlow) system for meibomian gland dysfunction: a prospective, randomized, observer-masked trial. Ocul Surf. 2014 Apr;12(2):146-54.
11. Goto E, Monden Y, Takano Y, et al. Treatment of non-inflamed obstructive meibomian gland dysfunction by an infrared warm compression device. Br J Ophthalmol. 2002.
12. Olson MC, Korb DR, Greiner JV. Increase in tear film lipid layer thickness following treatment with warm compresses in patients with meibomian gland dysfunction. Eye Contact Lens. 2003 Apr;29(2):96-9.
13. Romero JM, Biser SA, Perry HD, et al. Conservative treatment of meibomian gland dysfunction. Eye Contact Lens. 2004 Jan;30(1):14-9.
14. McMonnies CW, Korb DR, Blackie CA. The role of heat in rubbing and massage-related corneal deformation. Cont Lens Anterior Eye. 2012 Aug;35(4):148-54.
15. Suzuki T, Teramukai S, Konoshita S. Meibomian glands and ocular surface inflammation. Ocul Surf. 2015 Apr;13(2):133-49.
16. Prabhasawat P, Tesavibul N, Mahawong W. A randomized double-masked study of 0.05% cyclosporine ophthalmic emulsion in the treatment of meibomian gland dysfunction. Cornea. 2012 Dec;31(12):1386-93.
17. Chen M, Gong L, Sun X, et al. A multicenter, randomized, parallel-group, clinical trial comparing the safety and efficacy of loteprednol etabonate 0.5%/tobramycin 0.3% with dexamethasone 0.1%/tobramycin 0.3% in the treatment of Chinese patients with blepharokeratoconjunctivitis. Curr Med Res Opin. 2012;28:385-94.
18. White EM, Macy JI, Bateman KM, Comstock TL. Comparison of the safety and efficacy of loteprednol 0.5%/tobramycin 0.3% with dexamethasone 0.1%/tobramycin 0.3% in the treatment of blepharokeratoconjunctivitis. Curr Med Res Opin. 2008;24:287-96.
19. Foulks GN, Borchman D, Tappert M, et al. Topical azithromyci and oral doxycycline therapy of meibomian gland dysfunction: a comparative clinical and spectroscopic pilot study. Cornea. 2013 Jan;32(1):44-53.
20. Kashkouli MB, Fazel AJ, Kiavash V, et al. Oral azithromycin versus doxycycline in meibomian gland dysfunction: a randomized double-masked open-label clinical trial. Br J Ophthalmol. 2015 Feb;99(2):199-204.
21. Oleñik A, Mahillo-Fernández I, Alejandre-Alba N, et al. Benefits of omega-3 fatty acid dietary supplementation on health-related quality of life in patients with meibomian gland dysfunction. Clin Ophthalmol. 2014 Apr;30(8):831-6.
22. Sanclement J, Webster P, Thomas J, et al. Bacterial biofilms in surgical specimens of patients with chronic rhinosiunusitis. Laryngoscope. 2005 Apr;115(4):578-82.
23. Guillon M, Maissa C, Wong S. Eyelid margin modification associated with eyelid hygiene in anterior blepharitis and meibomian gland dysfunction. Eye Contact Lens. 2012 Sep;38(5):319-25.
24. Lindsley K, Matsumura S, Hatef E, Akpek EK. Interventions for chronic blepharitis (review). Cochrane Database Syst Rev. 2012;16:5.
25. Guillon M, Maissa C, Wong S. Symptomatic relief associated with eyelid hygiene in anterior blepharitis and MGD. Eye Contact Lens. 2012 Sep;38(5):306-12.
26. Suhalim JL, Parfitt GJ, Xie Y, et al. Effect of desiccating stress on mouse meibomian gland function. Ocul Surf. 2014 Jan;12(1):59-68.
27. Paugh JR, Knapp LL, Martinson JR, Hom MM. Meibomian therapy in problematic contact lens wear. Optom Vis Sci. 1990 Nov;67(11):803-6.

Opening the Vault

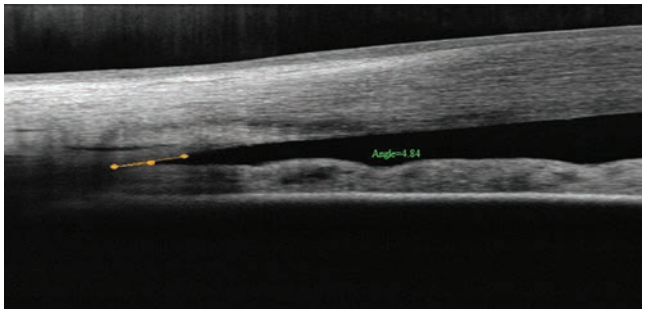
A recently implanted collamer lens is causing a patient's eye pain. How can the optometrist address it? **Edited by Paul C. Ajamian, OD**

Q Are severe eye pain and elevated intraocular pressure (IOP) common side effects of a Visian implantable collamer lens (ICL) procedure?

A The Visian ICL (Staar Surgical) is a flexible phakic IOL made from a biocompatible collagen copolymer that is placed in the ciliary sulcus behind the iris and in front of the natural lens. FDA-approved to correct myopia ranging from -3D to -20D in non-presbyopic adults, the collamer lens is a refractive option for patients looking for an alternative to LASIK or PRK. On average, the out-of-pocket cost is $3,500 per eye.

"One of the most important considerations in ICL selection is the size of the lens relative to the sulcus diameter," says Linh Hong, OD, an optometric resident at SouthEast Eye Specialists in Chattanooga, Tenn. "A lens that is too long will increase anterior vaulting. Too much vault will crowd the angle, and too little vault can result in anterior subcapsular opacification. Because of the proximity of the optics to the pupil, peripheral iridotomies (PI) are made a week prior to ICL implantation to prevent pupillary block." Ideally, the ICL vault will be between 250µm and 750µm, or approximately 100% of the corneal thickness.[1]

Pupillary block and acute angle closure are two uncommon but possible side effects of ICL implantation, says Dr. Hong. Peripheral iridotomy obstruction can occur with healing, blood clot formation or the presence of an undersized ICL. For this reason, experts recommend performing two PIs, in case one becomes occluded or non-functional.[2] Acute angle closure can be caused by an oversized ICL, which results in excessive anterior vaulting.[2] Excessive vault pushes the iris forward, which leads to appositional closure of the angle, Dr. Hong says. If the PIs are patent, this is likely the cause.



Pupil block and narrow angle shown in a patient's left eye on OCT.

Dr. Hong describes a recent case: a 24-year-old Caucasian female patient presented complaining of severe eye pain, with a history of a Visian ICL implanted nine days prior. The patient's IOP at the time of the visit was 46mm Hg. Both eyes were implanted, with IOLs displaying excessive anterior vaulting (300% OD, 500% OS). The patient had patent PIs at 11:00 in the right eye and at 1:00 in the left. OCT imaging confirmed that both eyes showed pupillary block, and the left eye demonstrated severe angle narrowing.

The patient was treated initially with 250mg of Diamox (acetazolamide, Duramed Pharmaceuticals) and one drop of Lumigan (bimatoprost ophthalmic solution 0.01%, Allergan) and Combigan (brimonidine tartrate/timolol maleate ophthalmic solution 0.02%/0.05%, Allergan) to reduce IOP to 38mm Hg. Attending doctors achieved further reduction in IOP to 15mm Hg using compression pressure with a four-mirror gonio lens. The posterior force of the gonio lens mechanically opened the iridocorneal angle, resulting in rapid lowering of IOP.

The patient was instructed to continue using Lumigan QHS OS, Combigan BID OS, and Diamox Sequels 500mg (acetazolamide ER, Duramed Pharmaceuticals) PO BID in addition to her postsurgical drops, until a follow-up could be scheduled in four days with her surgeon. The surgeon agreed with the proposed treatment plan, and scheduled an ICL exchange with a shorter lens upon her return. ■

1. Chun YS, Park IK, Lee HI, et al. Iris and trabecular meshwork pigment changes after posterior chamber phakic intraocular lens implantation. J Cataract Refract Surg. 2006 Sep;32(9):1452-8.
2. Choi KH, Chung SE, Chung TY, Chung ES. Ultrasound biomicroscopy for determining visian implantable contact lens length in phakic IOL implantation. J Refract Surg. 2007. Apr;23(4):362-7.

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OMEGA-3s: What They Can Do for You

Whether you agree or disagree with the claims, make sure you know the science.

By Susan Summerton, OD

While a staple in the average diet, "fat" has long had negative connotations in society; however, not all types are bad. Omega-3 essential fatty acids (EFAs) are key constituents in human growth and function, particularly in the brain and cell membranes. Omega-6s, the other category of dominant essential fatty acids, also play a role in brain function, as well as skin and hair growth, metabolism and the reproductive system.

Humans must rely on food or supplements to meet their omega-3 and omega-6 fatty acid needs and learn to balance the two to prevent adverse effects. It's up to practitioners to understand the differences in source, form, delivery and dosage to help patients select the best option.

Omega-3 Varieties

Two varieties of omega-3s exist: long chain and short chain. Long chain, polyunsaturated omega-3 fatty acids include eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), which can be found in cold-water oil fish like herring, mackerel, salmon, sardines, trout, bluefin and albacore tuna. As the most important and most bioactive of the EFAs, EPA and DHA are known to play vital roles in heart, circulatory, brain, mental and eye health, and are associated with anti-aging, longevity and good immune function.[1] EPA and DHA may also be effective pharmacological anti-inflammatories; however, the specifics remain under debate.[2] A recent study found that macrophages (a type of white blood cell) use DHA to produce maresins, which fight inflammation.[4]

The short chain omega-3 fatty acids include alpha linolenic acid (ALA) from plants (e.g., flax and chia seeds, walnuts and algae) and stearidonic acid (SDA) from plant-sourced oil (e.g., echium oil, black currant seed oil and genetically modified soybean oil). ALA is the primary source of omega-3 in the diet; both it and SDA must undergo a metabolic conversion to function in the body as EPA and DHA. The rate varies depending on enzymes and other factors such as genetic influences, gender, health status and quality of diet.

Types of omega-6 fatty acids include linoleic acid (LA), arachidonic acid (AA), gamma-linolenic acid (GLA) and dihomo-gamma-linolenic acid (DGLA). Rich sources of omega-6s include seeds and grains, sunflower, safflower and corn oils.

Balanced Fatty Acids

Investigators believe an imbalance between omega-3 and omega-6 levels leads to certain inflammatory and autoimmune diseases.[3] While omega-6s are essential for certain body processes, they also produce inflammatory eicosanoids, initiate more blood vessel constriction and create more pain and mucus. If a patient has

excessive omega-6 intake compared with omega-3 intake, the omega-6s can saturate enzyme activity and prevent the manufacturing of anti-inflammatory substances, even when omega-3 fatty acids are available.

Many Americans have an imbalance of omega-6 to omega-3 between 20:1 and 40:1—a healthy ratio is believed to be anywhere from 1:1 to 5:1.[3] Thus, lowering omega-6, along with increasing omega-3, should be a consideration in the standard American diet.

How Much Do You Need?

Patients can achieve adequate intake of omega-3s either by eating fish, through supplementation or a combination of both. In 2002, the Institutes of Medicine set the dietary reference intakes (DRIs) for adults for adequate intake of ALA omega-3 at 1.6g a day for men and 1.1g per day for women.[5] Subsequent recommendations made by the Academy of Nutrition and Dietetics in 2014 suggest all Americans include a non-fried fatty fish in their diet two or more times a week, providing at least 500mg of EPA and DHA per day.[6]

In clinical trials, dosages vary depending on the condition being studied. National health organizations recommend at least 1,000mg/day of EPA and DHA for support of cardiovascular and mental health. For interventions such as hypercholesterolemia and hypertriglyceridemia, the recommendations go up to 4g per day. The FDA has established a generally regarded as safe (GRAS) level of 3g of EPA and DHA per day.[7]

Balanced Fatty Acids

If obtaining omega-3s via food, the method of cooking fish is important, as omega-3 levels can be affected if the fish is cooked over high heat. Deep fried fish, for example, will have minimal amounts of omega-3 because the frying process transforms the polyunsaturated fatty acids into trans or saturated fatty acids. The fish's diet also makes a difference in omega-3 availability. Wild fish eat algae, plankton and even other fish, all of which contribute to the total concentration of EPA and DHA. Farmed fish, however, typically eat grain and soy, which are high in omega-6s, thus leading to low omega-3s in oil from farmed fish.

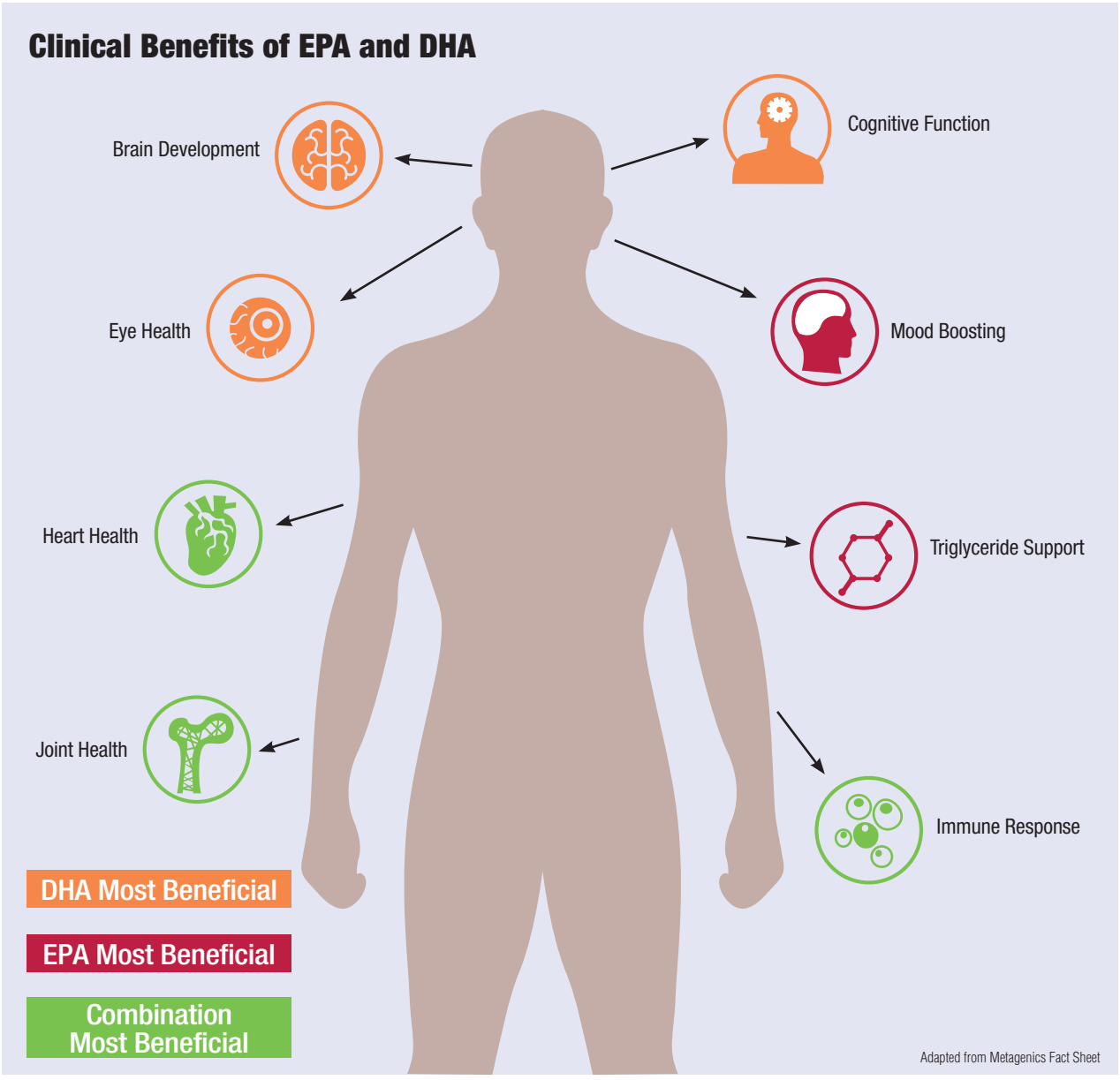
Marine oil supplements are better absorbed when taken with a fat-containing snack such as eggs or nuts. Finding a high quality, pure and potent product is imperative, as is taking the appropriate amount. (See "*What to Look for When Buying Fish Oil Supplements," at right.*)

The relative bioavailability of omega-3 fatty acids from different sources (e.g., fish, supplements or functional foods) and when consumed from different structural forms (i.e., triglyceride, ethyl ester, free fatty acid or phospholipid) is of considerable importance as well. A two-week study in 2010 showed the re-esterified triglyceride form to be more bioavailable than the ethyl ester form.[8]

Generally, plasma levels of EPA and DHA increase within hours of ingestion, but this is considered a poor biomarker of long-term omega-3 intake, especially when compared with red blood cell levels, which reflect a longer-term intake. Changes in the red blood cell membranes, by comparison, can take three to five months, thus requiring consistent dosing. Bioavailability can be increased if patients ingest their EPA/DHA supplements at or around mealtime rather than on an empty stomach, as absorption of fatty

What to Look for When Buying Fish Oil Supplements

- **Freshness.** Omega-3 oils are polyunsaturated and vulnerable to oxidation; thus, they should be kept away from excessive light and heat. Rancid oils can create "fish burps" and become pro-inflammatory, which defeats the purpose of taking them. Omega-3 oils should contain an antioxidant to stabilize the oil and preserve freshness, such as vitamin E in the form of mixed tocopherols. Avoid fish oils in clear containers that allow light to enter. Buy from a company with high product turnover or get it shipped overnight directly from the manufacturer to ensure you receive the freshest batch. Smell the product when you open the container—it should not smell "fishy."
- **Dosage.** The front label states the total fish oil contained, not the actual omega-3 content. For higher potency, the total milligrams on the front of the label should closely match the milligrams of EPA and DHA listed on the back.
- **Reputation.** The product should have third-party testing for purity. It should be independently verified to be free of polychlorinated biphenyls (PCBs), heavy metals and pesticides. Make sure the product has research reviews from independent lab companies. (Consumerlab.com is a subscription service. LabDoor.com has a free website to help determine the best over-the-counter supplements.)
- **Sustainability.** The fish used should be harvested in a sustainable manner.
- **Price.** Quality products cost more to process through molecular distillation to purify and concentrate the oils, so cost can indeed be an indicator of quality. If so, it's worth the investment.



acids is enhanced by pancreatic enzyme (lipase) and bile salts.

What are its Effects?

• ***Cardiac Effects.*** Higher doses of fish oil result in a lower risk of death from cardiovascular disease and up to 62% lower risk of fatal heart attack.[9] Additionally, the American Heart Association has concluded that omega-3s decrease arrhythmias, triglyceride levels, atherosclerotic plaque growth rate and blood pressure.

• ***Prostate Cancer.*** A 2013 study suggests an increased prostate cancer risk among men with a high blood concentration of long-chain omega-3s who participated in the Selenium and Vitamin E Cancer Prevention Trial (SELECT); however, controversy over the study's results exists.[10,11] A separate study suggests EPA and DHA intakes may reduce the risk of total and advanced prostate cancer.[12]

• ***Blood Thinning.*** EPA and DHA doses greater than 3g per day may increase prothrombin time and risk of bleeding.[5] The omega-3s may work by lowering thromboxane A2 supplies within the platelets and decrease clotting factor VII, so should not be recommended for those on warfarin

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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 Specific 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.

***Study Design:** Double-masked, randomized, parallel-group, multicenter non-inferiority comparison of the efficacy and safety of travoprost 0.004% preserved with benzalkonium chloride (BAK) to TRAVATAN Z® Solution after 3 months of treatment in patients with open-angle glaucoma or ocular hypertension. Baseline IOPs were 27.0 mm Hg (n=322), 25.5 mm Hg (n=322), and 24.8 mm Hg (n=322) at 8 AM, 10 AM, and 4 PM for TRAVATAN Z® Solution. At the end of Month 3, the TRAVATAN Z® Solution group had mean IOPs (95% CI) of 18.7 mm Hg (-0.4, 0.5), 17.7 mm Hg (-0.4, 0.6), and 17.4 mm Hg (-0.2, 0.8) at 8 AM, 10 AM, and 4 PM, respectively. Statistical equivalent reductions in IOP (95% confidence interval about the treatment differences were entirely within ±1.5 mm Hg) were demonstrated between the treatments at all study visits during the 3 months of treatment.

References: 1. Data on file, 2013. **2.** Lewis RA, Katz GJ, Weiss MJ, et al. Travoprost 0.004% with and without benzalkonium chloride: a comparison of safety and efficacy. *J Glaucoma.* 2007;16(1): 98-103. **3.** Drugs@FDA. FDA Approved Drug Products: TRAVATAN Z. www.accessdata.fda.gov/scripts/cder/drugsatfda/index.cfm?fuseaction=Search.DrugDetails. Accessed July 31, 2014.

TRAVATAN Z®

(travoprost ophthalmic solution) 0.004%

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TRAVATAN Z®

(travoprost ophthalmic solution) 0.004%

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.

DOSAGE 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 maximum effect reached after 12 hours.

TRAVATAN Z® 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
Travoprost ophthalmic solution has been reported to cause changes to pigmented tissues. The most frequently reported changes have been increased pigmentation of the iris, periorbital 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 to an increase in the number of melanocytes. 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. 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 nevi nor freckles 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.

Macular Edema
Macular edema, including cystoid macular 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 capsule, or in patients with known risk factors for macular 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® (travoprost ophthalmic solution) 0.004% and TRAVATAN Z® (travoprost ophthalmic solution) 0.004% 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. Ocular adverse reactions reported at an incidence of 1 to 4% in clinical studies with TRAVATAN® or TRAVATAN Z® Solutions included abnormal vision, blepharitis, blurred vision, cataract, conjunctivitis, corneal staining, dry eye, iris discoloration, keratitis, lid margin crusting, ocular inflammation, photophobia, subconjunctival hemorrhage and tearing.

Nonocular adverse reactions reported at an incidence of 1 to 5% in these clinical studies were allergy, angina pectoris, anxiety, arthritis, back pain, bradycardia, bronchitis, chest pain, cold/flu syndrome, depression, dyspepsia, gastrointestinal disorder, headache, hypercholesterolemia, hypertension, hypotension, infection, pain, prostate disorder, sinusitis, urinary incontinence and urinary tract infections.

In postmarketing use with prostaglandin analogs, periorbital and lid changes including deepening of the eyelid sulcus have been observed.

USE IN SPECIFIC POPULATIONS
Pregnancy
Pregnancy Category C
Teratogenic effects: Travoprost was teratogenic in rats, at an intravenous (IV) dose up to 10 mcg/kg/day (250 times the maximal recommended human ocular dose (MRHOD), evidenced by an increase in the incidence of skeletal malformations as well as external and visceral malformations, such as fused sternebrae, domed head and hydrocephaly. Travoprost was not teratogenic in rats at IV doses up to 3 mcg/kg/day (75 times the MRHOD), or in mice at subcutaneous doses up to 1 mcg/kg/day (25 times the MRHOD). Travoprost produced an increase in post-implantation losses and a decrease in fetal viability in rats at IV doses > 3 mcg/kg/day (75 times the MRHOD) and in mice at subcutaneous doses > 0.3 mcg/kg/day (7.5 times the MRHOD).

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

There are no adequate and well-controlled studies of TRAVATAN Z® (travoprost ophthalmic solution) 0.004% 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 radiolabeled 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 chronic use.

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

Hepatic and Renal Impairment
Travoprost ophthalmic 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, or 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 (MRHOD) 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 ocular dose of 0.04 mcg/kg/day on a mcg/kg basis (MRHOD)]. At 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 (75 times the MRHOD).

PATIENT COUNSELING INFORMATION
Potential for Pigmentation
Patients should be advised about 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 eyelash growth. Eyelash changes are usually reversible upon discontinuation of treatment.

Handling the Container
Patients should be instructed to avoid allowing 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.

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or heparin.[13] Supplementing isolated DHA without EPA, which inhibits platelet aggregation, can be considered if necessity arises.

• ***Pregnancy and Infants.*** DHA is known to play an essential role in maternal health and fetal development (i.e., brain, eyes, nerve and immune systems). Several studies have linked higher maternal intake of omega-3s with lower risk of child suboptimal verbal IQ, better visual function and long-term function of the visual parvocellular pathway in school-aged children.[14,15] The FDA recommends that pregnant women avoid fish that contain high levels of mercury, particularly shark, swordfish, king mackerel and tilefish.[15]

• ***Memory.*** DHA stimulates the growth of new neurons in our brains, leading to improved memory by activating the production of brain-derived neurotrophic factor, a protein important for neuronal survival and long-term memory. Supplementation has been shown to support cognitive health with aging.[8]

• ***Dry Eye Syndrome.*** Omega-3 intake is a well-known method to lower the incidence of dry eye.[16] Additionally, taking sea buckthorn oil containing omega-3 and omega-6 has been shown to attenuate tear film osmolarity and decrease redness and burning associated with dry eye.[17] Omega-3s can block the gene expression of the inflammatory cytokines tumor necrosis factor alpha (TNF-α), interleukin-1 alpha (IL-1α) and leukin-1 beta (IL-1β). This can be helpful in the case of Sjögren's syndrome, in which the lacrimal glands have increased TNF-α.

• ***Meibomian Gland Dysfunction (MGD) and Blepharitis.*** Omega-3 dietary supplementation for blepharitis and MGD may decrease the red blood cell and plasma ratios of omega-6 to omega-3 and improve overall ocular surface index (OSDI) score, tear break-up time (TBUT) and meibum score.[18]

• ***Macular Degeneration.*** Studies have suggested eating fish more than twice a week may cut risk of developing macular degeneration in half.[19] Additionally, a diet low in trans- and unsaturated fat and rich in omega-3 fatty acids and olive oil may reduce the risk of age-related macular degeneration.[20]

• ***Combining Compounds.*** Carnitine is the delivery system for long-chain fatty acids. The less available carnitine, the fewer fatty acids get into the cell. A combination of acetyl-l-carnitine, omega-3 fatty acids and coenzyme Q-10 can improve visual functions, including visual field mean defect, visual acuity and foveal sensitivity in patients with early age-related macular degeneration. They also can improve fundus alterations by decreasing drusen.[21]

• ***Retinitis Pigmentosa.*** Patients with X-linked retinitis pigmentosa have low levels of DHA in red blood cells compared with normal sighted control subjects.[22] DHA supplementation may correct a fatty acid deficiency and rod ERG functional loss in X-linked retinitis pigmentosa.[23]

• ***Glaucoma.*** A small study found that primary open-angle glaucoma patients have reduced blood levels of DHA and EPA compared with normal controls.[24] EPA and DHA appear to modulate impaired systemic microcirculation, ocular blood flow and optic neuropathy associated with glaucoma.[24]

• ***Cataracts.*** The Nurses' Health Study showed women with the highest EPA/DHA intake had 12% lower risk for cataract extraction.[25] Those who had more than three servings of fish per week had a 19% lower risk.[25]

• ***Uveitis.*** In vivo EPA inhibits multiple inflammatory molecules. In animal models, oral EPA showed a decrease in leukocyte adhesion to retinal vessels, a decrease in leukocyte infiltration into the vitreous cavity along with a decrease in pro-inflammatory cytokines.[26] ■

Dr. Summerton is a certified nutrition specialist and a clinical nutritionist at the University of Miami. She serves as secretary and chair for the fellowship committee

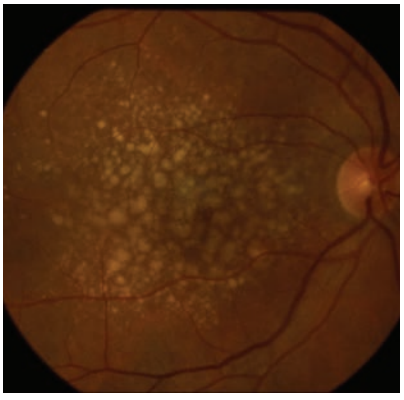


Photo: Jay M. Haynie, OD

Omega-3 fatty acids, in combination with other dietary supplements, may reduce risk of AMD.

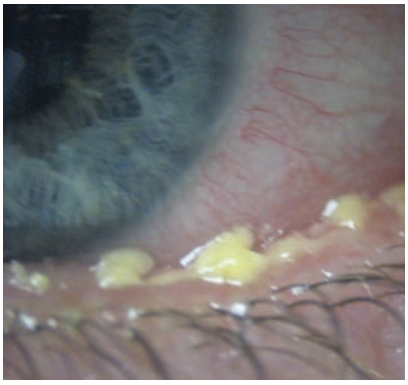
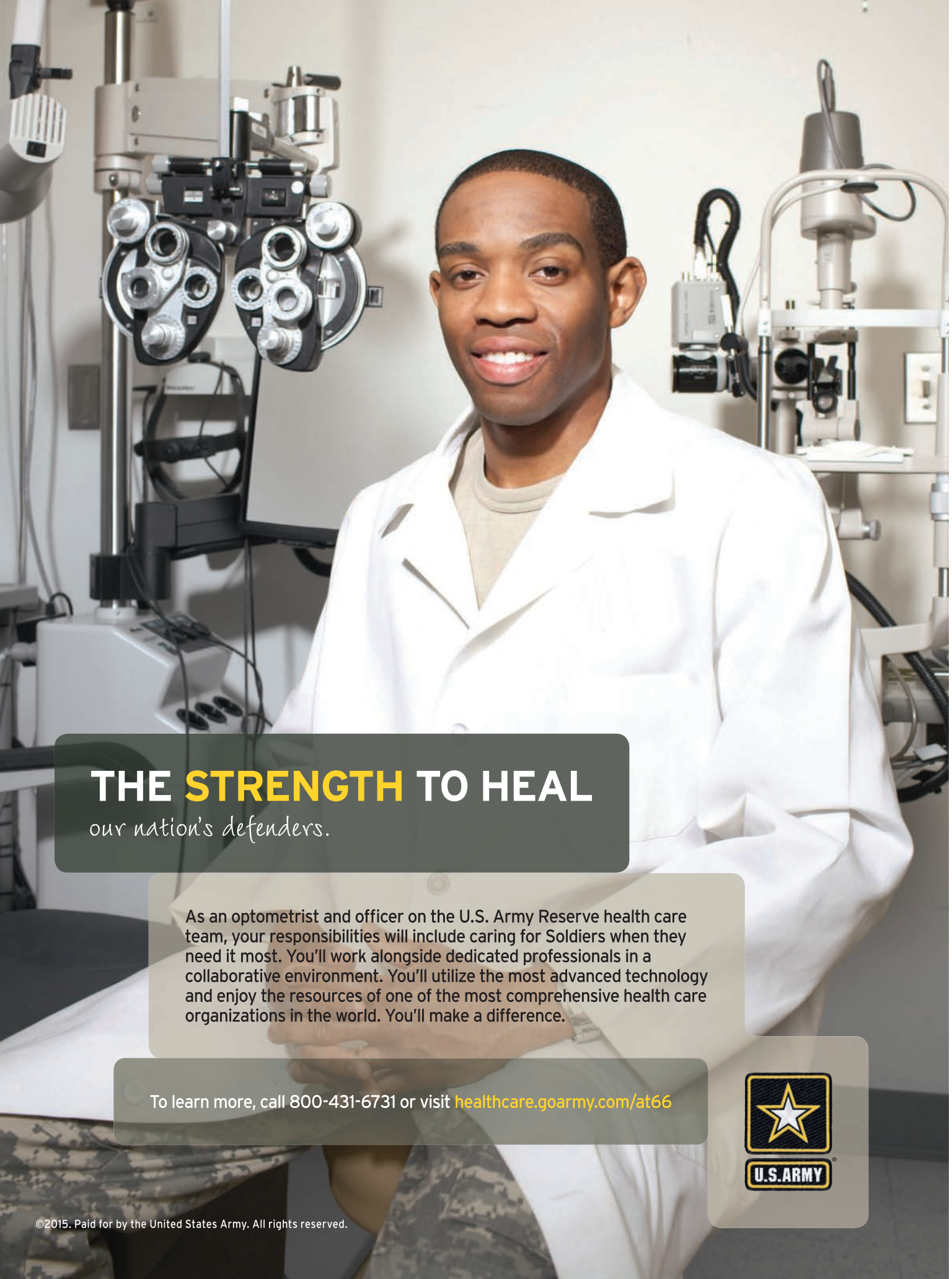


Photo: Christine W. Sindt, OD

Omega-3 dietary supplements may improve OSDI, TBUT and meibum scores in patients with blepharitis.

for the National Ocular Nutrition Society. She is also a member of the American College of Nutrition.

1. Swanson D, Block R, Mousa SA. Omega-3 fatty acids EPA and DHA: health benefits throughout life. Adv Nutr 2012 Jan;3(1):1-7.
2. Calder PC. Omega-3 polyunsaturated fatty acids and inflammatory processes: nutrition or pharmacology? Br J Clin Pharmacol. 2013 Mar;75(3):645-62.
3. Simopoulos AP. The importance of the ratio of omega-6/omega-3 essential fatty acids. Biomed Pharmacother. 2002 Oct:56(8):365-79.
4. Dalli J, Zhu M, Vlasenko NA, et al. The novel 13S,14S-epoxy-maresin is converted by human macrophages to maresin 1 (maR1), inhibits leukotriene A4 hydrolase (LTA4H), and shifts macrophage phenotype. FASEB J. 2013 Jul;27(7):2573-83.
5. Institute of Medicine. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein and Amino Acids (Macronutrients). Washington, DC: The National Academies Press, 2005.
6. Vannice G, Rasmussen H. Position of the academy of nutrition and dietetics: dietary fatty acids for healthy adults. J Acad Nutr Diet. 2014 Jan;114(1):136-53.
7. US Food and Drug Administration. Substances affirmed as generally regarded as safe. Federal Register. 1997;62(108):30757-7.
8. Yurko-Mauro K, McCarthy D, Rom D, et al. Beneficial effects of docosahexaenoic acid on cognition in age-related cognitive decline. Alzheimers Dement. 2010 Nov;6(6):456-64.
9. de Goede J. Geleijnse JM, Boer J, et al. Marine (n-3) fatty acids, fish consumption, and the 10-year risk of fatal and nonfatal coronary heart disease in a large population of Dutch adults with low fish intake. J Nutr. 2010 May:140(5):1023-28.
10. Brasky TM, Darke AK, Song X, et al. Plasma phospholipid fatty acids and prostate cancer risk in the SELECT trial. J Natl Cancer Inst. 2013 Aug 7;105(15):1132-41.
11. Council for Responsible Nutrition. CRN Says New Study on Omega-3 Conclusions are Overblown. Available at: http://www.crnusa.org/CRNPR13-Omega3071113.html. Accessed: June 14, 2015.
12. Leitzmann MF, Stampfer MJ, Michaud DS, et al. Dietary intake of n-3 and n-6 fatty acids and the risk of prostate cancer. Am J Clin Nutr. 2004 Jul:80(1):204-16.
13. Salonen R, Nikkari T, Seppänen K, et al. Effect of omega-3 fatty acid supplementation on platelet aggregability and platelet produced thromboxane. Thromb Haemost. 1987 Jun 3:57(3):269-72.
14. Hibbeln JR, Davis JM, Sterr C, et al. Maternal seafood consumption in pregnancy and neurodevelopmental outcomes in childhood (ALSPAC study): an observational cohort study. Lancet. 2007 Mar 19;99(6A):44C-46C.
15. Jacques C, Levy E, Muckle G, et al. Long-term effects of prenatal omega-3 fatty acid intake in school aged children. J Pediatr. 2011 Jan;158(1):83-90, 90.e1.
16. Bhargava R, Kumar P, Kumar M, et al. A randomized controlled trial of omega-3 fatty acids in dry eye syndrome. Int J Ophthalmol. 2013 Dec 186(6):811-6.
17. Larmo PS, Järvinen RL, Setälä NL, et al. Oral sea buckthorn oil attenuates tear film osmolarity and symptoms in individuals with dry eye. J Nutr. 2010 Aug;140(8):1462-8.
18. Macsai MS. The Role of Omega-3 Dietary Supplementation in Blepharitis and Meibomian Gland Dysfunction (An AOS Thesis). Trans Am Ophthalmol Soc. 2008 Dec;106:336–56.
19. SanGiovanni JP, Chew EY, Clemons TE, et al. Age-Related Eye Disease Study Research Group. The relationship of dietary lipid intake and age-related macular degeneration in a case-control study. AREDS Report No. 20. Arch Ophthalmol. 2007 May:125(5):671-9.
20. Chong EW, Robman LD, Simpson JA, et al. Fat consumption and its association with age related macular degeneration. Arch Ophthalmol. 2009 May;127(5):674-80.
21. Feher J, Kovacs B, Kovacs I, et al. Improvement of visual functions and fundus alterations in early age related macular degeneration treated with a combination of acetyl-L carnitine, omega-3 fatty acids and coenzyme Q10. Ophthalmologica. 2005 May-June;219(3):154-66.
22. Hoffman DR, Birch DG. Docosahexaenoic acid in red blood cells of patients with X-linked retinitis pigmentosa. Invest Ophthalmol Vis Sci. 1995 May;36(6):1009-18.
23. Hoffman DR, Locke KG, Wheaton DH, et al. A randomized, placebo-controlled clinical trial of docosahexaenoic acid supplementation for X-linked retinitis pigmentosa. Am J Ophthalmol. 2004 Apr;137(4):704-18.
24. Ren H, Magulike N, Ghebremeskel K, Crawford M. Primary open-angle glaucoma patients have reduced levels of blood docosahexaenoic and eicosapentaenoic acids. Prostaglandins Leukot Essent Fatty Acids. 2006 Mar;74(3):157-63.
25. Lu M, Cho E, Taylor A, et al. Prospective study of dietary fat and risk of cataract extraction among US women. Am J Epidemiol. 2005 May 15;161(10):948-59.
26. Suzuki M, Noda K, Kubota S, et al. Eicosapentaenoic acid suppresses ocular inflammation in endotoxin-induced uveitis. Mol Vis. 2010;16:1382-8.



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In the Eye of the Beholder: Aesthetic Procedures for the Periocular Area

Do you know how to talk to your patients about this important, but often overlooked, area? **By Aliza Becker, Associate Editor**

Optometrists are well versed in recognizing functional concerns and managing ocular disease treatment, yet many do not consider the various dermatological options and surgical procedures available for aesthetic enhancement of the eyes—and many patients, while curious, many not know how to ask.

Photos: Wendy W. Lee, MD

Before (left) and after (right) an injection of botulinum toxin between the eyebrows.

When discussing aesthetics around the eyes, says Wendy Lee, MD, an oculoplastics surgeon at Bascom Palmer Eye Institute in Miami, it's important to first remember the anatomy of the periocular area and why it is vulnerable to aging. Periorbital skin is the thinnest on the body and contains lower numbers of sebaceous glands than other areas. These glands produce sebum to lubricate and waterproof the skin; with fewer glands, periorbital skin tends to dry out and wrinkle faster. A lack of subcutaneous tissues beneath the thin eyelid skin means the skin tightly adheres to the underlying muscles, which are constantly contracting and transmitting movement to the delicate tissue.

As we age, our skin becomes thinner and loses its elasticity as a result of slower production of the proteins collagen and elastin, which impacts the skin's ability to repair itself and hold its shape.[1] Age also affects bone, tendon and muscle mass, which can alter the shape of the periorbital area.

The reason we *notice* signs of aging around the eyes faster, says Dr. Lee, is because the eyes are the aesthetic center of the face. It is typically the area we focus on when we're talking and listening to other people, so any irregularities or blemishes are noticed more quickly than if they were present on other parts of the body. For this reason, many patients are concerned with making sure their eyes and surrounding skin look as symmetrical, youthful and healthy as possible.

Hydrate and Protect

The most basic education optometrists can provide to their patients is on preventative care, namely: hydrate and protect. When it comes to hydration, "anyone who has dryness in the eyelids will notice more accentuation of fine lines and creases," says Hilary Johnson, MD, clinical assistant professor of dermatology at the University of Iowa Carver College of Medicine. Most facial creams contain a hydrating agent, but it's important to select one designed specifically for eyelid use to avoid complications.

Adequate sun protection is a must as well. "It's hard for us to think of the eyelids as one of those places to include sunblock, but we see a lot of skin cancer in the eyelid in older folks so it's not a bad idea. From a cosmetic point of view, a lot of our signs of aging, including the skin around the eyelid, is related to exposure from ultraviolet light from the sun," Dr. Johnson says. Patients should remember to apply a stick sunblock around the eyes, which she says is easy to apply and won't drip in the eyes.

Kimberly Cockerham, MD, an ophthalmologist in Stockton, Calif., agrees with Dr. Johnson that sun protection is key, and points out that powdered micronized zinc oxide and titanium oxide are other good ways to protect the eyelids, as they defend against ultraviolet (UV) light better than chemical blocks. While not a makeup, Dr. Cockerham says, some companies make the powders available in shades other than neutral.

While certainly not an easy fix, applying a clear or tinted UV-protective film to car windows could ensure patients are protected when they are the most vulnerable. "Most people get the vast majority of their sun exposure driving," Dr. Cockerham says. "Adding UV protection to your car window will help prevent premature aging, and it's going to help the eye-related issues that are secondary to UV exposure." Note, window tinting is regulated in most states.

Permanent Makeup

By Elyse L. Chaglasian, OD

Permanent tattooing of the eyelids, eyelashes and eyebrows has become a popular practice for those seeking a more convenient, longer-lasting and less irritating alternative to daily makeup application and removal. Known as blepharopigmentation, the procedure was first described by Giora Angres, MD, in 1984 and involves the application of iron oxide pigment along the lash line using a rotary needle.[2]

While blepharopigmentation yields desirable results, there are some concerns surrounding both the materials used and the skill of the operators. The FDA does not regulate the tattoo industry, including the inks or dyes used; these have been known to elicit infection, allergic reactions, granulomas and dermatitis in various individuals.[3-5] Technician training and licensing is also unregulated, increasing the risk of poorly trained individuals making a mistake.

Results from an FDA phone survey with 92 patients who reported adverse events after permanent makeup application indicated that tenderness (95%), swelling (91%), itching (88%) and bumps (83%) were the most common reactions. Sixty-three (68%) patients reported that their reactions had not completely resolved at the time of the survey; duration of the symptoms ranged from five and a half months to over three years.[6]

Topicals

Patients generally present with one of three main complaints, Dr. Lee says: dark circles, wrinkles or puffiness. Topical eye creams can be a great treatment option, but optometrists have to be careful to suggest the right ones, since "with topical eye creams, it's not one size fits all," says Dr. Lee. "Antioxidants for instance, like vitamin C and green tea, help to fight free radicals, so, not only damage from the sun but also environmental factors [like pollution]." Different products target different problems, so it is important to listen to your patient about what bothers them the most, then try to match that with physical findings.

Dr. Lee recommends targeting wrinkles using creams containing retinols, which help to stimulate collagen. Dark circles, on the other hand, are much tougher to treat because they are due to several factors. Dark circles are partially the result of oxygenated blood pooling in the veins underneath the thin under-eye skin, which can be exacerbated by a variety of factors, including allergies, fatigue and age. The fat-soluble vitamin K, which plays an essential role in blood clotting, has been shown to reduce the severity of bruising following cosmetic laser surgery and may have a similarly minimizing effect on blood that pools in the veins underneath the eye.[7] Topical retinol may also help reduce the appearance of dark circles by thickening the lower layer of the epidermis and dermis, in turn increasing blood flow and collagen production within the skin.[8]

Puffiness around the eyes is also a concern patients can address with topical creams, notes Dr.

Lee, although she warns that they won't work for all patients. "My take on puffiness is to be realistic. What is the puffiness due to? Most of the time, when my patients come in complaining of puffiness, it's really fat prolapse, which is a surgical issue. Topicals are not going to take that puffiness away."

-If the swelling is edema from lifestyle choices like smoking or sleep deprivation, Dr. Lee recommends the use of topical arnica, a homeopathic remedy sometimes used to treat muscle aches and post-surgical pain and swelling. However, take care to avoid getting it directly in the eye, and keep in mind its limitations on decreasing actual edema around the eyes.

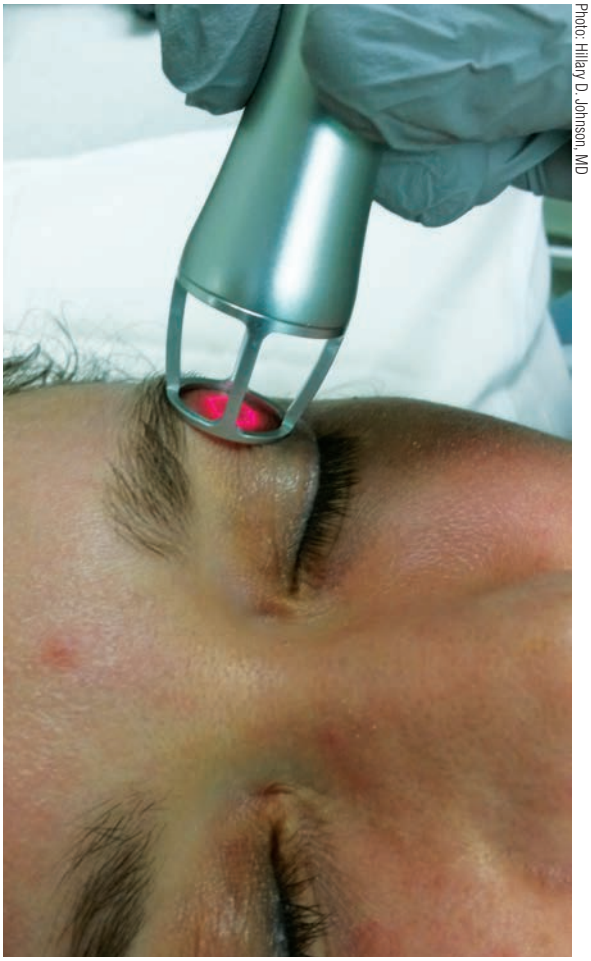

Photo: Hillary D. Johnson, MD

The Co_2 laser in action on an eyelid.

Injectables

Botulinum toxin is a neurotoxin produced by the bacterium *Clostridium botulinum* that, when injected, temporarily prevents muscle contraction.[9] First demonstrated to treat strabismus in 1981, it has since been approved by the FDA under the brand names Botox (Allergan), Dysport (Galderma) and Xeomin (Merz Pharma) for the treatment of various spastic muscle dystonias and similar medical conditions.[9]

Cosmetically, botulinum toxin is used to shape the periocular region, explains Dr. Lee. "Not only can dynamic wrinkles be softened in the glabella region, but you can also create a nice medial brow lift at rest. As far as lateral canthal lines go, you can rejuvenate and improve the crow's feet area," she says. Botulinum toxin can also be injected underneath the eyelids into the pretarsal orbicularis to decrease the roll patients sometimes develop. It can also work to create a subtle widening of the palpebral fissure, making the eyes appear more open.

However, caution should be taken when making these injections in patients with preexisting dry eye, Dr. Lee warns, as they can exacerbate the condition by weakening the orbicularis. Additionally, while botulinum toxin can be used off-label in the lateral brow area and forehead, "you have to be careful when injecting that area because you can actually drop the brows and crowd the periorbital area," Dr. Lee says. Optometrists should be sure to educate themselves and their patients regarding the anatomy in this area, as well as all potential consequences.

Dermal fillers are another injectable better suited for reducing static wrinkles, or wrinkles that are present when the face is at rest. If injecting around the eyes, Dr. Lee recommends use of hyaluronic acid, since it is reversible with hyaluronidase. "The tear trough is probably the most popular area around the eyes to inject, as it helps fill in the volume deficits and contour changes between the lid-cheek junction," she says. At the same time, however, "the tear trough is very unforgiving because of the thinness of the skin and if you inject too much, you're going to see lumps and bumps."

Lasers

Laser skin resurfacing is a nonsurgical technique capable of reducing facial wrinkles, scars and blemishes by stimulating growth of collagen fibers.[10]

"It's one of the few devices I think that truly smooths and tightens the skin. It's a nice thing to do along with eyelid surgery," Dr. Johnson says. "It helps reduce signs of aging, reduces brown spots and wrinkles and builds for tighter skin." Recent laser resurfacing technology uses a fractional form, treating microscopic columns of skin. In general, fractional laser-resurfacing treatments fall under two categories: abla-

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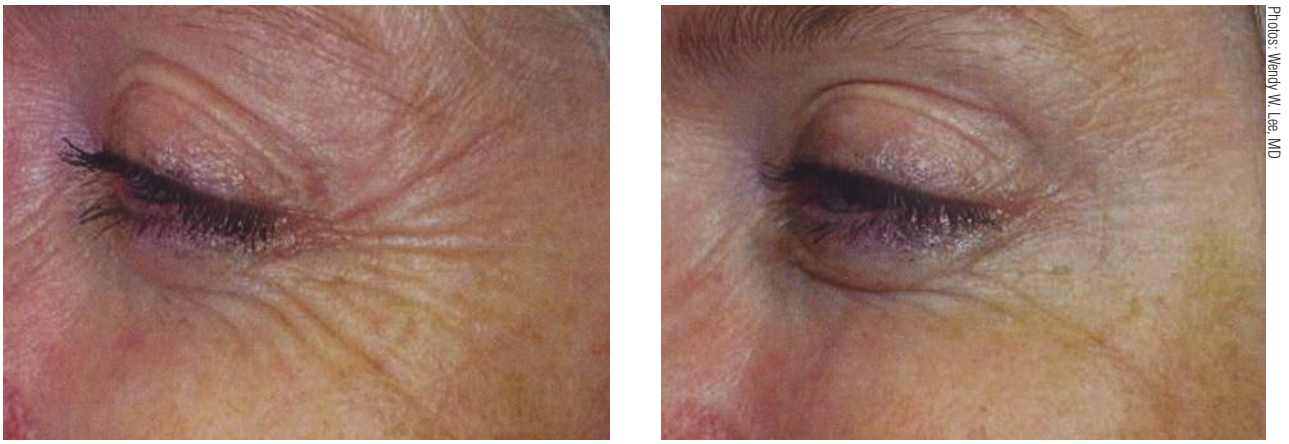

Photos: Wendy W. Lee, MD

Before (left) and after (right) an injection of botulinum toxin to soften crow's feet.

tive and non-ablative. Fractional ablative lasers are more invasive, require more recovery time and are more effective. Non-ablative fractional resurfacing lasers and lasers that use radio frequency technology require less downtime but are also less effective.[11,12] "Carbon dioxide fractional laser resurfacing is my all-time favorite for safe resurfacing of the eyelids with noticeable improvement after one treatment, but you would want to make sure that the person operating the laser has a lot of experience," Dr. Johnson says.

Regardless of the type of laser or light used, Dr. Lee cautions that the patient and everyone in the operating room should wear eye protection during the procedure. "Most of [the lasers] are very absorbent in water, but there are some that will absorb into pigment and hemoglobin as well, which puts your retina and iris at risk," she says.

Dr. Johnson recommends the patient be given certain medications in advance to handle the discomfort associated with healing time and to protect against reactivation of cold sores or herpes simplex virus. Patients with rosacea, including ocular rosacea, do not see a large difference in how the skin heals. However, rosacea patients "tend to have more sensitive skin in general. We use a different laser on the rest of the face, the pulsed dye laser, to reduce the appearance of red blood vessels, and folks with rosacea tend to be more sensitive in terms of having more skin allergies," she says.

For patients with hyperpigmentation, the effects of laser resurfacing depend on the cause of the skin coloring. Brown spots caused by irritation or allergic reactions often fade on their own, and skin conditions like melasma can be treated using sunblock and prescription bleaching cream. "That tends to have a better track record than certain lasers that target pigment," Dr. Johnson says. "Then there are folks who have brown around the eyelids from mole cells (nevus of ota). We can use a laser for that, which targets the pigment in the skin cells."

Dr. Cockerham notes laser resurfacing should be avoided in patients who have a higher amount of melanin in their skin than Caucasian patients. "The laser works by causing thermal damage to the skin. Thermal damage to melanocytes can result in hyperpigmentation and scarring," she says, adding that microneedling is often a good alternative for these patients. Optometrists in states that allow injections into skin could potentially provide this service.

Surgery

The most common surgical enhancements for periorbital rejuvenation are blepharoplasties of both the upper and lower lids, which are particularly useful for patients with fat prolapse and excess skin, Dr. Lee says. During the procedure, incisions are typically made along the natural upper eyelid crease or through the conjunctiva or just below the eyelashes on the lower eyelid. Fat, muscle and excess skin are then removed and the lids are tightened as needed.

Lower eyelid blepharoplasty is usually more cosmetic in nature to reduce under-eye puffiness eye bags or improve wrinkles, while upper eyelid blepharoplasty is more commonly done to address functionality in older patients with vision impairment caused by ptosis. A third type of blepharoplasty, known as double eyelid surgery, is popular among Asians and Asian

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Americans; it involves the creation of a crease in the upper eyelid.[13]

Optometrists can work together with ophthalmologists to enhance pre- and postoperative care, says Erin Shriver, MD, assistant professor in the Department of Ophthalmology and Visual Sciences for University of Iowa Health Care. For example, patients with blepharitis should be placed on a regimen of warm compresses to improve lid hygiene before surgery, and then for several weeks after, she says.

Additionally, an optometrist should be consulted if the patient wears contact lenses, especially if the lenses are being used for purposes other than refractive correction, such as to preserve the ocular surface. Typically, says Dr. Shriver, contact lenses should remain out for at least a week following surgery to prevent aggravation of dryness that can result following the procedure.

Optometrists are uniquely qualified to manage ocular surface conditions such as ocular rosacea, blepharitis and dry eye before and after eyelid and other cosmetic periocular procedures; thus, they can play a pivotal role in the education and comanagement of patients interested in ocular aesthetics—be it topicals, injectables, laser resurfacing or surgery. ■

1. The International Dermal Institute. Structural Changes Associated with Aging Skin. Available at: www.dermalinstitute.com/us/library/11_article_Structural_Changes_Associated_with_Aging_Skin.html. Accessed May 5, 2015.
2. Angres GG. Eye-liner implants: A new cosmetic procedure. Plast Reconstr Surg. 1984 May:73(5):833-6.
3. De Cuyper C. Permanent makeup: indications and complications. Clin Dermatol. 2008 Jan-Feb;26(1):30-4.
4. Vagefi MR, Dragan LD, Hughes SM, et al. Adverse reactions to permanent eyeliner tattoo. Ophthal Plast Reconstr Surg. 2006 Jan-Feb;22(1):48-51.
5. Rubianes EL, Sanchez JL. Granulomatous dermatitis to iron oxide after permanent pigmentation of the eyebrows. J Dermatol Surg Oncol. 1993 Jan;19(1):14-6.
6. Straetemans M, Katz LM, Belson M. Adverse reactions after permanent-makeup procedures. N Engl J Med. 2007 Jun;356(26):2753.
7. Shah NS, Lazarus MC, Bugdodel R, et al. The effects of topical vitamin K on bruising after laser treatment. J Am Acad Dermatol. 2002 Aug:47(2):241-4.
8. Mukherjee S, Date A, Patravale V, et al. Retinoids in the treatment of skin aging: an overview of clinical efficacy and safety. Clin Interv Aging. 2006 Dec;1(4):327-38.
9. Nigam PK, Nigam A. Botulinum toxin. Indian J Dermatol. 2010 Jan-Mar;55(1):8-14.
10. American Society of Plastic Surgeons. What is laser skin resurfacing? Available at: www.plasticsurgery.org/cosmetic-procedures/laser-skin-resurfacing.html. Accessed May 5, 2015.
11. Alexiades-Armenakas MR, Dover JS, Arndt KA. The spectrum of laser skin resurfacing: nonablative, fractional and ablative laser resurfacing. J Am Acad Dermatol. 2008 May:58(5):719-37.
12. Preissig J, Hamilton K, Markus R. Current laser resurfacing technologies: A review that delves beneath the surface. Semin Plast Surg. 2012 Aug;26(3):109-16.
13. Nguyen MQ, Hsu PW, Dinh TA. Asian blepharoplasty. Semin Plast Surg. 2009 Aug;(3):185-97.

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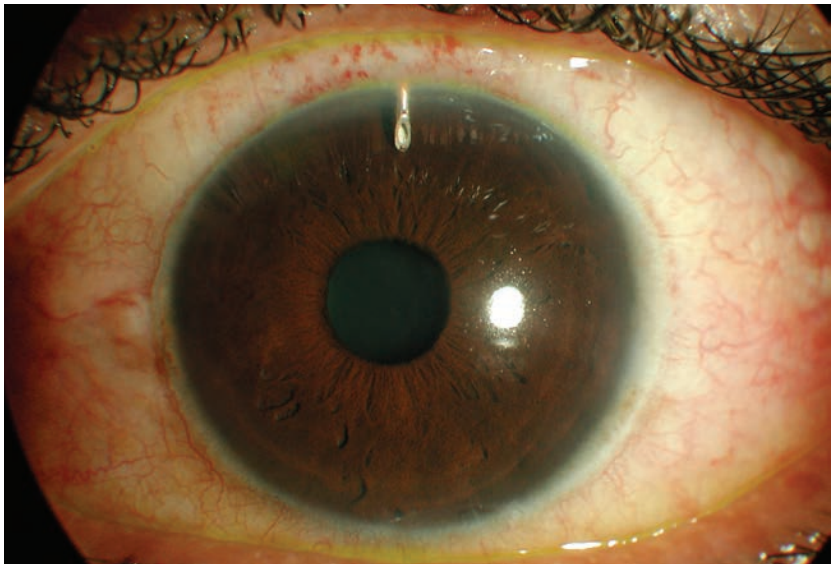
1. Chalmers RL, Hickson-Curran SB, Keay LJ, Gleason W. Safety of hydrogel and silicone hydrogel daily disposables in a large post-market surveillance registry—the TEMPO registry. Presented at: ARVO 2014 Annual Meeting: May 4-8, 2014; Orlando, FL.

An OD's Guide to Glaucoma Surgery

Some patients will opt for drainage implants or other procedures. How will they impact how optometrists monitor and treat? **By Richard Zimbalist, OD, and Angela Gentry, OD**

For the most part, optometrists opt to initiate glaucoma treatment using topical medications. In our practice, we strive to treat each patient to the fullest extent of our licensure. But what happens when a patient's IOP cannot be adequately controlled with medical intervention alone? That's when we refer to a glaucoma specialist. Sometimes, that means the patient will undergo glaucoma surgery. Fortunately for them, surgical options are less invasive and safer than ever before. Laser and surgical procedures such as laser trabeculoplasty, minimally invasive glaucoma surgery (MIGS) or incisional glaucoma surgery may be just the solution for these patients. But just because they're seeking treatment in an MD's office, that doesn't mean our job is done.

This article provides an overview of these modern procedures and reviews the OD's role in managing patients who have undergone them.



The Express shunt under a scleral flap, post-op day one. The bleb is elevated but fairly diffuse, the chamber is deep and there is little inflammation. This is one of several available glaucoma drainage implants.

Drop Compliance

Medication compliance is an elusive part of glaucoma management. We have all encountered patients who insist that they comply with their eye drops, yet have not filled the order since treatment was initiated.

That lack of adherence, a study shows, turns out to be one of the primary causes of IOP fluctuation.[1] And IOP fluctuation is associated

with an increased risk of visual field progression, particularly in patients with a lower initial mean IOP.[2-4]

It is imperative for patients to understand the need for strict compliance with therapy to prevent fluctuations that hasten the progression of their disease.

Although several topical glaucoma drops may significantly lower eye pressure, some patients may need additional methods of intervention to reach a suitable IOP. The Advanced Glaucoma Intervention Study (AGIS): 7 shows a lower IOP is associated with a reduced progression of visual field defects.[5] Furthermore, those with IOPs less than 18mm Hg showed minimal visual field worsening in a six-year period.[5]

These patients may be treated with procedures such as trabeculectomy or receive one of several available glaucoma drainage implants (GDI).

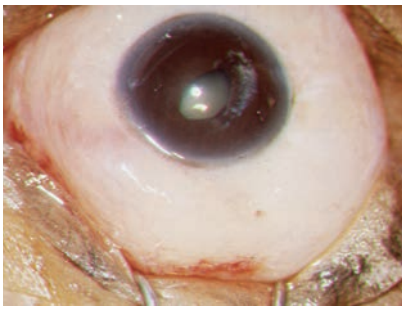
Trabeculectomy

Trabeculectomy is a commonly performed, non-laser glaucoma surgery in which a fistula connecting the anterior chamber and the subconjunctival space is created to provide an alternative pathway of aqueous outflow.[6-8] Success depends upon establishing proper filtration and ensuring the patency of the fistula.[9]

Antimetabolites

Antimetabolites are chemical agents typically used during incisional glaucoma surgery to reduce conjunctival scarring and the likelihood of procedure failure. The two antimetabolites commonly used today are 5-fluorouracil (5-FU) and mitomycin-C (MMC).

5-FU inhibits fibroblast proliferation whereas MMC causes crosslinking of DNA and may also inhibit RNA and protein synthesis. Both antimetabolites work well, but must be used with caution, as corneal toxicity and wound leaks are common. In particular, MMC is historically considered more potent because the complications are more severe.[10]



A completed trabeculectomy with a diffuse, 360-degree filtration bleb.

Late Postoperative Trabeculectomy Complications

When evaluating a patient with a trabeculectomy, doctors should examine certain key features. Signs of a healthy, well-functioning bleb include the presence of multiple intraepithelial microcysts (which can be viewed easily with fluorescein sodium), minimal vascularity, decreased height, thick walls and the absence of corkscrew vessels. It is also good practice to use a fluorescein strip to evaluate for the presence of a bleb leak as evidenced by a positive Seidel test. Since aqueous does not take up fluorescein dye, a bleb leak will appear as dark flow surrounded by normal-appearing dye on the corneal surface.[11]

Late trabeculectomy complications are often attributed to changes in the bleb's structure. The most common of these include chronic hypotony, bleb leaks and blebitis.[12]

Chronic hypotony is defined as an intraocular pressure of less than 5mm Hg for at least a three-month period. Non-surgical treatment options include the use of a soft contact lens, cryotherapy to reduce bleb size, autologous blood injection with or without compression sutures, and applying an argon laser to the bleb. However, surgical revision is the most successful modality to elevate intraocular pressure. Rarely, choroidal effusion or suprachoroidal hemorrhage may result from longstanding hypotony.[12]

The use of antimetabolites is

Trabeculectomy Steps

Trabeculectomy is performed using the following steps:

- The eye is anaesthetized and a superficial incision involving the conjunctiva and Tenon's capsule is made approximately 8mm to 10mm superior to the limbus.
- The conjunctival-Tenon's flap is bluntly dissected to the limbus. The surgeon may then use an antimetabolite to minimize conjunctival scarring. The antimetabolite is soaked in a sponge and held on the sclera for two to five minutes.
- The surgical area is then irrigated and a wide scleral incision, typically 4mm wide, that extends to the corneal limbus, is made.
- A piece of the sclera that contains a portion of trabecular meshwork is removed.
- A surgical iridectomy is performed by grasping the iris with forceps and cutting a small section with scissors.
- The scleral flap is closed with three nylon sutures.
- An outflow test is then performed with saline solution.
- The conjunctiva and Tenon's capsule are closed with absorbable sutures.
- Lastly, the surgeon paints the conjunctival suture line with a fluorescein strip to ensure no wound leaks exist.[1]

1. Khaimi M, Reyes M. Filtering Surgery in the Management of Glaucoma. In: Kahook MY SJED, ed. Chandler and Grant's Glaucoma. 5th ed. Thorofare, NJ:SLACK, Inc.;2013.

associated with thin-walled blebs and bleb leaks.[10,11] Aqueous suppressants, broad-spectrum antibiotics, patching and soft contact lenses may be used to close the leak. Other treatment options include cyanoacrylate glue, fibrin tissue glue, injection of autologous blood and surgical intervention.[12]

Thin-walled blebs are also at a higher risk for infection and blebitis. The infected bleb will typically exhibit a local conjunctival hyperemia and a milky-white appearance. In addition to thin walls, the presence of myopia, releasable sutures, respiratory infections, inferior limbus blebs, unguarded filtration surgery and diabetes increase the risk for bleb infection. Blebitis may also be associated with bleb leak, hypopyon, vitreous reaction, and may even develop into an endophthalmitis.[12]

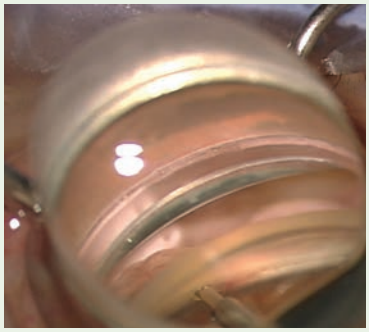
Symptomatic blebs provide varying degrees of discomfort and often result in superficial punctate keratopathy, dellen formation and ocular surface irregularities. The recommended treatment to alleviate irritation and discomfort is copious artificial tears and lubrication. If symptoms persist after frequent use of lubrication, compression sutures may be placed to compress the bleb to the sclera.

Trabeculectomies remain the initial incisional glaucoma procedure for many surgeons; however, the glaucoma drainage implants have grown in popularity recently due to their successful use in secondary glaucoma and failed blebs.

Drainage Implants

GDIs

Glaucoma drainage implants (GDI) are devices constructed of a tube that shunts aqueous humor from the anterior chamber to an encapsulated fibrous plate located at the equatorial region of the globe. The aqueous fluid is absorbed by the periocular capillaries and lymphatic system of the subconjunctival space. Although the basic concepts of all GDIs are the same, the implants differ with respect to shape, material, size and presence of a valve.[13] Despite all of the design differences, a systematic literature review found that all glaucoma drainage implants effectively lowered the preoperative IOP by at least 50%.[14]



The Trabectome goes through a clear corneal incision and uses bipolar cautery to open up parts of the trabecular meshwork.

Identifying MIGS candidates

MIGS is growing in popularity because it is effective and less disruptive to the native ocular tissue than prior surgical interventions.

Procedures such as Trabectome and shunt or stent implantation are potentially useful options for mild to moderate glaucoma, but may not be a suitable for patients with various forms of secondary, advanced or refractory glaucoma.[1-4]

1. AGIS Investigators. The Advanced Glaucoma Intervention Study (AGIS):7. The relationship between control of intraocular pressure and visual field deterioration. Am J Ophthalmol. Oct 2000;130(4):429-40.
2. Minckler D, Mosaed S, Dustin L. Trabectome Study Group. Trabectome (trabeculectomy-internal approach): additional experience and extended follow-up. Trans Am Ophthalmol Soc. 2008;106:149-59.
3. Craven E, Katz L, Wells J, Giamporcaro J. Cataract surgery with trabecular micro-bypass stent implantation in patients with mild-to-moderate open-angle glaucoma and cataract: two-year follow-up. J Cataract Refract Surg. 2012;38(8):1339–45.
4. Sameulson J, Katz L, Wells J, et al., US iStent Study Group. Randomized evaluation of the trabecular micro-bypass stent with phacoemulsification in patients with glaucoma and cataract. Ophthalmology. 2011;118(3):459–67.

Implant Size

A study comparing single and double-plated Molteno implants proposed that larger end plates provide a larger IOP reduction.[15] However, this data has not been thoroughly substantiated. End plate size does appear to have some effect on the IOP, although this may be true only to an extent. The ideal size of the end plate is not known, and research shows conflicting data.[16] A recent study comparing Baerveldt Glaucoma Implants (BGI) with end plate sizes of 250mm^2 and 350mm^2 concluded that there was no difference in surgical success, IOP, VA or topical medications used throughout a three-year period.[17]

Valved vs. Non-Valved

The design of a GDI may include the presence of a valve, which regulates aqueous flow through the tube to the end plate. This idea may seem contradictory to the intended purpose of a GDI; however, research shows the function to be an asset in the postoperative period. A valved GDI helps to restrict further aqueous outflow if the IOP drops too low. Furthermore, valved implants also appear to reduce the risk of postoperative hypotony.[13]

The drainage implants most commonly used are the Ahmed Glaucoma Valve (New World Medical) and the Baerveldt Glaucoma Implant (Advanced Medical Optics). Other implants that are also available include the Krupin slit valve (Hood Laboratories) and the Molteno implant (Molteno Ophthalmic Limited).[13]

Late Post-op GDI Complications

Glaucoma drainage implants have several complications not typically

seen with trabeculectomies. These complications include tube and plate migration, bleb encapsulation, diplopia and device exposure.[13]

Tube and plate migration can occur as a late postoperative complication for glaucoma drainage implants. Tube extenders are used to lengthen retracted tubes. A migrating plate occurs less commonly than tube migration, but can be corrected by resuturing the GDI.

Encapsulation of the bleb may result in a failure to control IOP after glaucoma drainage implant surgery. This complication is comparable to an encapsulated bleb that develops after trabeculectomy. Both are generally treated with topical antihypertensive medications.

Persistent restrictive strabismus may occur after a GDI because of scarring between the rectus or oblique muscles and the implant,

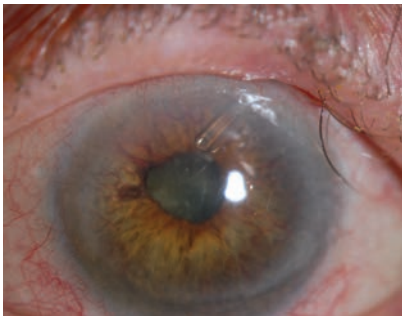


Image: Peter A. Netland, MD, PhD.

A patient with neovascular glaucoma after central retinal vein occlusion, with an Ahmed Glaucoma Valve.

or due to a crowding effect from a large bleb with limitation of extraocular motility. The most common treatment for diplopia secondary to drainage devices is prismatic correction, but muscle surgery or removal of the drainage implant may be necessary to negate the diplopia.

Partial or complete exposure of the GDI is one of the more frequent and challenging complications of glaucoma surgery. Exposure of the device is difficult to treat and may require removal of the device.

The Studies

Tube vs. Trabeculectomy Study

The Tube vs. Trabeculectomy (TVT) study was a multicenter randomized clinical trial comparing the safety and efficacy of tube shunt surgery with the Baerveldt glaucoma implant (BGI model 101-350) and trabeculectomy with mitomycin C in patients with previous cataract surgery, filtering surgery or both. Several interesting features can be teased out from this study through five years of follow up:[21]

- Tube shunt surgery with a GDI had a higher success rate than trabeculectomy with MMC during the first five years of the study.
- Long-term IOP was slightly lower with trabeculectomy over tube shunt.
- Early postoperative complications occurred more frequently after trabeculectomy with MMC than after GDI surgery.
- The rates of late postoperative complications and reoperation for complications were similar with both surgical procedures during five years of follow up.
- The rate of vision loss was similar between both groups.

Overall, the results demonstrated no clear superiority of one glaucoma operation over the other. Both surgical procedures are viable options for patients with previous cataract extraction, failed trabeculectomy, or both. The TVT Study does support the practice pattern shift of a trend towards the use of more GDIs in this population.[18]

GDI Studies

The Ahmed Baerveldt Comparison Study (ABC) compared the safety and efficacy rate of IOP lowering after implantation of the Ahmed Glaucoma Valve (AGV model FP7) and Baerveldt Glaucoma Implant (BGI model 101-350) devices in 267 refractory glaucoma patients. Both AGV and BGI devices reduced IOP and medication usage through five years. Both groups in the ABC study observed a greater than 50% reduction in IOP and similar rate of treatment failure, albeit for different reasons. Most AGV failures were attributed to high IOP, while most BGI failures were a result of low IOP or postoperative complications. One notable comparison is the finding that the BGI group had slightly lower IOPs at five years while taking considerably fewer postoperative glaucoma medications.[19,20]

The Ahmed Versus Baerveldt (AVB) study compared the long-term effectiveness of the AGV with the BGI in patients with refractory or high-risk glaucoma. The 238 patients enrolled in this study had

iStent Implant

By Barbara Fluder, OD

The iStent (Glaukos) is essentially a small snorkel comprised of heparin-coated titanium implanted nasally into Schlemm's canal during cataract surgery to improve aqueous outflow by creating a permanent conduit to the trabecular meshwork.

Contraindications: Primary or secondary angle closure, neovascular glaucoma, thyroid eye disease, Sturge-Weber syndrome or any other condition that may cause elevated episcleral venous pressure.

Preoperative care: According to the company's website, gonioscopy should be performed before surgery to rule out peripheral anterior synechiae, rubeosis and any angle abnormalities or conditions that would occlude sufficient visualization of the angle that could lead to improper placement of the stent.

Postoperative care: Cataract recovery drops as well as the patient's prescribed glaucoma medications.

uncontrolled IOP at the time of surgery, and many had a previously failed trabeculectomy. The AVB study found both GDIs to be effective at reducing intraocular pressure and the dependence on topical glaucoma medications. The Baerveldt group had a lower failure rate, but experienced more hypotony-related vision threatening complications through three years of follow up.[20]

The ABC and AVB studies have found both the Ahmed Glaucoma Valve and Baerveldt Glaucoma Implant to be effective surgical options for the management of refractory glaucoma. The studies also determined that AGVs were more likely to fail due to high intraocular pressure postoperatively whereas BGIs failed due to a low pressure and associated complications. These results can be partly explained by the presence of a flow-restricting valve on the AGV implant and the valveless design of the BGI. A GDI with a valve is advantageous for early postoperative IOP control and reduced hypotony risk.[18,21]

Your patients may be good candidates for incisional glaucoma surgery if they have advanced glaucoma, a form of secondary glaucoma, demonstrate significant IOP fluctuation or cannot tolerate medical treatment. Although the patient and ophthalmologist will ultimately make the surgical decision, it is important for optometrists to understand the basic concepts of incisional glaucoma surgery and potential postoperative complications. The initial glaucoma procedure for many ophthalmologists is a trabeculectomy; however, results from the TVT study show that the IOP reduction and overall surgical success is generally equal between a trabeculectomy and a glaucoma drainage implant. As such, the use of glaucoma drainage implants has increased in recent years. Patients with failed trabeculectomies or those with high risk may benefit from aqueous drainage devices. ■

Dr. Zimbalist and Dr. Gentry practice at the Harry S. Truman Memorial Veterans' Hospital in Columbia, Mo.

GDI Steps

A GDI is commonly performed using the following steps:[1,2]

- A conjunctival flap is created in the desired surgical location.
- The conjunctival flap is made large and thick to minimize conjunctival tearing and wound dehiscence.
- The base of the implant is slid under the conjunctiva near the recti muscle insertion and sutured to the sclera.
- The surgeon will use absorbable sutures with a "rip cord" or a releasable intraluminal stent with non-valved implants to help control the intraocular pressure postoperatively. Surgeons may also elect to use non-absorbable sutures at the tip of the tube for IOP control; this can be removed with laser at a later date.
- A small needle is introduced to the anterior chamber which serves as a conduit for the tube. The placement of the tube is the single most important factor of this surgery—with the ideal position being parallel and just anterior to the iris plane.
- A patch of sclera is placed over the anterior surface of the tube and is secured with several absorbable sutures.
- Small fenestrations are then made in the tube between the scleral graft and the intralumen stent to help maintain proper IOP control prior to the ligature being opened in the postoperative period.

1. Salim S, Kahook M. Aqueous Shunting Procedures. In: Kahook MY SJED, ed. Chander and Grant's Glaucoma. 5th ed. Thorofare NJ: Slack Inc; 2013.
2. Ayyala R. Seven easy steps allow Ahmed Glaucoma Valve implantation under modified topical anesthesia. Healio. April 1, 2003. Available at: http://www.healio.com/ophthalmology/glaucoma/news/print/ocular-surgery-news/%7B2be5c9fc-e104-4722-95b6-f6435407f7e0%7D/seven-easy-steps-allow-ahmed-glaucoma-valve-implantation-under-modified-topical-anesthesia. Accessed March 11, 2015.

1. Gray T, Fenerty C, Harper R, et al. Individualised patient care as an adjunct to standard care for promoting adherence to ocular hypotensive therapy: an exploratory randomised controlled trial. Eye (Lond). 2012 Mar;26(3):407-17.
2. Caprioli J, Coleman A. Intraocular pressure fluctuation a risk factor for visual field progression at low intraocular pressures in the advanced glaucoma intervention study. Ophthalmology. 2008;115(7):1123-9.
3. Hong S, Seong G, Hong Y. Long-term intraocular pressure fluctuation and progressive visual field deterioration in patients with glaucoma and low intraocular pressures after a triple procedure. Arch Ophthalmol. Aug 2007;125(8):1010-3.
4. Komori S, Ishida K, Yamamoto T. Results of long-term monitoring of normal-tension glaucoma patients receiving medical therapy: results of an 18-year follow-up. Graefes Arch Clin Exp Ophthalmol. 2014 December;252(12):1963-70.
5. AGIS Investigators. The Advanced Glaucoma Intervention Study (AGIS):7. The relationship between control of intraocular pressure and visual field deterioration. Am J Ophthalmol. Oct 2000;130(4):429-40.
6. Healthwise Staff. Trabeculectomy (Filtration Surgery) for Glaucoma. WebMD. February 28, 2012. Available at: http://www.webmd.com/eye-health/trabeculectomy-filtration-surgery-for-glaucoma. Accessed December 11, 2014.
7. Sugar H. Some recent advances in the surgery of glaucoma. Am J Ophthalmol. 1962;54:917-29.
8. Cairns J. Trabeculectomy. Preliminary report of a new method. Am J Ophthalmol. 1968;66:673-9.
9. Pia Uyloan de Guzman M. Trabeculectomy. eMedicine. Jan 22, 2014. Available at: http://emedicine.medscape.com/article/1844332-overview. Accessed Dec 08, 2014.
10. De Fendi L, Arruda G, Scott I, Paula J. Mitomycin C versus 5-fluorouracil as an adjunctive treatment for trabeculectomy: a meta-analysis of randomized clinical trials. Clin Experiment Ophthalmol. 2013 Nov;41(8):798-806.
11. Picht G, Grehn F. Classification of filtering blebs in trabeculectomy: biomicroscopy and functionality. Curr Opin Ophthalmol. 1998;9(2):2-8.
12. Vijaya L, Manish P, Ronnie G, Shantha B. Management of complications in glaucoma surgery. Indian J Ophthalmol. 2011;59:S131-40.
13. Schwartz K, Lee R, Gedde S. Glaucoma drainage implants: a critical comparison of types. Curr Opin Ophthalmol. 2006;17:181-89.
14. Hong C, Arosemena A, Zurakowski D, Ayyala R. Glaucoma drainage devices: a systematic literature review and current controversies. Surv Ophthalmol. Jan-Feb 2005;50(1):48-60.
15. Heuer D, Lloyd M, Abrams D, et al. Which is better? One or two? A randomized clinical trial of single-plate versus double-plate Molteno implantation for glaucomas in aphakia and pseudophakia. Ophthalmology. Oct 1992;99(10):1512-9.
16. Freedman J. Preoperative Evaluation of Patients Undergoing Drainage Implant Surgery. In: Shaarawy TM SMHRCJ, ed. Glaucoma. 2nd ed. China: Elsevier Limited; 2015.
17. Allan E, Khaimi M, Jones J, Ding K, Skuta G. Long-term Efficacy of the Baerveldt 250 mm(2) Compared with the Baerveldt 350 mm(2) Implant. Ophthalmology. Mar 2015;122(3):486-93.
18. Budenz D, Barton K, Gedde S, et al. Ahmed Baerveldt Comparison Study Group. Five-year treatment outcomes in the Ahmed Baerveldt Comparison Study. Ophthalmology. 2015;122(2):308-16.
19. Barton K, Feuer W, Budenz D, et al. Ahmed Baerveldt Comparison Study Group. Three-year treatment outcomes in the Ahmed Baerveldt Comparison Study. Ophthalmology. 2013;121(8):1547-57.
20. Christakis P, Tsai J, Kalenak J, et al. The Ahmed Versus Baerveldt Study: three-year treatment outcomes. Ophthalmology. 2013;120(11):2232-40.
21. Gedde S, Schiffman J, Feuer W, et al. Tube Versus Trabeculectomy Study Group. Treatment outcomes in the Tube Versus Trabeculectomy (TVT) Study after five years of follow-up. Am J Ophthalmol. 2012;153(5):789-803.

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More than Meds: Treating Glaucoma in the Real World

Focusing on patient quality of life is key to disease management.

By Murray Fingeret, OD, and Aliza Becker, Associate Editor

Clinicians today use more sophisticated technologies than ever before to manage patients through the onset and progression of glaucoma. While these technologies help us manage a patient's intraocular pressure (IOP) and other factors, optometrists must keep the patient's comfort, happiness and quality of life in mind when creating a treatment plan.

Traditionally, eye doctors would suggest glaucoma treatment begin when a patient's IOP measures 21mm Hg. However, researchers now believe that determining when to treat requires a multifaceted approach that uses a complex array of factors.[1] But, perhaps the time to start treating can't be based on numbers alone. In fact, the patient's ability to function visually day to day may be just as integral as any diagnostic measurement.

This article reviews the impact glaucoma has on our patient's lives and the measures we can take to help prevent it from robbing them of their independence.

Perception

Patient perception of quality of life depends on the severity of the disease—some patients with severe vision loss have indicated a willingness to trade years of their lives for better vision. A study comparing the responses of 228 patients with glaucoma to 12 blind patients and 12 controls with normal vision found 45 (20%) glaucoma patients were willing to trade time for improved vision, compared with six (50%) blind patients.[2] Glaucoma patients with counting fingers or worse vision in their better eye also indicated more willingness to trade time or risk death (i.e., standard gamble method) for better vision, compared with patients with 20/20 or 20/25 in their better eye.[3] Patients with more severe binocular visual field loss also reported feeling less confident and more anxious during certain daily activities, such as crossing the street.[4]

Autonomy

Difficulty with reading is one of the most frequent complaints among people with glaucoma, and the most common cause for low vision referrals.[4] On average, those with advanced bilateral visual field loss have been shown to read an average of 29 words per minute slower and are twice as likely to make a mistake than those without glaucoma.[5] Silent reading skills are especially affected: patients with glaucoma exhibited a 16% decline in silent reading speed over 30 minutes compared

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Glaucoma's effect on peripheral visual acuity can leave many patients feeling anxious or apprehensive about driving.

with controls, but only a 7% decline of out-loud reading speed.[6]

Independent mobility, which is critical to living outside of an assisted living or nursing home setting, is another function impacted by glaucoma progression. An increased risk for falls as a result of peripheral visual field loss is one of the primary factors leading to a more sedentary lifestyle.[7] This can put patients at risk for heart disease, diabetes and bone thinning, among others.[8]

Even if glaucoma patients remain active, they may be less so than their healthy counterparts: patients with glaucoma have been shown to participate in 3.2 minutes less (12.9 vs. 16.1 minutes) of moderate or vigorous physical activity and take 887 fewer steps (5,004 vs. 5,891 steps) than patients with normal vision.[9] Patients with bilateral glaucoma also walk an average of 2.4m/minute slower around obstacles and experience 1.65 times the number of bumps, compared to those without glaucoma, according to research published in 2012.[10] Interestingly, performance of those with unilateral glaucoma was not significantly different statistically compared with normal controls.[10]

Due to its effect on the patient's peripheral vision, glaucoma can also impede safe operation of a motor vehicle. On average, every 5dB of visual field loss in the better eye doubles a patient's odds of driving cessation.[11] For those who drive despite vision loss, simulations show they are three times more likely to have an accident compared with normal controls.[12]

Treatment Adherence

Because they are faced with so many potential impacts to quality of life as a result of glaucoma progression, we'd expect patients to strictly adhere to their treatment regimen. Getting some patients to use their eye drops, however, is an uphill battle, especially if they do not believe vision loss will occur or if they have difficulty using—or even remembering to use—the drops.[13]

Lack of transportation to the pharmacy and the doctor's office also impacts treatment adherence, as patients are severely deterred from refilling their prescriptions or attending follow-up visits. Conversely, those who travel more frequently are also less likely to take their drops due to the change in routine.

Patients with poor understanding of the disease and its progression are less likely to be compliant with administering their drops. Thus, it is imperative that doctors and patients communicate effectively. A study assessing interactions between doctors and patients found doctors typically did the majority of the talking during appointments (saying roughly 70% of the words) and asked two-thirds of the questions.[14] The majority of these questions were closed-ended (94%), and patients were rarely questioned about their opinion or understanding of their disease and how it affected them.[14] Thus, asking open-ended questions (i.e., "How are you doing with your drops?" rather than "Are you taking your drops?") in a non-judgmental environment may help increase compliance.[15] Improving educational efforts in the office may also improve understanding and subsequent compliance.[16]

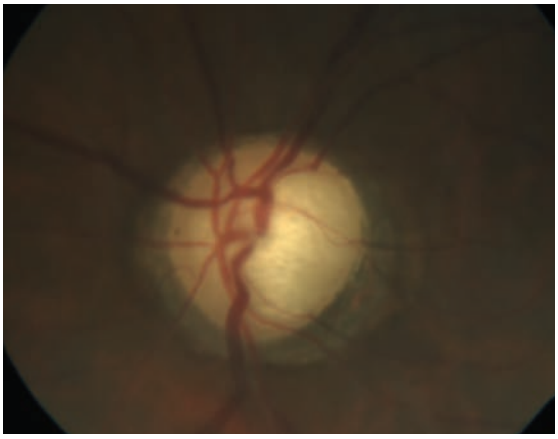

Photo: Marta Fabrykowski, OD

See the patient as more than just a glaucomatous nerve. Understanding and discussing glaucoma's impact on their life will strengthen your rapport with the patient.

Patients who need to administer glaucoma medicine more than twice daily and those who take different medications for multiple conditions are also less adherent to their glaucoma treatment regimen, research shows.[17] Electronic monitoring of glaucoma patients also revealed that patients report higher medication use than their actual behavior; in fact, nearly 45% of patients who knew they were being monitored and who were receiving free medication used their drops less than 75% of the time.[18] Research shows automatic reminders, either through a telecommunication-based system managed by the doctor's office or patient self-initiation, improve compliance.[15,19]

With respect to compliance attending follow-up visits, noncompliant patients are: more likely to be suspects for glaucoma rather than exhibiting definite signs of the disease; be dissatisfied with the wait time for or cost of the examination; and be noncompliant with medicine use.[20] Poor compliance with follow-up visits has been linked to increased disease severity.[21] ■

Dr. Fingeret is chief of the optometry section at the VA New York Harbor Healthcare System's Brooklyn Campus. He is a founding member and past president of the Optometric Glaucoma Society.

1. Detry-Morel M. Currents on target intraocular pressure and intraocular pressure fluctuations in glaucoma management. Bull Soc Belge Ophthalmol. 2008:(308):35-43.
2. Jampel HD. Glaucoma patients' assessment of their visual function and quality of life. Trans Am Ophthalmol Soc. 2001;99:301-17.
3. Brown GC. Vision and quality-of-life. Trans Am Ophthalmol Soc. 1999;97:473-511.
4. Nelson P, Aspinall P, O'Brien C. Patients' perception of visual impairment in glaucoma: a pilot study. Br J Ophthalmol 1999;83:546-52.
5. Ramuli PY, West SK, Munoz B, et al. Glaucoma and reading speed; the Salisbury eye evaluation project. Arch Ophthalmol. 2009 Jan:127(1):82-7.
6. Ramulu PY, Bonnielin KS, Jefferys JL, et al. Difficulty with out-loud and silent reading in glaucoma. Invest Ophthalmol Vis Sci. 2013 Jan;54(1):666-72.
7. Freeman EE, Munoz B, Rubin G, West SK, et al. Visual field loss increases the risk of falls in older adults: the Salisbury eye evaluation. Invest Ophthalmol Vis Sci. 2007 Oct:48(10):4445-50.
8. Warburton DER, Nicol CW, Bredin SSD. Health benefits of physical activity: the evidence. CMAJ. 2006 Mar 14:174(6):801-9.
9. Friedman DS, Freeman E, Munoz B, et al. Glaucoma and mobility performance: the Salisbury Eye Evaluation Project. Ophthalmology. 2007 Dec;114(12):2232-7.
10. Ramulu PY, Maul E, Hochberg C, et al. Real-world assessment of physical activity in glaucoma using an accelerometer. Ophthalmology. 2012 Jun:119(6):1159-66.
11. Ramulu PY, West SK, Munoz B, et al. Driving cessation and driving limitation in glaucoma: the Salisbury Eye Evaluation Project. Ophthalmology. 2009 Ot:116(10):1846-53.
12. Szlyk JP, Mahler CL, Seiple W, et al. Glaucoma and on-road driving performance. IOVS. 2008;49:3035-41.
13. Stryker JE, Beck AD, Primo SA, et al. An exploratory study of factors influencing glaucoma treatment adherence. J Glaucoma. 2010 Jan:19(1):66-72.
14. Friedman DS, Hahn SR, Quigley HA, et al. Doctor-patient communication in glaucoma care: analysis of videotaped encounters in community-based office practice. Ophthalmology. 2009 Dec;116(12):2277-85.
15. Chang JS, Lee DA, Petursson G, et al. The effect of a glaucoma medication reminder cap on patient compliance and intraocular pressure. J Ocul Pharmacol. 1991 Summer;7(2):117-24.
16. Friedman DS, Hahan SR, Gelb L, et al. Doctor-patient communication, health-related beliefs and adherence in glaucoma results from the Glaucoma Adherence and Persistency Study. Ophthalmology. 2008 Aug:115(8):1320-7.
17. Gurwitz JH, Glynn RJ, Monane M, et al. Treatment for glaucoma: adherence by the elderly. Am J Public Health. 1993 May:83(5):711-6.
18. Okeke CO, Quigley HA, Jampel HD, et al. Adherence with topical glaucoma medication monitored electronically the Travatan Dosing Aid study. Ophthalmology. 2009 Feb;116(2):191-9.
19. Boland MV, Chang DS, Frazier T, et al. Automated telecommunication-based reminder and adherence with once-daily glaucoma medication dosing: the automated dosing reminder study. JAMA Ophthalmol. 2014 Jul:132(7):845-50.
20. Kosoko O, Quigley HA, Vitale S, et al. Risk factors for noncompliance with glaucoma follow-up visits in a residents' eye clinic. Ophthalmology. 1998 Nov;105(11):2105-11.
21. Ung C, Murakami Y, Zhang E, et al. The association between compliance with recommended follow-up and glaucomatous disease severity in a county hospital population. Am J Ophthalmol. 2013 Aug;156(2):362-9.
22. Lichter PR, Musch DC, Gillespie BW, et al. CIGTS Study Group. Interim clinical outcomes in the Collaborative Initial Glaucoma Treatment Study comparing initial treatment randomized to medications or surgery. Ophthalmology. 2001;108(11):1943-53.
23. The AGIS Investigators. The Advanced Glaucoma Intervention Study (AGIS), 7: the relationship between control of intraocular pressure and visual field deterioration. Am J Ophthalmol. 2000;130(4):42-40.
24. Heijl A, Leske MC, Bengtsson B, et al. Early Manifest Glaucoma Trial Group. Reduction of intraocular pressure and glaucoma progression: results from the Early Manifest Glaucoma Trial. Arch Ophthalmol. 2002;120(10):1268-79.
25. Glaucoma Research Foundation. How compliance can help save your sight. www.glaucoma.org/treatment/how-compliance-can-help-save-your-sight.php.
26. Sleath B, Blalock S, Covert D, et al. The relationship between glaucoma medication adherence, eye drop technique, and visual field defect severity. Ophthalmology. 2011 Dec;118(12):2398-402.
27. Sleath B, Blalock SJ, Carpenter DM, et al. Ophthalmologist–patient communication, self-efficacy, and glaucoma medication adherence. Ophthalmology. 2014 Dec 24. [Epub ahead of print.].
28.American Academy of Ophthalmology. More than half of people with glaucoma skip or improperly administer medications, risking permanent vision loss. 2015 Jan. www.aao.org/newsroom/news-releases/detail/more-than-half-of-people-with-glaucoma-skip-improp#_edn3.

Five Tips For Improving Glaucoma Patients' Medication Adherence

Medication adherence for glaucoma patients is an ongoing battle for eye doctors. While effective medical treatment can prevent most vision loss, as many as 25% of glaucoma patients don't take their meds.[22-25] Here are five tips to help you improve your patients' adherence to their medical treatment:

1. Build trust with your patients. Your patients need to feel comfortable admitting they are not keeping up with the treatment plan. Their honesty will allow you to address the problem swiftly. Research suggests skipping doses can increase disease severity, so handling your patients' issues as soon as they arise can significantly impact the progression of their glaucoma.[26]

2. Educate, educate, educate. Recent research suggests educating patients on how to use their glaucoma drops properly is the only communication factor that can increase medication adherence.[27] Show every patient how to pull and pinch the lower lid to create a pocket and instill the drops.[28] They should then close their eyes and apply pressure to the inner corners, without blinking, for two to three minutes.[28] You can demonstrate this as many times as necessary, even during repeat visits, to make sure patients are doing it correctly. Create a tip sheet patients can take home.

3. Provide written instructions. Many glaucoma patients struggle to remember complicated treatment regimens, especially if it involves multiple prescriptions with specific dosing. Write out the required steps in large, clear font so patients can refer back to these directions when they leave your office.

4. Offer reminder strategies for taking medication. Suggest setting a daily alarm or mark off each day on the calendar when drops are to be taken. Patients can integrate their medication regimen into their daily routine by storing the drops next to their toothbrush or pillbox to serve as a visual reminder. If they continue to struggle, recommend they ask a caregiver or family member to accompany them to the appointment. The second person can help remind the patient of the treatment plan at home.

5. Make sure patients understand their insurance coverage. Patients, especially those new to using glaucoma drops, may find themselves refilling the prescription early, and insurance companies may handle this differently. For example, Medicare Part D will only cover prescription refills after 70% of the predicted time has elapsed (i.e., 21 days for a 30-day supply).[28]

GLAUCOMATOCYCLITIC CRISIS:

A Not-So-Benign Disease?

Do you know how to recognize and manage this potentially devastating condition?

By Rim Makhlouf, OD, and Joseph Sowka, OD

Glaucomatocyclitic crisis (GCC), or Posner-Schlossman syndrome, was first described by Posner and Schlossman in 1948 as a rare, recurrent and typically unilateral inflammatory ocular hypertensive disease.[1] It generally affects one eye at a time, and its recurrence usually afflicts the same eye. Bilateral and simultaneous involvement is very uncommon. The individual attacks may last from a few hours to a few weeks, but rarely persist over two weeks. Episodes may occur with varying frequency and without any apparent cause. It can affect adults of all ages (reports range from 23 to 67 years), especially between the third and sixth decade of life.[2,3] Only two pediatric cases of GCC have been reported.[2,4] One study looking at population statistics in Finland found the prevalence to be 1.9 per 100,000.[5] No racial or sexual predilection has been reported.

The classical presentation of GCC is a unilateral ocular hypertension with mild anterior segment inflammation (cyclitis) and few clinical symptoms. The elevation of intraocular pressure (IOP), which can range from 40mm Hg to 70mm Hg or above, is out of proportion with the minimal symptoms reported by the patient and is characteristic of this inflammatory disease. Recurrent episodes further help confirm the diagnosis. During intervals between attacks, the patient is asymptomatic.

Pathophysiology

The cause and pathogenesis of GCC remains poorly understood, in part because the mild anterior chamber reaction noted during attacks does not correlate with the magnitude of IOP elevation, as opposed to what is usually seen in uveitic glaucoma. It is therefore believed that the locus of the inflammation is the trabecular meshwork, leading to decreased aqueous outflow, which in turn leads to elevated IOP. However, it is not yet clear what causes the inflammation and whether the mechanism is infectious in nature or strictly inflammatory.

Using polymerase chain reaction analysis, studies investigating aqueous humor composition during acute attacks of GCC have not found a conclusive causative factor. In one study, examination of the aqueous humor identified the presence of cytomegalovirus (CMV) but not other herpes viruses.[6] Another study found herpes simplex virus (HSV) within the aqueous while testing negative for varicella zoster virus (VZV) or CMV.[7] Yet more studies that analyzed the contents of the aqueous humor found that, during the acute phase, there is a measurable increase in the aqueous levels of prostaglandins, particularly prostaglandin E, which levels have been found to positively correlate with IOP.[7] During periods of remission, the study also dem-

onstrated these same levels were markedly decreased with concurrent increased aqueous outflow.

A likely hypothesis about the exact relationship between prostaglandin induction and increased IOP is a dual mechanism characterized by the stimulation of aqueous production as well as the inhibition of its outflow. The outflow inhibition caused by breakdown of the blood-aqueous barrier, leading to the release of inflammatory cells, which results in clogging of the trabecular meshwork.[8,9] However, it has not yet been shown whether the presence of prostaglandin is causative of the disease or simply an epiphenomenon of an underlying mechanism.

A recent article analyzing the different aqueous humor composition studies argued that the most reliable and reproducible studies—taking into consideration the sample size and whether the study included testing for other particles—were those that found CMV had the greatest association with GCC.[10] However, the exact mechanism of GCC, as well as whether the presence of viral particles is causative of the disease or is indirectly related to it through an unknown mechanism, is still unclear.[11]

Fig. 1. Small, centrally located keratic precipitates in an eye with glaucomatocyclitic crisis.

Clinical Presentation

Patients typically present during active episodes of GCC with symptoms ranging from slight orbital or ocular discomfort to colored halos or blurred vision resulting from corneal edema caused by elevated IOP. In cases where IOP is very high, significant decrease in visual acuity due to corneal edema and pain may be present.[2]

Clinical findings often include markedly elevated IOP, open anterior chamber angles seen by gonioscopy, mild cyclitis presenting as rare aqueous inflammatory cells and a few small keratic precipitates *(Figure 1)*. IOP may increase to as much as 70mm Hg or more in some cases. Conjunctival hyperemia is usually absent unless IOP is very high, in which case mild congestion may be noted. Given the small degree of anterior chamber inflammatory reaction, posterior synechiae are not typically present. Similarly, peripheral anterior synechiae are also absent. Heterochromia can be present where the affected eye is light-colored due to either sectoral or diffuse iris atrophy resulting from prolonged IOP elevation and tissue ischemia.

In general, optic discs are normal and no visual defects are noted, especially with initial episodes of GCC. However, repeated or prolonged episodes can cause typical glaucomatous damage. Additionally, two cases of non-arteritic anterior ischemic optic neuropathy (NAAION) associated with GCC attack have been published.[12,13] One postulated that GCC-induced elevated IOP, along with risk factors such as systemic conditions (e.g., hypertension and diabetes) and a "disc at risk" (i.e., crowded disc and minimal physiological cup), contributed to a decreased ocular disc perfusion, leading to vision loss and subsequent optic nerve atrophy.[12]

The original description of GCC by Posner and Schlossman stated that during intervals between attacks, no sign of inflammation is present, and IOP is generally normal.[1] However, some patients may show slightly elevated pressure in both the affected and unaffected eye.[1] One study that followed patients with GCC between attacks identified a high incidence of elevated IOP, decreased facility of aqueous outflow, higher IOP response to topical steroids, glauco-

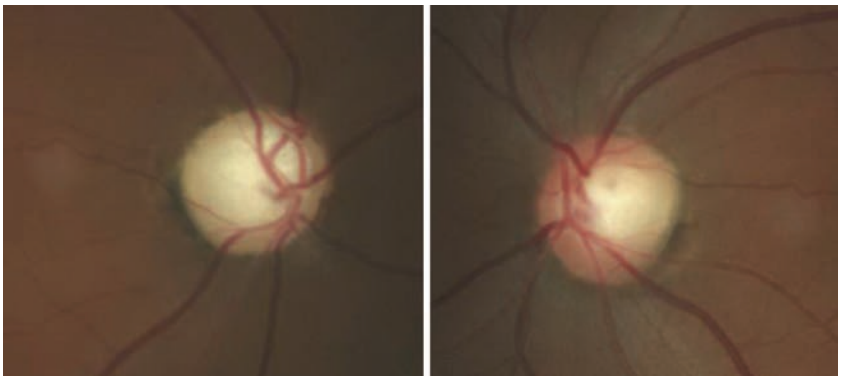

Fig. 2. Glaucomatous optic nerve damage in the right eye of a patient with GCC and repeated IOP elevations exceeding 55mm Hg.

matous disc damage and visual field loss in the non-affected and GCC-involved eye.[14] These observations indicate that patients with GCC are at a greater risk of developing primary open angle glaucoma in either eye.[14]

Differential Diagnoses

Several diseases can be easily confused with GCC. These include:

• ***Acute Angle-closure Glaucoma.*** This condition presents with symptoms of unilateral pain, redness, blurry vision, headaches, nausea and vomiting. Clinical signs include a closed angle seen by gonioscopy, peripheral anterior synechiae, corneal edema and markedly increased IOP. Glaucomatous damage typically ensues quickly if the attack is not managed; this is considered an ocular emergency.

• ***Uveitic Glaucoma.*** This condition has elevated IOP often associated with a significant anterior chamber reaction, which is not present in GCC.

• ***Fuchs' Heterochromic Iridocyclitis.*** This condition is a chronic mild cyclitis usually associated with heterochromia. Similar to GCC, it is unilateral with an absence of acute symptoms. However, the increase in IOP that results from this disease is rarely as high as seen in GCC. Fuchs' heterochromic iridocyclitis is associated with cataract formation and does not respond to anti-inflammatory agents.

• ***Herpetic Keratouveitis.*** This condition clinically presents as a painful, red eye—in contrast to the less inflamed eye often seen with GCC—with elevated IOP and mild anterior segment inflammation. Patients often have a history of a previous ocular herpetic infection.

When presenting as a primary infection, herpetic keratouveitis rarely comes in the form of trabeculitis or cyclitis; however, lid lesions, follicular conjunctivitis or epithelial corneal dendrites are commonly found. Reactivation of the latent virus after primary infection can lead to more severe ocular complications, including stromal keratitis, trabeculitis, uveitis and endothelialitis.[14] When trabeculitis presents alone, possible features that can help in the differential diagnosis are the presence of eyelid or epithelial scars and loss of corneal sensitivity from a previous primary infection.

Management

Glaucomatocyclitic crisis is generally considered a self-limited condition because the inflammation and subsequently elevated IOP will subside by itself within a few days to a few weeks. However, when it occurs, exceptionally high IOP during the crisis can cause permanent nerve damage if left untreated. Also, due to the recurrent nature of the disease, the repetitious IOP elevation can result in glaucomatous vision loss over time *(Figures 2 and 3)*.

Given that the mechanism of the disease is primarily inflammatory, the management of GCC largely includes treating the underlying inflammation with a short course of topical steroids. Additionally, the initial management includes lowering IOP with topical anti-glaucomatous medications. Successful management with β-blockers, α-agonists, carbonic anhydrase inhibitors (CAIs) or a combination of the three, has been reported.[16,17] Prostaglandin analogs and miotics should be generally avoided since these agents are known to potentially increase inflammation. Anti-inflammatory and anti-glaucoma medications should be maintained until complete resolution of the attack. Second-line therapy may include oral CAIs to manage the elevated IOP as well as topical NSAIDs, oral NSAIDs or both, to control the inflammation.

In patients with multiple recurrences or prolonged attacks, more definitive surgery has been proposed to fully control IOP while avoiding the complications of long-term topical steroid use. Trabeculectomy has been shown to be a safe and effective method in GCC patients and should be considered as soon as they start showing glaucomatous changes, since progression can be quite rapid in these patients.[3,14,18,19] As a filtering procedure, it provides an alternative pathway for aqueous outflow and thus prevents spikes during episodes of cyclitis.[3]

Long-term management should include periodic observation in order to detect a possible conversion to open angle glaucoma in either eye, which has a higher incidence among GCC patients. The risk of developing open angle glaucoma is directly related to the number and duration of GCC attacks.[3] Patients who present with longer duration attacks should be monitored more carefully.

No definitive treatment has been reported for prevention of GCC recurrences. Given the strong association with CMV, investigators have tried some promising antiviral treatments. Thus far, topical ganciclovir 0.15% has demonstrated the greatest potential in decreasing the recurrence of GCC attacks with the least amount of side effects.[11] One study demonstrated topical ganciclovir was effective for clearing the viral load, assisting in IOP control and preserving the corneal endothelium of CMV-positive GCC patients.[20] Researchers surmised topical ganciclovir could assist in the management of patients with GCC, possibly decreasing the risk of advanced glaucoma and helping avoid glaucoma surgery in long-lasting cases.[20]

Another study indicated that long-term oral therapy with Valcyte (valganciclovir, Hoffmann–La Roche) 900mg BID for three weeks, followed by 450mg BID for a mean period of 20 months, lowered the recurrence rate in patients with GCC.[21] Two of the 11 patients studied had recurrence once the medication was stopped, but the others remained disease-free.[21] More studies are needed to test the efficacy and side effects resulting from the use of these drugs for extended periods.

While GCC typically presents with few, if any, symptoms, there are exceptions where the patient may present in acute distress. Considering GCC in the differential diagnosis in the presence of an acute unilateral elevated IOP is important, as proper management and treatment can prevent vision loss in some cases. Taking a detailed history and evaluating the anterior segment, including gonioscopy, plays a key role in the correct diagnosis. Additionally, while GCC is sometimes considered a benign, self-limiting condition, it is clear that some patients experiencing recurrent attacks can develop glaucomatous vision loss.

Patient education should stress the recurrent nature of the disease and the potential for visual compromise, as well as the importance of regular follow ups even between the attacks. ■

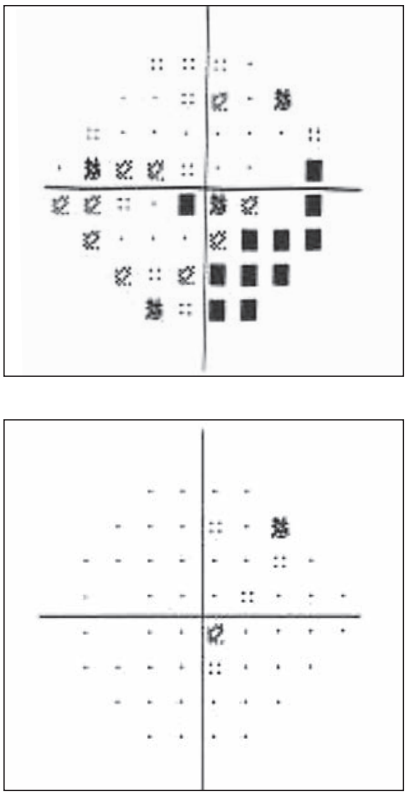

Fig. 3. Visual field testing reveals glaucomatous field loss OD.

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1. Posner A, Schlossman A. Syndrome of unilateral recurrent attacks of glaucoma with cyclitic symptoms. Arch Ophthal. 1948;39:517-35.
2. Burnstein Y, Shelton K, Higginbotham EJ. Glaucomatocyclitic crisis in a child. Am J Ophthalmol. 1998;126:136-7.
3. Jap A, Sivakumar M, Chee SP. Is Posner Schlossman syndrome benign? Ophthalmology. 2001;108:913-18.
4. Teekhasaenee C, Ritch R. Glaucomatocyclitic crisis in a child. Am Journal Ophthalmol. 1999;127:626-7.
5. Paivonsalo-Hietanen T, Tuominen J, Vaahtoranta-Lehtonen H, et al. Incidence and prevalence of different uveitis entities in Finland. Acta Ophthalmol Scand. 1997;75(1):76-81.
6. Bloch-Michel E, Dussaix E, Cerqueti P, Patarin D. Possible role of cytomegalovirus infection in the etiology of the Posner-Schlossmann syndrome. Int Ophthalmol. 1987;11:95-6.
7. Yamamoto S, Pavan-Lagston D, Tada R, et al. Possible role of herpes simplex virus in the origin of Posner-Schlossman syndrome. Am J Ophthalmol.1995;119:796-8.
8. Eakins KE. Increased intraocular pressure produced by prostaglandins E1 and E2 in the cat eye. Exp Eye Res. 1970;10:87-92.
9. Grant WM. Clinical measurements of aqueous outflow. Am J Ophthalmol. 1951;34:1603-5.
10. Takusagawa HL, Liu Y, Wiggs JL. Infectious theories of posner-schlossman syndrome. International Ophthalmology Clinics. 2011;51:105-15.
11. Teoh SB, Thean L, Koay E. Cytomegalovirus in aetiology of Posner-Schlossman syndrome: evidence from quantitative polymerase chain reaction. Eye. 2005;19:1338-40.
12. Irak I, Katz BJ, Zabriskie NA, et al. Posner-Schlossman syndrome and nonarteritic anterior ischemic optic neuropathy. J Neuroophthalmol., 2003;23:264-7.
13. Kim R, Van Stavern G, Juzych M. Nonarteritic anterior ischemic optic neuropathy associated with acute glaucoma secondary to Posner-Schlossman syndrome. Archives Ophthalmol, 2003;121:127-28.
14. Kass MA, Becker B, Kolker AE. Glaucomatocyclitic crisis and primary open-angle glaucoma. Am J Ophthalmol.1973;75:668-73.
15. Saini JS, Agarwala R. Clinical pattern of recurrent herpes simplex keratitis. Indian J Ophthalmol. 1999;47:11-4.
16. Muthusamy P. Apraclonidine in the management of glaucomatocyclitic crisis. Eye (London, England); 1994;8: 367-368.
17. Hong C, Song KY. Effect of apraclonidine hydrochloride on the attack of Posner-Schlossman syndrome. KJO, 1993;7:28-33.
18. Dinakaran S, Kayarkar V. Trabeculectomy in the management of Posner-Schlossman syndrome. Ophthalmic Surg Lasers. 2002;33:321-22..
19. Zhong Y, Cheng Y, Liu X, Feng P. Trabeculectomy in the management of glaucomatocyclitic crisis with visual field defect. Ocul Immunol Inflamm. 2012;18:233-36.
20. Su CC, Hu FR, Wang TH, et al. Clinical outcomes in cytomegalovirus-positive Posner-Schlossman syndrome patients treated with topical ganciclovir therapy. Am J Ophthalmol. 2014;158(5):1024-3.
21. Sobolewska B, Deuter C, Doycheva D, Zierhut M. Long-term oral therapy with valganciclovir in patients with Posner-Schlossman syndrome. Graefes Arch Clin Exp Ophthalmol. 2014;252(1):117-24.

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21st Annual Glaucoma Report

The Structure-Function Junction

Improve your glaucoma diagnostic abilities by looking beyond the OCT.

By Craig Thomas, OD

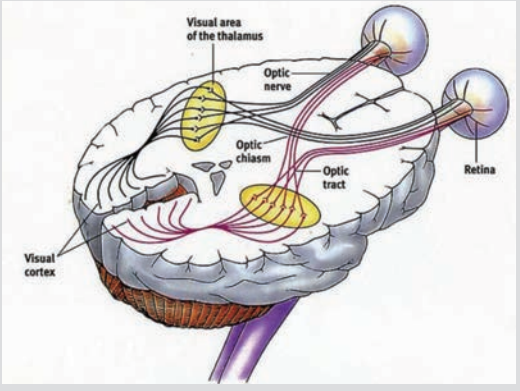
All practicing optometrists are tasked with evaluating the likelihood of glaucoma development in each of our patients. Fortunately, for most individuals the risk is low. However, population studies show that people with early glaucoma see the optometrist again and again without being diagnosed. As a result, patients are unnecessarily losing vison.[1]

Diagnosing this "sneak thief of sight" is often a subjective process that requires a multifaceted, patient-by-patient approach.

Traditionally, we've been told that structural damage must precede visual field loss.[2] New research challenges the conventional wisdom that said at least 25% to 35% of retinal ganglion cells must be lost before the first visual field defect appears.[2]

Although detectable structural abnormality may precede functional abnormality in some patients, the opposite may be true in others.[2] Complicating the issue is the fact that functional abnormalities vary widely between individual patients and on repeated measurements.[2] As a result of this lack of consistency

The Visual Pathway



Structures involved in the transmission of sensory information along the afferent visual sensory system (e.g., visual pathway).

Receptors
- **Rod cells in retina.**
- **Cone cells in the retina.**

Transmitters
- **Optic nerve.**
- **Optic chiasm.**
- **Optic tract.**
- **Lateral geniculate nucleus.**
- **Optic radiations.**

Visual Cortex
- **Receives axonal projections from the lateral geniculate nucleus and uses neural circuits to process visual information.**

Release Date: July 2015
Expiration Date: July 1, 2018
Goal Statement: Glaucoma's prevalence continues to increase because of an aging majority population and significant growth in high-risk populations. Evidence-based medicine demonstrates that combining functional testing with structural imaging can improve diagnosis and detection of early glaucoma. This article reviews the natural history of glaucoma and the current applications of functional testing.

Faculty/Editorial Board: Craig Thomas, OD
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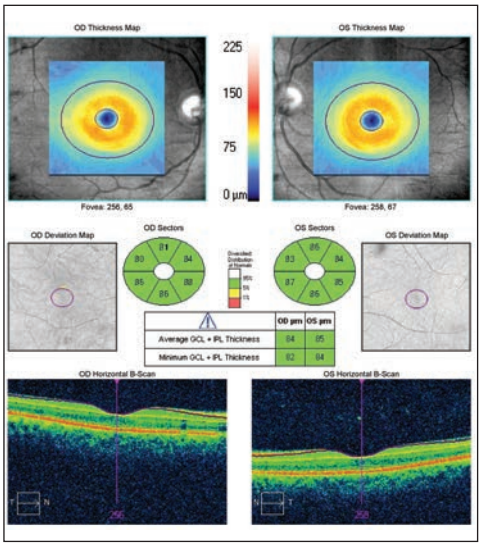
Disclosure Statement: Dr. Thomas is a paid consultant for Konan Medical and Johnson & Johnson Vision Care.

in diagnostic test results, optometrists should use measurements in one domain (structure or function) to support the interpretation of measurements in the other.[2] In other words, doctors today must decode a combination of diagnostic test results to deliver a comprehensive assessment of a patient suspected of developing glaucoma.

This article explains the why and how of a comprehensive examination that includes both structural and functional testing and how recognizing the value of functional testing can help us better diagnose glaucoma earlier.



Normal ganglion cell complex measured in both eyes despite the presence of retinal nerve fiber layer defects in each eye.

Natural History

Primary open-angle glaucoma is characterized by structural changes to the optic nerve head and peripapillary retina that are associated with characteristic functional deficits. In patients suspected of developing glaucoma, structural investigations of the eye should focus on the retinal ganglion cells and their axons. For these, we use imaging devices such as optical coherence tomography (OCT) to provide quantification of the neuroretinal rim area, retinal nerve fiber layer thickness and macular ganglion cell analysis.[2]

In addition to progressive ganglion cell loss, glaucoma is associated with aqueous outflow restrictions, nonphysiologic intraocular pressure, abnormal ocular perfusion and an abnormal rate of cellular apoptosis.

Today, researchers believe glaucoma is a neurodegenerative disease that affects the brain as well as the eye. Clear evidence from electrophysiologic studies shows glaucoma-induced damage is present throughout the neural pathways of the brain.[3]

The first indication of glaucoma-induced neurodegenerative brain damage is early impairment of the ganglion cells. The second indication involves the impairment of the brain's parallel ganglion cell pathways secondary to transynaptic degeneration and the third indication involves impairment of brain function at the level of the lateral geniculate nucleus.[3]

The Case for Functional Tests

In progressive glaucoma, a structure-function relationship exists between optic disc cupping and changes in the visual field.[2] Research shows that, for most people, minimal field change occurred when the vertical cup-to-disc ratio enlarged from 0.30 to 0.60 and that more marked visual field changes occurred when the cup-to-disc ratio enlarged from 0.60 to 1.0.[2] In the majority of patients with glaucoma, there is a functional latency period where structural change occurs early in the natural history of the disease without functional vision loss.[2] In other words, especially in early glaucoma, we usually see a greater amount of glaucoma-induced structural damage relative to the amount of glaucoma-induced functional damage. Alternatively, in more advanced glaucoma, it appears as if functional vision loss changes at a greater rate than structural damage.[2]

This curvilinear relationship between structural measures of glaucomatous optic atrophy and visual field measurements explains the contemporary opinion of a "functional reserve" period in most patients with early glaucoma where the optic nerve gets worse but the visual field does not.

Even though most patients with glaucoma demonstrate this functional latency period early in the natural history of their disease, some do not. Substantial evidence shows some patients with glaucoma develop visual field defects before structural defects.[2]

One explanation for this finding is the concept of ganglion cell dysfunction. In early glaucoma, ganglion cells may become dysfunctional before they die.[2] This dysfunction produces a reduction in visual field

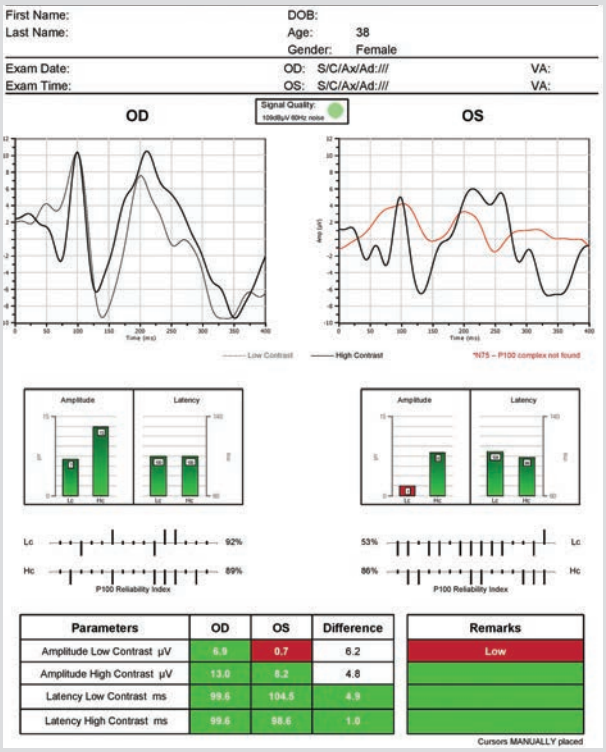
Structural Changes and Functional Deficits

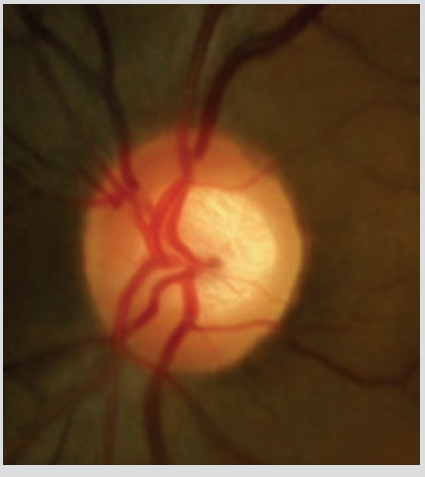
STRUCTURAL CHANGES	FUNCTIONAL DEFICITS
• Progressive loss of retinal ganglion cells • Enlargement of optic cup • Changes in optic nerve head coloration • Fallout of the retinal nerve fiber layer • Wipeout of the neuroretinal rim	• Visual field defects • Abnormal visual evoked potential waveforms • Abnormal electroretinography waveforms • Relative afferent pupillary defect • Loss of chromatic discrimination

At right, the NOVA-VEP (Diopsys) uses a pattern-reversal stimulus protocol elicited by checkerboard stimuli. The test displays the waveform responses with traces on a two-dimensional graph. The low contrast trace represents the integrity of the magnocellular neural pathway and the high-contrast trace represents integrity of the parvocellular neural pathway. Although glaucoma-induced VEP abnormalities can exhibit the same variability inherent in other functional tests in patients with glaucoma, the following abnormalities are the typical findings in patients with glaucoma:

- **Delayed P100 peak time of the low-contrast response.**
- **Delayed P100 amplitude of the low-contrast response.**
- **Wave shape perturbation.**

Below, mild glaucomatous optic atrophy in the left eye—normal neuroretinal rim appearance and normal optic disc coloration in both eyes—no asymmetry.





Sponsel WE, Groth SL, Satsangi N, et al. Refined data analysis provides clinical evidence for central nervous system control of chronic glaucomatous neurodegeneration. Trans Vis Sci Technol. 2014 May;3(3):1.

sensitivity that does not correlate with OCT measurements of the retinal nerve fiber layer or ganglion cell complex. It also explains how patients can develop measurable visual field defects before they develop clinically measurable structural damage.[2]

Standard Automated Perimetry

The traditional method of measuring glaucoma-induced functional abnormality is standard automated perimetry (SAP). In fact, when most of us talk about functional changes in glaucoma, we are usually talking about changes in the visual field measured by an automated threshold perimeter. However, a disturbance of any test of visual function can indicate glaucoma-induced functional changes.

To begin the functional testing component of your glaucoma evaluation, most optometrists perform a threshold visual field examination. Automated threshold perimeters measure the visual field by plotting the threshold luminescence value of the patient in various locations of the visual field. The luminescence of the light stimulus is represented by nonspecific units of measurement called decibels (dB).

Automated perimeters characterize specific parameters of the overall visual field status by the use of numbers called global indices. Two of the indices, mean deviation (MD) and pattern standard deviation (PSD), express the raw data generated by the instrument.

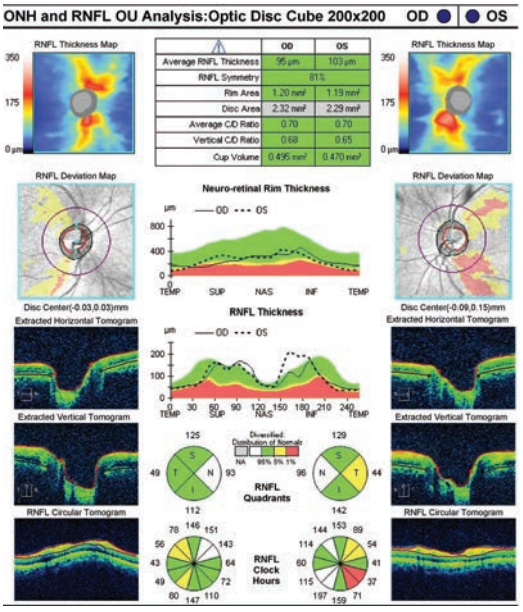
A visual field defect can be classified as mild, moderate or advanced based upon an abnormal mean deviation:

- Mild: 0.00dB through -5.99dB.
- Moderate: -6.00dB through -11.99dB.
- Advanced defect: Greater than -12.00dB.

Glaucoma produces several changes in the visual field, including a widespread, nondescript loss of retinal sensitivity. This diffuse loss of retinal sensitivity should be considered highly diagnostic of glaucoma when it is asymmetric and correlates with asymmetric changes in intraocular pressure or optic disc appearance.

In most cases, the loss of sensitivity occurs in characteristic patterns and locations (i.e., nasal step, arcuate scotoma, paracentral scotoma) that often correlate with changes in the optic nerve or retinal nerve fiber layer or both.

Remember, a threshold visual field examination can be normal in a patient with early glaucoma. To continue the diagnostic program, many optometrists now employ electrodiagnostic vision testing to assist in their medical decision-making.



Retinal nerve fiber layer fallout with the left eye worse than the right. Glaucomatous optic atrophy is not measurable without an OCT scan.

Visual Evoked Potential Tests

Recent research shows that in some patients, glaucoma-induced vision loss can be detected with visual evoked potential (VEP) testing before measurable visual field defects are detected.[3]

Evoked potential studies are recorded electrical responses to stimulation of a sensory organ. The VEP is an objective electric sign of visual pathway function and is based on the general principles of neural electrophysiology. A VEP test is classified as a sensory nerve conduction study; it measures the speed and strength of the evoked responses along the visual pathway.

Because its parameters are sensitive to abnormalities in the visual pathway, VEP testing can be used to evaluate the integrity of the afferent visual sensory system. Abnormal waveform peak latencies and wave shape amplitude help to identify pathologies in many locations ranging from the eye to the primary visual cortex.[4]

Modern VEP technologies are designed to measure retinal ganglion cell functional losses and, in particular, losses to the magnocellular pathway, which conveys low luminous contrast information. Detecting loss of low contrast function is thought to be of value in diagnosing early glaucomatous damage.[5] Although not diagnostic of glaucoma in itself, abnormal VEP test results, in addition to other abnormal clinical findings, can assist in the often-difficult diagnosis of early glaucoma.[5]

I usually order a VEP test when performing a glaucoma evaluation. The first benefit associated with VEP testing occurs when the OCT test results don't match the visual field examination test results. In these cases, you are left with IOP or other clinical measures to make a decision. You can be guided one way or the other by using the VEP testing results. For example, if your OCT scan shows characteristic fallout of retinal nerve fiber layer but your visual field is normal, do you treat the patient, or do you follow up for signs of progression? Do you continue the diagnostic process and perform more functional testing and get more information? What if the OCT scan and the VEP were abnormal? Would that make a difference? Would you initiate treatment sooner? Would you follow more aggressively? Is the opposite true if the VEP test results are normal?

Diagnostic Sensitivity for Glaucoma

- Cup-to-disc ratio = 56%
- Intraocular pressure = 65%
- Nerve fiber layer analysis = 67%
- Ganglion cell complex analysis = 68%
- RAPDx pupillary testing = 81%
- Combination of nerve fiber layer, ganglion cell complex, and cup-to-disc ratio = 86%

Huang JY, Pekmezci M, Mesiwala N, et al. Diagnostic power of optic disc morphology, peripapillary retinal nerve fiber layer thickness, and macular inner retinal thickness in glaucoma diagnosis with fourier-domain optical coherence tomography. Journal of Glaucoma. 2011;20(2):87-94.

What doctors need to see is that incorporating VEP test results can help answer these questions and can make a difference in how you treat the patient.

Note, all functional testing in patients with glaucoma can be variable; also, there are many patients with glaucoma who have normal VEP test results. Knowing that, you may consider performing electroretinography in addition to VEP testing to help detect early glaucoma.

VEP-Based Decision Making

Occasionally, we'll see patients who are initially identified as "glaucoma suspects" because of elevated intraocular pressures. These are the easiest patients to identify as glaucoma suspects, but we still must determine whether they actually have glaucoma. When structural testing and visual fields are inconclusive, visual evoked potential testing can help guide treatment decisions.

For example, a 63-year-old black woman was examined three times over the course of one month with intraocular pressure measurements in the mid-twenties at each visit. Her risk factors for glaucoma were race, age, elevated intraocular pressures and large optic cups.

Although her OCT testing confirmed the large optic cups, the

neuroretinal rim was healthy in each eye and the retinal nerve fiber layer thickness was normal and symmetric. Threshold visual fields showed scattered paracentral scotomas, but there were no definite glaucomatous visual field defects. At this point, my structural evaluation told me that the eye was normal. My first functional vision test, the threshold visual field exam, said there might be a problem, but if so, it was mild. In this patient, I needed another functional test to provide more information. My first two diagnostic tests did not correlate. When that happens in my office, we usually go to the next functional vision test, and the next test is usually the VEP.

In this case, the patient's VEP was normal. That information, combined with the normal OCT scan, helped me to decide to not initiate treatment for glaucoma and to diagnosis the patient as a high-risk glaucoma suspect that will be re-evaluated at six-month intervals.

Electroretinography

Electroretinography testing evaluates the integrity of the retina. To best detect glaucoma-induced vision loss, a specialized pattern electroretinogram (pERG) protocol uses a contrast-reversing pattern for the stimulus to produce information about ganglion cell function.

A recent study suggests pERG testing detects glaucoma-induced functional abnormalities while retinal ganglion cells are still alive, several years before OCT testing reveals clinically significant fallout of the retinal nerve fiber layer or threshold perimetry detects a visual field defect.[6]

The pERG is used in a similar manner as the VEP test—it is an adjunct test of visual function. It is no better or worse than any other functional test and it is just as important as any other test. It is not standard of care; it is cutting-edge care. Because its clinical value is highest early in the natural history of glaucoma, pERG testing may have particular benefit in an evaluation of patients suspicious for developing glaucoma.

Pupillary Light Reflex Testing

The pupillary light reflex controls the diameter of the pupil in response to the intensity of light that falls on the retina. Normal pupils have an equal response to light stimulus. Light entering the eye produces pupillary constriction (the direct response) as well as constriction in the pupil of the unstimulated eye (the indirect response).

It is an objective sign of visual pathway function, and it can be used as a clinical test for the detection and quantification of abnormalities in the retina, optic nerve, optic chiasm, optic tract and the pretectal area of the midbrain. Testing pupillary reactivity involves comparing the velocity and amplitude of the pupillary responses.

In the pupillary light reflex pathway, the neural elements are separated into afferent and efferent conduction pathways. The goal of pupillary light reflex testing is to determine if there is a defect in either pathway.

Asymmetry in the pupillary light response is a relative afferent pupillary defect (RAPD). The manual testing for the detection of RAPD is performed by alternately illuminating each eye while comparing the velocity and amplitude of the pupillary responses. If asymmetry is detected, neutral density filters in 0.3 logarithmic steps aid in quantification of RAPD. The size of the RAPD can be quantified by the density of the filter required to balance the response of each eye.[7]

Because early glaucomatous damage to the visual system is often asymmetric, patients with early glaucoma may demonstrate an RAPD on pupillary light reflex testing. Unfortunately, manual detection of a subtle RAPD can be challenging. For more accurate and objective results, many doctors prefer using computer-assisted automated pupillometry.

Pupillary Light Reflex Pathway

Afferent Neural Pathway

- Ganglion cells connect to the pretectal nucleus of the upper midbrain, bypassing the lateral geniculate nucleus.

Efferent Neural Pathway

- Axons from the Edinger-Westphal nucleus run to both the right and left oculomotor nerves to innervate the constrictor muscle of the iris.

Color Vision Examination

Loss of chromatic discrimination (i.e., dyschromatopsia) has been reported in patients with glaucoma for more than 100 years.[8] Modern research shows color vision defects may precede visual field defects in many patients with glaucoma. Although 20% to 40% of patients with glaucoma have normal color vision, most do not.[8] Prevalence estimates for the different types of color vision defects found in the dyschromatopsia of glaucoma are:[8]

- 30% to 50% blue-yellow defects.
- 20% to 30% unspecified loss of chromatic discrimination.
- 5% red-green defects.

Testing color vision is an important step in detecting the earliest signs of glaucoma. However, color vision abnormalities are not specific and can occur in a wide variety of ophthalmologic and neurologic conditions. Diseases that result in a loss of foveal function, optic nerve disease, retinal dystrophies, neurologic disease, neurologic injury and visual field defects can be signaled by color

vision abnormalities as well.

Like most acquired color vision defects, the loss of chromatic discrimination found in the dyschromatopsia of glaucoma is primarily blue-yellow in nature. The predominance of these tritan-like defects has several possible explanations:

• Blue-yellow cones and their neuronal connections are more fragile than red-green cones and are less able to resist the effects of elevated intraocular pressure.

• Blue-yellow cones are more susceptible to the effects of elevated IOP and are selectively damaged because they have larger receptive fields than red-green cones and have a unique morphology and connectivity to second order neurons.

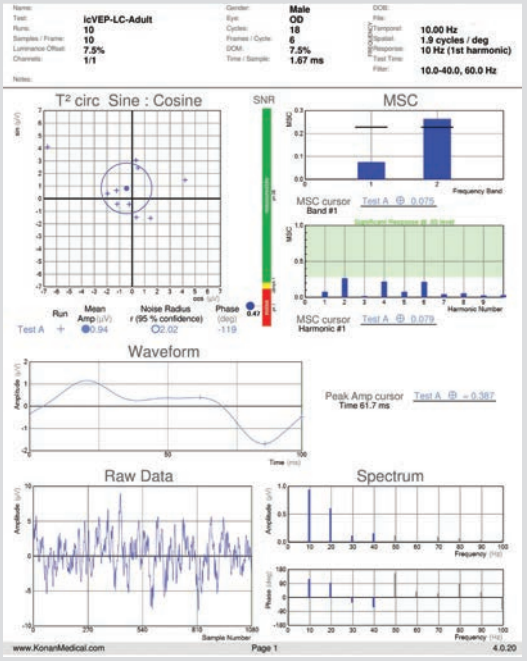
• Blue-yellow cones are fewer in number and there is little overlap between adjacent receptive fields. Because of this, the loss of a small number of blue-yellow ganglion cells may produce a preferential impairment of blue-yellow discrimination compared with red-green, even if the proportion of damaged axons is the same for both the magnocellular pathway (red-green channel) and the koniocellular pathway (blue-yellow).

In addition to a loss of chromatic discrimination, recent investigations have shown that color contrast thresholds are elevated in patients with glaucoma.[9] However, just like the other tests of visual function, there are no rules about glaucoma-induced color vision defects. Some patients with glaucoma never develop color vision defects, while others develop the dyschromatopsia of glaucoma only in the advanced stages of the disease.[9]

There are several ways to measure a person's vision, and glaucoma can produce abnormalities in all of them. Although threshold visual field testing is the standard method of evaluating vision loss, new functional testing has proven that, in many patients, glaucoma-induced vison loss can be detected with other technologies before visual field loss can be detected on an automated perimeter. Earlier detection of glaucoma is possible if we used all the available diagnostic tests. It's up to us.

Our primary responsibility as optometrists is to protect, preserve and enhance our patient's vision. Because undiagnosed glaucoma remains a common cause of preventable blindness, we still have lots of work to do. Fortunately, new science and technology is forging a new frontier in early glaucoma detection.

The science demonstrates that we can't rely on a visual field examination alone, and the technology has begun to follow that lead. This new class of instruments provides the clinical measurements of visual function necessary to diagnose early glaucoma and initiate treatment before permanent vision loss occurs. ■



Isolated-Check VEP

• Preferentially tests the "on" and "off" subdivisions of the magnocellular neural pathway.

• Test strategy present 10 two-second runs.

• Runs are compared for consistency, outliers are repeated, and the mean of the runs is presented.

• This very short test time may be better for children and the elderly.

• EvokeDx features an infrared gaze tracker to improve the reliability of the results.

• The organic LED removes any unwanted luminance artifact in the screen displays.

Dr. Thomas practices in Dallas, Texas, is the owner of BMG Consulting Group and the founder of DecisionmakerPLUS.com.

1. Tielsch J, Sommer A, Katz J, et al. Baltimore Eye Survey. JAMA. 1991 Jul;266(3):369-74.
2. Malik R, Swanson W, Garway-Heath D. The 'structure-function' relationship in glaucoma—past thinking and current concepts. Clin Exp Ophthalmol. 2012 May-Jun:40(4):369-80.
3. Punjab O, Lin S, Stamper R. Advances in mapping the glaucomatous visual field: from confrontation to multifocal visual evoked potentials. The Internet Journal of Ophthalmology and Visual Science. 2006;4(1).
4. Miller N, Newman N, Biousse V, Kerrison J. Examination of the vision sensory system. In: Walsh and Hoyt's Clinical Neuro-Ophthalmology: The Essentials. 2nd ed. Philadelphia: Lippincott Williams & Wilkins; 2008:39-41.
5. Zemon V, Tsai JC, Forbes M, et al. Novel electrophysiological instrument for rapid and objective assessment of magnocellular deficits associated with glaucoma. DocOphthalmol. 2008 Nov;117(3):223-43.
6. Banitt M, Ventura L, Feuer W, et al. Progressive loss of retinal ganglion cell function precedes structural loss by several years in glaucoma suspects. Invest Ophthalmol Vis Sci. 2013 March;54(3):2346-52.
7. Younis A, Eggenberger E. Correlation of relative afferent pupillary defect and retinal nerve fiber layer loss in unilateral or asymmetric demyelinating optic neuropathy. Invest Ophthalmol Vis Sci. 2010 Aug;51:4013-6.
8. Pacheo-Cutilla M, Edgar D. Acquired colour vision defects in glaucoma. Br J Ophthalmol. 1999;83:1396-1402.
9. Niwa Y, Muraki S, Naito F, Minamikawa T. Evaluation of acquired color vision deficiency in glaucoma using the Rabin Cone Contrast Test. Invest Ophthalmol Vis Sci. 2014 Oct;55:6686-90.

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You can obtain transcript-quality continuing education credit through the Optometric Study Center. Complete the test form *(page 69)*, and return it with the $35 fee to: Optometric CE, P.O. Box 488, Canal Street Station, New York, NY 10013. To be eligible, please return the card within one year of publication.

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Please check with your state licensing board to see if this approval counts toward your CE requirement for relicensure.

1. Which of the following is true about functional testing in patients suspected of having glaucoma?
a. Glaucoma-induced functional abnormalities exhibit a wide variability between individual patients.
b. Glaucoma-induced functional abnormalities exhibit a wide variability between repeated measurements.
c. Accurate measurements of glaucoma-induced vision loss are difficult to ascertain.
d. All of the above.

2. In a comprehensive assessment of patients suspected of developing glaucoma, structural investigations should focus on which of these clinical measurements?
a. Visual acuity.
b. Intraocular pressure.
c. Retinal nerve fiber layer thickness.
d. All of the above.

3. Glaucoma is considered a neurodegenerative disease based on which test?
a. Visual field testing.
b. Retinal imaging.
c. Electrodiagnostic testing.
d. Pupillary light reflex testing.

4. The structure-function relationship in glaucoma is based on which of the following?
a. The relationship between optic disc cupping and changes in the visual field.
b. At least 25% to 35% of retinal ganglion cells must be lost before the first visual field defect appears.
c. A linear relationship between structural measures of glaucomatous optic atrophy and visual field measurements.
d. All of the above.

5. Which of the following is true of most patients with advanced glaucoma?
a. There is a functional reserve period where the optic nerve gets worse, but the visual field does not.
b. Functional vision loss changes at a greater rate than structural damage.
c. Most patients demonstrate a functional latency period late in the natural history of the disease.
d. All of the above.

6. Which is not a component of the 'ganglion cell dysfunction' concept?
a. In early glaucoma, ganglion cells may become dysfunctional before they die.
b. At least 25% to 35% of retinal ganglion cells must be lost before the first visual field defect appears.
c. Ganglion cell dysfunction produces a reduction in visual field sensitivity that does not correlate with OCT measurements or ganglion cell complex.
d. Patients can develop measurable visual field defects before they develop clinically measurable structural damage.

7. Automated perimeters plot the luminescence value of the patient in nonspecific units of measurement called what?
a. Lumens.
b. Foot-candles.
c. Decibels.
d. Lux.

8. Which of the following is not a common glaucomatous visual field defect?
a. Nasal step.
b. Arcuate scotoma.
c. Hemianopia.
d. Paracentral scotoma.

9. Glaucomatous visual field defects often correlate with changes in which of these?
a. Intraocular pressure.
b. Optic nerve.
c. Visual acuity.
d. All of the above.

10. Which is true about threshold VF examinations in glaucoma patients?
a. Glaucoma produces a widespread, non-descript loss of retinal sensitivity.
b. A threshold visual field examination can be normal in a patient with early glaucoma.
c. Glaucomatous visual field defects usually occur in characteristic patterns and locations.
d. All of the above.

11. Which is not true about VEP testing in patients suspected of having glaucoma?
a. VEP testing is based on the general principles of neural electrophysiology.
b. VEP testing evaluates the integrity of the afferent visual sensory system.
c. Detection of high contrast function is thought to be of value in diagnosing early glaucoma damage.
d. VEP abnormalities may precede visual field defects in many patients with glaucoma.

12. Which of the following neural conduction channels in the visual pathway conveys low luminous contrast information to the visual cortex?
a. Parvocellular pathway.
b. Magnocellular pathway.
c. Koniocellular pathway.
d. Pupillary light reflex pathway.

13. Which is true about pERG testing?
a. It measures the strength and speed of the neural response along the visual pathway.
b. Detection of loss of low contrast function is thought to be of value in diagnosing early glaucomatous damage.
c. Glaucoma-induced vision loss can be detected with pERG testing before measurable visual field defects are detected.
d. All of the above.

14. Pattern electroretinography testing has high clinical value because:
a. It is easier to perform than VEP testing.
b. It is more important than VEP testing.
c. Early in the natural history of glaucoma, testing may detect functional abnormalities while retinal ganglion cells are still alive.
d. Abnormal findings are diagnostic of glaucoma.

15. Which anatomic area is not evaluated with pupillary light reflex testing?
a. Retina.
b. Optic nerve.
c. Visual cortex.
d. Optic tract.

16. Which is not true about the pupillary light reflex?
a. Testing pupillary reactivity involves com-

OSC QUIZ

paring the velocity and amplitude of the pupillary responses.
b. It is a subjective sign of visual pathway function.
c. Asymmetry in the pupillary light response is a relative afferent pupillary defect.
d. The neural elements that transmit information are separated into afferent and efferent conduction pathways.

17. What percentage of patients with glaucoma have normal color vision?
a. 70% to 90%.
b. 60% to 80%.
c. 40% to 60%.
d. 20% to 40%.

18. Which of the following is not true about the dyschromatopsia of glaucoma?
a. Color vision defects may precede VF defects in many patients with glaucoma.
b. Most glaucoma-induced color vision defects are blue-yellow in nature.
c. Color contrast thresholds are elevated in patients with glaucoma.
d. All glaucoma patients eventually develop some loss of chromatic discrimination.

19. Which of the following neural conduction channels in the visual pathway transmits red-green chromatic information to the visual cortex?
a. Magnocellular pathway.
b. Parvocellular pathway.
c. Koniocellular pathway.
d. None of the above.

20. Which of the following could explain the predominance of blue-yellow defects in acquired color vision deficiencies?
a. Blue-yellow cones and their neuronal connections are more fragile than red-green cones.
b. Blue-yellow cones are selectively damaged because they have larger receptive fields than red-green cones.
c. Blue-yellow cones are fewer in number and there is little overlap between adjacent receptive fields.
d. All of the above.

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12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
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18.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
19.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
20.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

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'I Think I'm Getting AUDITED!'

Understanding the process and avoiding the red flags are the first steps to making it through an audit like a winner. **By John Rumpakis, OD, MBA, Clinical Coding Editor**

No one wants to be audited. Still, health care audits are an integral part of the quality control cycle for all health care stakeholders—federal and state governments, third party insurers, health care providers and patients. Health care fraud drains the economic resources of all participants and also costs us in terms of patient health. Yet, very little fraud, waste and abuse is properly identified and adjudicated. This "invisible tax" on our health care system is costly to all. In an environment of increasing scrutiny on everything that touches a patient encounter, technology and big data are playing an increasing role in identifying outliers and irregular patterns of care.

The Recovery Audit Program

The mission of the federal Recovery Audit Program (RAC) is to correct improper Medicare payments through the detection and collection of overpayments made on claims of health care services and the identification of underpayments to providers.[1] The program also allows the Centers for Medicare and Medicaid Services (CMS) to implement actions that will prevent future improper payments.[1]

There are four recovery audit contractors, each responsible for identifying overpayments and underpayments in approximately a quarter of the country. The RAC auditors work and are paid on a contingency basis, so they have significant financial incentive to recover improper payments.

Additionally, in 2013 the Office of Inspector General (OIG) mandated increased collaboration between the RAC auditors, CMS and program integrity contractors to ensure that the RAC auditors refer all instances of suspected fraud to both the OIG and CMS. Reports of improper payments are accessible on the CMS website, under the Comprehensive Error Rate Testing (CERT) heading.[2] In 2013, RAC auditors identified and recovered $3.75 billion in improper payments.

So we know that audits provide a great return on investment for the federal government. But other third-party payers are also getting into the audit business as well. This is not just for medical carriers either; managed vision care plans, such as VSP, are also aggressively auditing their providers. So, what do practitioners have to do to survive an audit? Because, as the maxim goes, "it is not a question of if, but of when."

Fraud vs. Waste & Abuse

The most important step in avoiding a negative audit outcome is understanding what these organizations are looking for. CMS has

Corrections by Recovery Auditors in 2013[3]

	Overpayments Collected		Underpayments Restored		Total Corrected Payments	
Recovery Auditor	Number of Claims	Amount Collected	Number of Claims	Amount Restored	Number of Claims	Amount Corrected
Performant	365,435	$762,312,114	3,823	$14,708,223	369,258	$777,020,336
CGI	132,787	$528,731,497	2,416	$7,781,593	135,203	$536,513,091
Connolly	537,690	$1,219,049,512	23,203	$48,358,754	560,893	$1,267,408,266
HDI	453,622	$1,140,666,285	13,167	$31,521,627	466,789	$1,172,187,913
Unknown	104	$155,217	2	$38,307	106	$193,524
Total	**1,489,638**	**$3,650,914,625**	**42,611**	**$102,408,504**	**1,532,249**	**$3,753,323,129**

consolidated all of its fraud and abuse information into its Medicare Learning Network (MLN).[4] These key resources can help practitioners identify "red flags" within their practice that would be subject to scrutiny under the US Federal False Claims Act (FCA), Anti-Kickback Statute, Physician Self-Referral Law (Stark Law), Social Security Act and US Criminal Code.

According to CMS, abuse describes practices that, either directly or indirectly, result in unnecessary costs to the Medicare program.[5] Abuse includes any practice that does not provide patients with services that are medically necessary, does not meet professionally recognized standards and is not priced fairly. Examples include billing for services that were not medically necessary; charging excessively for services or supplies; and misusing codes on a claim, such as upcoding or unbundling codes.[5]

In contrast to abuse, CMS defines fraud as knowingly submitting false statements or making misrepresentations of fact to obtain payment; soliciting, paying and/or accepting remuneration to induce or reward referrals; making prohibited referrals; billing for services not furnished, supplies not provided, or both; or billing for services at a level of complexity higher than the service actually provided.[5]

Penalties for these violations are not insignificant. Let's look at the False Claims Act as an example.

False Claims Act

The FCA protects the government from being overcharged or sold substandard goods or services. It imposes civil liability on any person who knowingly submits, or causes the submission of, a false or fraudulent claim to the federal government. The "knowing" standard includes acting in deliberate ignorance or reckless disregard of the truth related to the claim. For

What Exactly is 'Medical Necessity'?

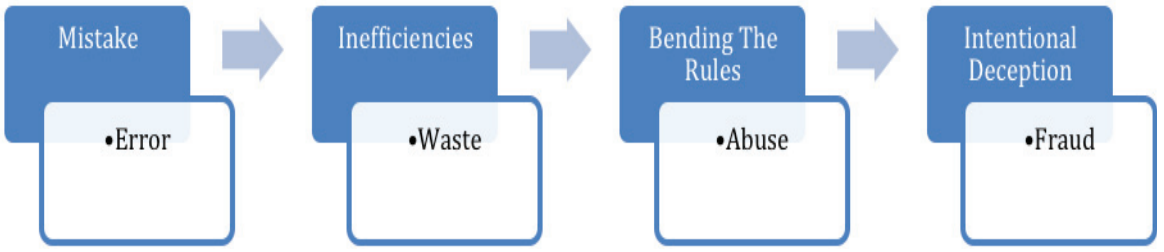
According to the American Medical Association, medical necessity is defined as "services or procedures that a prudent physician would provide to a patient in order to prevent, diagnose or treat an illness, injury or disease or the associated symptoms in a manner that is:

- In accordance with the generally accepted standard of medical practice.
- Clinically appropriate in terms of frequency, type, extent, site and duration.
- Not intended for the economic benefit of the health plan or purchaser or the convenience of the patient, physician or other health care provider."[1]

CMS defines medical necessity as the need for an item(s) or service(s) to be reasonable and necessary for the diagnosis or treatment of disease, injury or defect. The need for the item or service must be clearly documented in the patient's medical record. Medically necessary services or items must be:

- Appropriate for the symptoms and diagnosis or treatment of the patient's condition, illness, disease or injury.
- Provided for the diagnosis or the direct care of the patient's condition, illness, disease or injury.
- In accordance with current standards of good medical practice.
- Not primarily for the convenience of the patient or provider.
- The most appropriate supply or level of service that can be safely provided to the patient."[2,3]

1. American Medical Association. Statement of the American Medical Association to the Institute of Medicine's Committee on Determination of Essential Health Benefits. January 14, 2011. www.iom.edu/~/media/8D03963CAEB24450947C1AEC0CAECD85.ashx.
2. Riva Lee Asbell. Medical necessity: can you please define that? Part I. www.rivaleeasbell.com/articles/mednec1.pdf.
3. OptiCare Managed Vision. Medical necessity: can you please define that? – Part I. www.opticare.com/2014/01/10/medical-necessity-can-please-define-part/.



The progression from an innocent mistake to intentional deception. Penalties for these violations are not insignificant.

example, a physician who submits claims to Medicare for a higher level of medical services than he actually provided or that the medical record documents.

The civil penalties for violating the FCA can include a fine of $5,500 to $11,000 per false claim and up to three times the amount of damages sustained by the government as a result of the false claim. Individuals or entities that submit false claims could also face criminal penalties.[5]

Often, the main focus of an audit is proper billing for rendered services. Rendered services adhere to a fairly strict yet straightforward standard: medically necessary services. This means we must document in the record why a particular service or procedure is medically necessary and provides a benefit or aids in the patient's outcome.

Failure to meet the requirement of medical necessity is often the most cited omission during an audit process. Overtesting to protect oneself from medical liability is an often-used defense by practitioners. Available technology is also often used as prognostic testing rather than its required diagnostic value when a physician is suspicious of a specific disease or problem. This is best summed by CMS' position on worthless services.[6] While not specifically defined in the False Claims Act, worthless services are generally services that are:

- Not accepted as safe and effective by the medical community.
- **Not supported in peer-reviewed medical literature.**
- Experimental or investigational.
- **Not medically necessary in a specific case or medical diagnosis.**
- **Furnished at a level, duration, dosage or frequency not appropriate for a specific patient or clinical condition.**
- Not furnished in a manner consistent with standards of care.
- Not furnished in a setting consistent with the patient's medical needs and condition.
- **Furnished in a manner for patient or provider convenience.**
- **A device not FDA approved.**
- An obsolete test or service.[6]

Optometrists have the greatest exposure in the statements in bold.

So, how do you avoid performing worthless services? Stick to the established clinical guidelines for eye care provided by the American Academy of Ophthalmology's Preferred Practice Patterns and by the American Optometric Association's Clinical Practice Guidelines.[7,8]

Legal Liability

Relationships with patients are increasingly dominated by the contractual relationship of a third-party payer. Contractual agreements generally stipulate rules for accurate coding and billing practices, documentation, appropriate prescription authority and assignment within that particular health care system.

In clinical practice, of course, we don't send our actual records to a third party each time we want to get reimbursed. Instead, we represent the service or procedure with a five-character code: a CPT code, a Level II HCPCS code or a Level III HCPCS code. We never submit our clinical findings or medical judgment of a patient's condition; rather, we submit an ICD-9 code on a claim form—either electronically through your EMR or by paper on a CMS-1500 form or its derivation. Your signature on the form signifies that you have reviewed all information and it is true and accurate in all aspects.

If you have never read the back of your CMS-1500 form, you should. It reminds you of your legal obligations and the possibility of civil penalties if information is missing or misrepresented.

HIPAA requires us to follow the rules of both CPT and ICD—and we have to follow all of the rules, not just the ones that are convenient for us. That, in turn, means that you must know the rules before you submit any code representing the clinical care you performed and the diagnoses you

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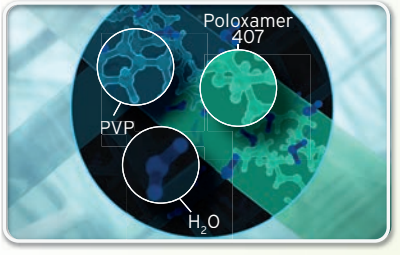

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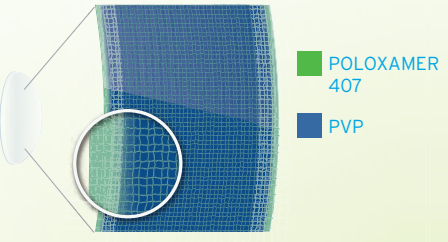

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REFERENCE: 1. Multiple-Packaged Lenses Comparison, Tyler's Quarterly – Professional Edition, September 2013 **2**. Twenty-two subjects participated in a randomized, double masked, contralateral eye study to evaluate water loss of Biotrue ONEday, 1-Day Acuvue Moist, 1-Day Acuvue TruEye contact lenses. After 4,8,12, and 16 hours of wear, lenses were removed and immediately weighed (wet weight). The lenses were then completely dried and reweighed (dry wet). The percent water loss was then calculated for each lens from the wet and dry weights.

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CMS Sharpens its Stance Against Fraud and Abuse[9]

Past	Present
• Providers suspected of fraudulent activity were put on prepay review, sometimes indefinitely • CMS initiated overpayment recovery • Law enforcement determined if any arrest is appropriate	• Denies individual claims • Its contractors use prepay review as an investigative technique • Revokes providers' licenses for improper practices • Collaborates with law enforcement before, during and after case development • Addresses the root cause of identified vulnerabilities

attributed to the patient encounter. The clinical care you provide and the codes you use to represent that care are inseparable. They are the only legal representation of the services you provided and must be accurately represented within the medical record that leaves the boundaries of your office.

Fraud Detection

There is no question that technology used to capture the patient encounter is making record keeping more accurate—and time consuming. The emergence of meaningful use also means that the EMR must be consistent with specific federal standards. You must spend more time making sure that your record is truthful, accurate and complete before signing it and translating it into CPT and ICD codes for billing purposes.

With the increasing specificity of ICD-10, you'll need to spend an even greater amount of time ensuring that the chief complaint, clinical findings, diagnosis(es) and clinical plan all match appropriately with the coding represented on the claim.

The RED Flags

So how can you avoid an adverse result? These red flags might trigger the attention of a third-party payer:

- Using codes under OIG review
- Not reviewing your claims against recovery audit issues
- Abusing codes
- Aberrant or inconsistent billing patterns
- Maximizing revenue without sufficient documentation
- Cloning documentation
- Not understanding definitions of modifiers and inappropriate use of modifiers

The primary item a carrier can use against you in an audit is your medical record, which is also the primary item that you can use in your defense—and the only thing that is 100% in your control. So, the adverse outcome of an audit is completely preventable by putting good controls, self-auditing procedures and compliance measures into effect within your practice.

'I Think I'm Getting Audited'

The very first thing to do if you receive a letter from a carrier is to determine what you have received. There are different types of correspondence that are easily confused. A *heralding notice* alerts all providers that the payer intends to conduct audits systemwide. It does not necessarily mean that you are getting audited. A *notice of audit*, however, is an official notification that you are getting audited. If you are getting audited, consider these questions:

1. Has the carrier identified key issues of concern?
2. Is it for recovery or fraud?
3. Is it an educational or network-wide audit?
4. Is the payer asking for specific records?
5. Is it targeting specific diagnoses?
6. How many records is the payer asking for? Higher numbers may indicate a more compre-

Types of Audits

- **Pre-payment Audit:** These are generally automated, and you may never even know about it. If the payer requests documentation, they are looking at a specific issue.
- **Post-payment Audit:** After the claim is paid, the payer requests specific information to support the coding and claim.
- **Automated Review Audit:** This is a computer-generated review to identify violations in standard rules or edits. The review is usually associated with a very clear policy.
- **Comprehensive Review Audit:** This is a review, performed by a certified reviewer, of the entire medical record. The payer may apply standard criteria (such as CMS standards) to determine medical necessity requirements or to validate that the service was provided.
- **Fraud and Abuse Audit:** This is an audit conducted when there is suspicion of an intentional violation. If the Special Investigations Unit (SIU) is conducting the audit, it is because there is a very high degree of suspicion of intentional fraudulent behavior and the potential penalties can be much more significant.
- **Claim Recovery (Administrative Review) Audit:** An audit that is focused on violation of coding rules, where intentional fraud is not suspected.
- **Claim Focused Audit:** The payer is looking at specific types of claims or services, but is not necessarily focusing on your particular practice.
- **Provider-focused Audit:** This is focused specifically on your practice or a specific provider within your practice with concern surrounding specific coding and billing behaviors.

hensive review, with the expectation of a higher recovery.

7. Is the payer suspecting improper coding or inconsistent billing processes?
8. Is the payer questioning medical necessity of procedures or relationships with specific diagnoses and CPT codes?

Building Your Defense

If you are being audited, there are some important steps to take when building your defense. The first is creating a team of experienced individuals to assist you. That will most likely include an attorney who can help you understand your rights and requirements contained within your provider agreement/contract. It should also consist of a peer who specializes in audit defense; has a good understanding of CPT coding definitions, rules, regulations and requirements; and is familiar with all of the zip code-specific local coverage determinations and national coverage decisions that were in place on the dates of the service called into question. This is often a stressful time in your career, and it's good to have objective advice from professionals who regularly deal with this. Other tips to keep in mind:

- Find out who is conducting the audit. Learning the department can provide you with insight on the level of seriousness.
- Pay attention to all date-specific deadlines. The general limit to pull records is 45 days but can vary based upon your contract and your state's laws.
- Assemble the correct information to send. Don't fail an audit by neglecting to submit the requested information.
- Assign someone in the office as the primary contact for the carrier (someone familiar with your medical records).
- Send copies, not the originals. If you can't find a record in question, request more time.
- Never send less than what the carrier is requesting.
- If an audit leads to a request for recoupment of claims payment, ask for time to review the demand letter.
- Determine if you received the demand letter within the proper time period following the audit.
- Make sure the auditors provided proper rationale and justification, as well as how they determined the recovery amount.
- Check if the payer provided an explanation for each claim incorrectly paid or coded.
- Ensure the payer explained the statistical sampling and extrapolation.
- Be sure the payer provides information on your rights of appeal.

Getting audited does not have to be a frightening experience. While it is a serious issue and should be treated accordingly, you can prevent negative outcomes by practicing in accordance with local standards of care, keeping detailed and accurate medical records, and staying up-to-date with your legal requirements.

The health care environment is only going to get more demanding in the areas of compliance and proper medical coding. And the process of "checks and balances"—what we call audits—is only going to get more complex and arduous. Avoid fear of audits by using good preventive measures in how you provide, record and report your care for payment by a third party. ■

Guidelines for Good Practice

- Take care of the patient first and foremost. You have a fiduciary responsibility to the patient to put their medical outcome first.
- Take care of the medical record second. Make sure to record what you are doing and why you are doing it. Make an effort to record your thoughts and impressions about the patient's conditions and your care plan.
- Code your encounter from only what you have written in the record. Never assume you have provided a certain examination service level. Learn the definitions and specifications of the special ophthalmic procedures that you order.
- Learn how to use modifiers correctly. Their specific purpose is describing the episode of care and how it differed from normal.
- If you get audited, don't go it alone. Build your team with individuals who can properly assist you in audit defense. Use an OD-knowledgeable firm that specializes in audit defense and an attorney who can help you understand your rights and responsibilities under your provider contract.

1. Centers for Medicare and Medicaid Services. Recovery Audit program. Comprehensive Error Rate Testing (CERT). www.cms.gov/Research-Statistics-Data-and-Systems/Monitoring-Programs/Medicare-FFS-Compliance-Programs/Recovery-Audit-Program/.
2. Centers for Medicare and Medicaid Services. www.cms.gov/Research-Statistics-Data-and-Systems/Monitoring-Programs/Medicare-FFS-Compliance-Programs/CERT/CERT-Reports.html.
3. Centers for Medicare and Medicaid Services. Recovery auditing in Medicare for fiscal year 2013. www.cms.gov/Research-Statistics-Data-and-Systems/Monitoring-Programs/Medicare-FFS-Compliance-Programs/Recovery-Audit-Program/Downloads/FY-2013-Report-To-Congress.pdf.
4. Centers for Medicare and Medicaid Services. Medicare learning network (MLN) fraud & abuse products. www.cms.gov/Outreach-and-Education/Medicare-Learning-Network-MLN/MLNProducts/Downloads/Fraud-Abuse-Products.pdf.
5. Centers for Medicare and Medicaid Services. Medicare fraud & abuse: prevention, detection, and reporting. www.cms.gov/Outreach-and-Education/Medicare-Learning-Network-MLN/MLNProducts/downloads/Fraud_and_Abuse.pdf.
6. Morrow D, Richman H, Wartman R. Coverage indications, limitations and medical necessity. AOA Focus. 2014 Sept:51.
7. American Academy of Ophthalmology. Preferred practice pattern guidelines. www.aao.org/about-preferred-practice-patterns.
8. American Optometric Association. AOA optometric clinical practice guidelines. www.aoa.org/optometrists/tools-and-resources/clinical-care-publications/clinical-practice-guidelines?sso=y.
9. Centers for Medicare and Medicaid Services. Module: 10. Medicare and Medicaid fraud prevention. www.cms.gov/Outreach-and-Education/Training/CMSNationalTrainingProgram/Downloads/2013-Fraud-and-Abuse-Prevention-Workbook.pdf.

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Is Your Medical Model Working?

Billing and reimbursements are critical, but training, equipment and referrals are also key. **By Jane Cole, Contributing Editor**

For Milton Hom, OD, of Azusa, Calif., the idea of adopting the medical model in eye care is similar to HBO's sitcom *Silicon Valley*, which follows six entrepreneurs who found a start-up company. "To ensure survival, they learn that successful companies 'pivot'… take what you have and change directions," Dr. Hom says. "When I was in optometry school, the medical model was unheard of; now, it's the present and future of optometry."

As scope of practice laws have progressed in the majority of states, many optometrists have "pivoted" their practices from focusing entirely on routine eye exams to treating medical conditions such as dry eye, glaucoma and AMD, with the dual benefit of additional revenue and patient retention.

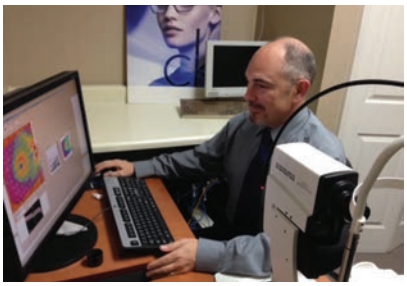
But if you've recently transitioned to a medical model or have been at it for several years, how can you ensure you are getting the most out of it? Here are seven questions you can ask yourself—and the answers shared by successful ODs who've been able to turn their medical practices into a success.

Dr. Sutton runs a VEP with a glaucoma patient.

1. Is My Staff Properly Trained?

Staff should always be educated on correct medical billing and medical insurance when you adopt a medical model, says Bryan Rogoff, OD, of Baltimore, Md. Education should be ongoing with updates provided during monthly meetings, he adds, considering insurance companies' policies are always changing. He also stresses the importance of staff education and its impact on patient satisfaction and customer service. "If staff doesn't understand medical coverage and what your practice can bill [for] and [they bill for something that's] not covered, the bill comes back to the patient. Obviously, this is inconvenient and the patient isn't happy."

TeShawna B. Sutton, OD, of 20/20 Eyecare in London, Ky., trains her staff to look back at pre-



Dr. Diecidue reviews an OCT of the retina for indications of diabetic retinopathy.

vious exams to determine if a glaucoma suspect, for example, has had the proper tests run and to alert the doctor if anything is missing. "That's why training staff on disease processes is important. They can be a huge resource to help keep us on track," she says.

Staff training on equipment is also critical, as reliable and reproducable test results depend on properly executed tests, she adds. For example, staff needs to understand how important it is for a patient to maintain proper fixation during a visual field, she says.

"We have trained our staff on what a hemoglobin A1C means and why it's important that we ask. They also know how important communication with the primary care physician is for a diabetes patient, so staff routinely asks for the doctor's name and contact information. These small steps are integral to our efficiency and ultimate success," Dr. Sutton says.

2. Am I Getting Reimbursed in a Timely Fashion?

Thirty is the magic number when it comes to reimbursement. You should aim to receive reimbursements in 30 days or less, Dr. Rogoff says. This often comes down to making sure there are no errors when you bill the insurance company, which can result in denied claims if you're billing for something that isn't covered. Timely reimbursement is integral to business success, as it ensures you can pay your bills and payroll without complications, Dr. Rogoff adds.

"It's great if you get paid a certain amount for glaucoma procedures, but if you don't get reimbursed for 60 days and you have bills to pay within 30 days, this can be a problem. In the medical model, cash flow is critical."

3. Am I Billing Correctly?

Staff needs to be adept at knowing how to bill for medical visits; otherwise, your boat will sink, says Anthony Diecidue, OD, of Stroudsburg, Pa. Medical visits are almost exclusively covered by medical insurance, so if you and your staff aren't trained in medical billing, it could put a severe financial strain on your practice.

Documentation is the key to staying organized and ensuring you are billing correctly, Dr. Sutton adds. Documenting everything will help you prove medical necessity—which all insurance companies demand—as well as keep track of what tests are accepted by certain carriers. Each insurer will be a little different, so "make sure you know your limits on testing with the different carriers," Dr. Sutton says. For example, different insurers have different rules about what tests can be done on the same day or when a test needs a modifier or is a bilateral procedure, so understanding the specifics of each code you are using is key.

"We tend to know what we want and what we need inside our heads, but there are rules for every test and limits for the number of times a test can be performed." Dr. Sutton says. At Dr. Sutton's practice, staff uses a coding verification website on a daily basis to make sure proper diagnosis codes are linked to proper procedure codes, she says.

You also should be careful about submitting documents that include red flags because of your billing patterns, Dr. Rogoff says. "Sometimes when you jump into a medical model of eye care, you may think you need to get reimbursed for everything, but it can snowball, so be sure you are billing for what's medically necessary," he adds. He cautions against overbilling, performing procedures that are not covered or administering specific tests to every patient without cause—all of which could be red flags and put your practice under the insurer's microscope.

4. Is My Practice Patient-Centric?

Jack Schaeffer, OD, of Birmingham, Ala., describes the medical model, which he prefers to call the integrated health care model, as 100% patient-centric and a comprehensive approach to care.

"The definition of an integrated health care model is working within the entire patient's medical

The Medical Model: Where to Start?

If you're thinking about adopting a medical model but aren't sure what conditions to treat, Dr. Hom suggests starting with allergy. "Prescribing an antihistamine drop and monitoring its effect is an easy first step," he says.

Another area in which to get your feet wet in the medical model is diabetic retinopathy fundus exams, Dr. Hom says. "Most primary care physicians require them, so the need is already built in."

In-office treatment of *Demodex*, although generally not covered by insurance, is another potential area to explore, he says.

Beware of Structural Impediments

By Pamela Joyce Miller, OD

Everyone talks about the benefits of the medical model and billing under a patient's medical insurance, but there are some basic problems that are encountered in many offices. Here are a number of issues to be prepared for when adopting the medical model:

1. Staff should ask every patient if there is any medical issue involved. For example, is the patient a diabetic, requiring specialized testing under their medical plan or an additional optometric office visit?
2. Staff should ask all patients about their medical coverage and if they must be referred to their primary care physician. If so, patients need to know that, regardless of licensure, the optometrist may not be on the medical panel, or that the charges will not be covered for their medical visit.
3. All patient copays should be collected up front, with the understanding of what the patient is or is not covered for. This is a major issue, because with so many different insurances, plans and items that may not be covered, it can be nearly impossible to remain current. Patients must be made aware that some charges may not be covered and they are responsible for out-of-pocket fees. Signed authorizations are a must, indicating the procedure, the amount, consent to treatment and the commitment to pay for services.
4. Many optometrists are simply not covered under a patient's medical insurance. Panels are frequently closed, not open to optometrists or they have several optometrist employees who work in-house, and the panel will not authorize or pay for services rendered by the non-employee or non-panel optometrist, even in an emergency.
5. While payment for medical services may be available, the reimbursement rate may be very low or the amount may not be known until after the billing has been either paid or rejected by the insurance company.
6. Although the medical model in optometry involves working with other professions and disciplines, the fact remains that it is often a one-way street. The optometrist who refers a patient out of office for care and sends a report to a fellow professional or requests a report of the consultation or tests run may receive nothing in return. Often, there is a lack of continuity of care and inter-professional communication.
7. In some jurisdictions, the medical model is virtually non-existent, due primarily to the insurance companies, physician-run clinics and ACAs. Until this changes, optometry will continue to use the medical model in theory, but fall short in practice in some areas of the country. This serves as a major detriment in caring for the patient, maintaining health care costs and providing comprehensive care within the medical arena.

The most significant structural impediments preventing optometry from doing better at transitioning to primary eye care revolve around the issues of access and parity. Until they are resolved, the medical model cannot be fully implemented.

community of doctors and ancillary personnel to ensure a long, healthy life for the patient. So that's working with their primary care doctor, endocrinologists, gastroenterologist and rheumatologists, depending on each patient's condition or wellness factor," he says.

Even if patients are coming in for a routine eye exam, the office should be prepared if the patient needs more comprehensive care.

"It's important the staff understands, from the time that patient enters your office, they are entering into an integrated health care model. The patient may look healthy and only need glasses or contacts," Dr. Schaeffer says, but oftentimes there may be more to the story. For example, pediatric patients who are developing myopia will need preventative care and patients with diabetes may need more testing during their routine exam—not to mention communication with their endocrinologist.

The concept of a patient-centric practice also means talking about protective eyewear for children who play sports, discussing blue light-blocking lenses with patients who use electronics on a daily basis, offering ocular vitamins and performing a complete evaluation for dry eye and other ocular surface diseases in patients who are at risk before a contact lens evaluation, Dr. Schaeffer adds.

5. Am I Getting Referrals and More Calls for Medical Visits?

Within months of launching a medical model, optometrists should expect to receive more calls for medical-related visits—and doctors can generate some of these additional visits themselves, Dr. Diecidue says. "If you see a patient for a regular eye exam and you notice they have a hemorrhage in their retina and they're diabetic, this person needs a specific evaluation." Take note of medical conditions, such as these, that warrant further evaluation, Dr. Diecidue says. You can then educate your patient on the medical services you can provide and schedule follow-up testing.

Likewise, if a patient has risk factors for medical conditions such as glaucoma or retinal detachment that could have heredity components, you can suggest other members of the family be examined, Dr. Diecidue says.

Now that you have a medical model, it is important to form relationships with other specialists in

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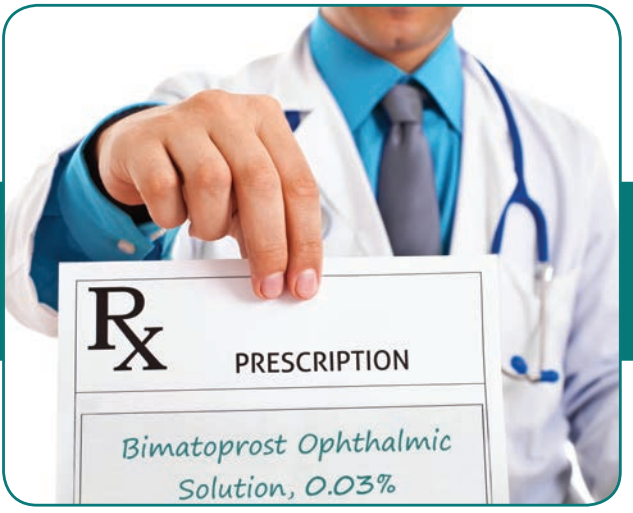

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IMPORTANT SAFETY INFORMATION

Bimatoprost ophthalmic solution 0.03% has been reported to cause darkening (pigmentation) of eye color, eyelid skin, and eyelashes as well as increased growth of eyelashes. Pigmentation changes can increase as long as bimatoprost ophthalmic solution 0.03% is used. After stopping bimatoprost ophthalmic solution 0.03%, darkening of eye color is likely to be permanent, while darkening of the eyelid skin and eyelash changes may be reversible. When only one eye is treated, there is a possibility of eyelash changes in the eye treated with bimatoprost ophthalmic solution 0.03%. These changes may result in differences between the eyes in eyelash length, thickness, darkness, number of eyelashes, and/or direction of eyelash growth. These changes are usually reversible upon stopping bimatoprost ophthalmic solution 0.03% therapy. Macular edema (swelling of the macula), including cystoid macular edema, has been reported during treatment with bimatoprost ophthalmic solution. Bimatoprost ophthalmic solution 0.03% should be used with caution in patients without a natural lens, in patients with a torn posterior lens capsule who have an artificial lens implant, or in patients with known risk factors for macular edema. Patients should avoid allowing 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. In the event patients 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 bimatoprost ophthalmic solution, 0.03%. Contact lenses should be removed prior to instillation of bimatoprost ophthalmic solution, 0.03% and may be reinserted 15 minutes following its administration. The most common side effects are conjunctival hyperemia, growth of eyelashes, and ocular pruritus.

LUPIN PHARMACEUTICALS, INC.

[1] SAVINGS An Economic Analysis of Generic Drug Usage in the U.S., GPhA, September 2011, page 1.

BIMA062315TRG

BRIEF SUMMARY OF FULL PRESCRIBING INFORMATION

BIMATOPROST Ophthalmic Solution, 0.03%

INDICATIONS AND USAGE

Bimatoprost ophthalmic solution, 0.03% is indicated for the reduction of elevated intraocular pressure in patients with open angle glaucoma or ocular hypertension.

In clinical studies of patients with open angle glaucoma or ocular hypertension with a mean baseline IOP of 26 mmHg, the IOP-lowering effect of bimatoprost ophthalmic solution, 0.03% once daily (in the evening) was 7 to 8 mmHg.

WARNINGS AND PRECAUTIONS

Pigmentation

Bimatoprost ophthalmic solution has been reported to cause changes to pigmented tissues. The most frequently reported changes have been increased pigmentation of the iris, periorbital tissue (eyelid) and eyelashes. Pigmentation is expected to increase as long as bimatoprost is administered. The pigmentation change is due to increased melanin content in the melanocytes rather than to an increase in the number of melanocytes. After discontinuation of bimatoprost, 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. 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 nevi nor freckles of the iris appear to be affected by treatment. While treatment with bimatoprost ophthalmic solution, 0.03% can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly.

Eyelash Changes

Bimatoprost ophthalmic solution, 0.03% 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

Prostaglandin analogs, including bimatoprost, have been reported to cause intraocular inflammation. In addition, because these products may exacerbate inflammation, caution should be used in patients with active intraocular inflammation (e.g., uveitis).

Macular Edema

Macular edema, including cystoid macular edema, has been reported during treatment with bimatoprost ophthalmic solution. Bimatoprost ophthalmic solution 0.03% should be used with caution in aphakic patients, in pseudophakic patients with a torn posterior lens capsule, or in patients with known risk factors for macular edema.

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 bimatoprost ophthalmic solution, 0.03% and may be reinserted 15 minutes following its administration.

ADVERSE REACTIONS

In clinical trials, the most frequent events associated with the use of bimatoprost ophthalmic solution, 0.03% occurring in approximately 15% to 45% of patients, in descending order of incidence, included conjunctival hyperemia, growth of eyelashes, and ocular pruritus. Approximately 3% of patients discontinued therapy due to conjunctival hyperemia.

Ocular adverse events occurring in approximately 3 to 10% of patients, in descending order of incidence, included ocular dryness, visual disturbance, ocular burning, foreign body sensation, eye pain, pigmentation of the periocular skin, blepharitis, cataract, superficial punctate keratitis, periorbital erythema, ocular irritation, and eyelash darkening. The following ocular adverse events reported in approximately 1 to 3% of patients, in descending order of incidence, included: eye discharge, tearing, photophobia, allergic conjunctivitis, asthenopia, increases in iris pigmentation, and conjunctival edema. In less than 1% of patients, intraocular inflammation was reported as iritis.

Systemic adverse events reported in approximately 10% of patients were infections (primarily colds and upper respiratory tract infections). The following systemic adverse events reported in approximately 1 to 5% of patients, in descending order of incidence, included headaches, abnormal liver function tests, asthenia and hirsutism.

USE IN SPECIFIC POPULATIONS

Pregnancy Category C

Nursing Mothers

It is not known whether bimatoprost is excreted in human milk, although in animal studies, bimatoprost has been shown to be excreted in breast milk. Because many drugs are excreted in human milk, caution should be exercised when bimatoprost 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 chronic use.

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May 2015

your area that may refer patients to your practice, Dr. Diecidue adds. In his community, Dr. Diecidue forged professional relationships with pediatricians, emergency room nurses, internists and other primary care physicians who now often refer to him if they see a patient with an eye-related medical condition. Dr. Diecidue also approached non-medical members of his eye care community who don't do advanced testing who now routinely refer to him.

6. Do I Have the Necessary Equipment and Physical Space?

If you are just getting set up to adopt the medical model, make sure your practice can handle the added equipment and caseload, Dr. Schaeffer says. While a non-medical practice can flourish with a dispensary and one exam room, the medical model demands a lot more space—and technology. "It's important your physical plant shows you are there to take care of the patient, whether that means medical, glasses or contacts, or just an eye exam. You need equipment necessary to do the work," Dr. Schaeffer says.

For example, in glaucoma treatment, an optometrist would need a perimeter, pachymeter, OCT, retinal camera and gonioscope. "Those are minimal requirements to truly follow a glaucoma patient in your practice, and you'd report those findings to the patient's primary care physician," Dr. Schaeffer says.

Just because you take on more medical services doesn't mean you need to buy every piece of equipment available. "If you don't have an OCT, that's fine, as long as you have access to one and your glaucoma patient has an OCT test done on regular intervals of six months to a year," Dr. Schaeffer says.

Dr. Sutton reviews a retinal image with a diabetes patient.

7. Are My Books and Schedule Balanced?

When you adopt the medical model, expect to spend more time in the exam room. "With a routine eye exam, you can almost clock how much time you'll spend with a patient. You need to adjust your schedule to accommodate patients there for medical visits, especially ones with multiple medical issues. You don't want to rush through these exams," Dr. Diecidue says.

A lot will change in your practice with the adoption of the medical model, including the need for more technicians and longer patient appointments—both of which are going to cost you, Dr. Schaeffer says. "Integrated health care is a low-volume, high-cost model," he explains. "It can't be high-volume, low-cost or even high-volume, medium-cost.

"Some patients will select out, so be prepared to lose some patients," he adds.

Dr. Rogoff suggests adding hours to your practice's schedule if you already have a full book and strong productivity. "If you have 60% to 70% activity in your books because you've added a medical model, you need to extend your book," he says.

Once you start a medical model, be sure to watch revenues on both sides of your practice. The medical revenue will undoubtedly increase, but also keep an eye on how it affects the optical and contact lens side to find the right balance, Dr. Rogoff says.

"Optometry students today come out of school well prepared to institute a medical model immediately," says Dr. Sutton. "With refractive plans becoming more and more popular and ophthalmology being overly busy, we stand to really make our practices profit from using what most of us are already doing anyway. You can become an integral part of your patients' primary care team, and I think that's a goal we should all set for ourselves." ■

Scars: Not All Doom and Gloom

Does an *Acanthamoeba* infection mean the end for a successful LASIK procedure?

Edited by Joseph P. Shovlin, OD

Q Are there any frank contraindications to LASIK following a contact lens-related protozoan infection? The patient has a very small anterior stroma mid-corneal scar. The diagnosis was made early two years ago and the patient responded well to topical agents.

A Two distinct factors should be considered in a case such as this, says Christopher J. Rapuano, MD, a corneal specialist at Wills Eye Hospital in Philadelphia. First, optometrists must decide whether the eye is a good candidate for LASIK following a parasitic corneal infection and, second, determine whether LASIK is the best option for patients with corneal scars in the area of the flap.

Corneal infections resulting from *Acanthamoeba* can be difficult to treat. The protozoa's life cycle comprises two distinct stages: an infective trophozoite and a dormant cyst that forms when the active organism is exposed to adverse conditions. *Acanthamoeba* cysts are highly durable and in some cases may survive through initial treatment and subsequently reactivate, perpetuating the infection and necessitating further aggressive therapy.

Dr. Rapuano recommends patients with significant corneal scarring from *Acanthamoeba* who wish to undergo corneal transplantation to improve vision be taken off all medications for a minimum of three months—and ideally, six to 12 months—before surgery. "I would wait for an eye to be off all *Acanthamoeba* treatment for at least one year before considering any refractive surgery," he says. "I would also warn patients that the infection could recur, with or without refractive surgery." Confocal microscopy can be performed to try and determine whether any presence of the protozoa remains.

Photo: Elyse L. Chaglasian, OD, and Gregg Eric Russell, OD

Corneal scars, like the one pictured here, can impede the success of LASIK.

Onset of corneal scarring typically involves some loss of stromal tissue, which is often filled in by epithelial cells. These cells are not nearly as strong as the corneal stroma itself, and this weakness increases risk of a LASIK flap buttonhole, which can result from an abnormal lamellar incision made with either a microkeratome or a femtosecond laser. If this happens, says Dr. Rapuano, the flap should be replaced immediately and allowed to heal. Excimer laser treatment should not be performed, as this can lead to an irregular corneal surface and possibly loss of correctable visual acuity from a contour mismatch between the flap and the stromal bed.

James Aquavella, MD, a professor in the department of ophthalmology at the University of Rochester, cautions that LASIK should not be performed if the scar is central, as visual acuity would remain affected following the procedure. LASIK is also contraindicated for patients with thin corneas (i.e., less than 500µm) to avoid problems associated with secondary ectasia. Dr. Aquavella recommends a minimum residual stromal bed of 300µm should remain following the incision.

In patients like these, PRK is a better option, says Dr. Rapuano. The procedure, which removes layers of the epithelium to expose the underlying cornea, may decrease corneal scar depth or even remove the scar entirely, and obviates the flap complications characteristic of LASIK. Healing time and risk of infection following PRK, however, are slightly increased in comparison to LASIK. ■

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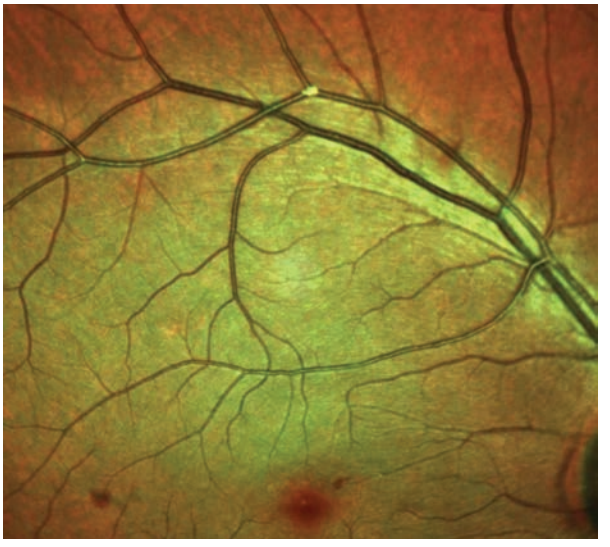
Reference: 1. Patel S, Henderson R, Bradley L, Galloway B, Hunter L. Effect of visual display unit use on blink rate and tear stability. *Optom Vis Sci.* 1991;68(11):888-892.

Act FAST With Stroke

Stroke is the fifth leading cause of death in the United States. Blur, visual field loss and diplopia may be presenting signs. **By Carlo J. Pelino, OD, and Joseph J. Pizzimenti, OD**

Each year, nearly 800,000 people in the United States suffer a stroke, and one American dies from a stroke every four minutes, on average.[1] A stroke is an acute loss of neurological function due to an abnormal perfusion of brain tissue. A stroke occurs when the blood supply to part of the brain is interrupted or severely reduced, depriving brain tissue of oxygen and nutrients. Within minutes of the blood flow interruption, brain cells begin to die. Also called cerebrovascular accident (CVA), stroke is a medical emergency.



Retinal emboli increase the risk of stroke-related death.

Symptoms of stroke may include sudden loss of speech, dizziness, confusion, weakness or paralysis of one side of the body, headache and seizure. Common ocular symptoms in stroke patients may include blur, visual field loss and diplopia. Prompt treatment is crucial, as early action can minimize brain damage and potential complications.

Stroke patients are at significantly high risk for another event. One of every four survivors has another stroke within five years.[1,2]

Types of Stroke

There are three categories of stroke:

Ischemic Stroke. Most strokes (85%) are ischemic and commonly result from an arterial obstruction by a thrombus or embolus.[2]

Hemorrhagic Stroke. A hemorrhagic stroke occurs when a blood vessel in the brain leaks or ruptures. The hemorrhage applies pressure to brain cells and causes damage. These leaks or ruptures occur either within the primary brain tissue or in the subarachnoid space.

Transient Ischemic Attack (TIA). A TIA is also known as a "mini-stroke." It is different from the ischemic and hemorrhagic types in that blood flow to the brain is blocked for only a short time—usually no more than five minutes.

As a warning sign of an impending ischemic or hemorrhagic cerebrovascular event, TIA is considered a medical emergency. More than a third of those who have a TIA end up having a major stroke within one year if they don't receive treatment, and 10% to 15% will have a major stroke within three months of the TIA.[1,2] Recognizing and promptly treating TIAs can reduce the risk of a major stroke and severe disability.

Ocular Signs and Symptoms

As many as 75% of those who sustain a stroke also experience one or more TIAs in the preceding days to months, and some of these initial events involve visual symptoms and signs.[2] Symptoms are similar, though more pronounced, in ischemia and hemorrhagic stroke. A patient experiencing a TIA may have monocular vision loss—known as *amaurosis fugax*, Greek for "fleeting blindness"—that occurs rapidly and resolves in a few minutes.

Retinal emboli also may be a premonitory sign of cerebral stroke.[3] Cholesterol emboli originate from plaques in the carotid artery or occasionally the aortic arch. Calcific emboli usually arise from the heart or the great vessels. Platelet-fibrin emboli can occur from the heart or blood vessels. Retinal emboli are associated with an increased incidence of vascular disease and stroke-related death.[3]

Bilateral transient monocular vision loss (TMVL) may result from vertebrobasilar insufficiency to the brain's posterior aspect. Other possible ocular manifestations of

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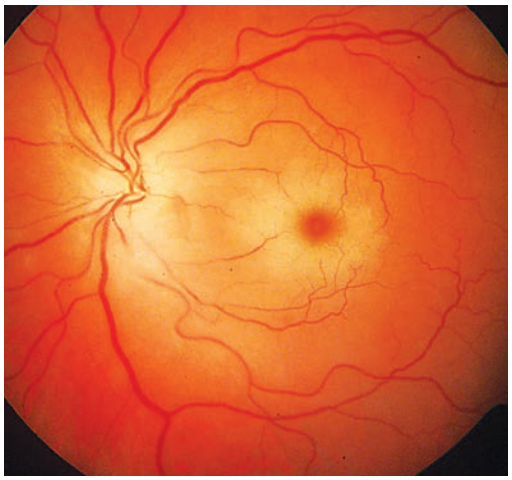


A central retinal arterial occlusion (CRAO) may result from an embolus or atherosclerotic changes. Similarly, ischemic stroke often results from an arterial obstruction by a thrombus or embolus.

stroke include diplopia (cranial nerve involvement) and homonymous field loss. Stroke patients are more likely to develop ocular ischemic syndrome, retinal arterial occlusions and anterior ischemic optic neuropathy.[3]

FAST Intervention

Acting FAST can help stroke patients get the urgent treatment they desperately need. The most effective treatments are only available if the stroke is diagnosed within three hours of the first symptoms. Stroke patients may not be eligible for the most effective treatments if they don't arrive at the hospital in time.

If you think someone may be having a stroke, perform the following simple tests:[3-5]

Face: Ask the person to smile. Does one side of the face droop?

Arms: Ask the person to raise both arms. Does one arm drift downward?

Speech: Ask the person to repeat a simple phrase. Is his or her speech slurred or strange?

Time: If you observe any of these signs, call 9-1-1 immediately. Note the time symptoms first appear. Call an ambulance so that medical personnel can begin life-saving treatment on the way to the emergency room.

Treatment

If a patient gets to the hospital within three hours of the first symptoms of an ischemic stroke, the health care team may administer a thrombolytic (or clot-busting) drug. Tissue plasminogen activator (tPA) is an example of such an agent that improves the chance of recovery. Several studies show that ischemic stroke patients who received tPA are more likely to fully recover or have less disability than those who do not receive the drug.[6,7] In addition, patients treated with tPA are less likely to need ongoing care in a long-term health facility.[6,7]

In hemorrhagic stroke, basic life support, as well as controlling bleeding, seizures, blood pressure (BP) and intracranial pressure (ICP), are critical. Medications, surgery or endovascular procedures may be necessary to stop the bleeding and save brain tissue.

Medications. Classes of medications used in the treatment of acute hemorrhagic stroke include anticonvulsants (to prevent seizure recurrence), antihypertensive agents (to reduce BP and other risk factors of heart disease) and osmotic diuretics (to decrease ICP in the subarachnoid space).[8,9]

Endovascular procedures. These procedures are less invasive and less risky for the patient than surgical treatments. A long tube is inserted through a major artery in the leg or arm and then guided to the site of the weak spot or break in a blood vessel. The tube is then used to install a device to repair the damage or prevent bleeding.[8,9]

Surgery. Hemorrhagic strokes may be treated surgically. If the bleeding is caused by a ruptured aneurysm, a metal clip may be put in place to stop blood loss.[8,9]

Risk Factors for Stroke[1,4,5,8]

- Hypertension
- Diabetes
- Atrial fibrillation
- Smoking
- Hyperlipidemia
- Carotid stenosis
- Lack of physical activity
- Age >55
- Male gender
- Black race
- Family history of stroke
- Personal history of stroke
- Sickle cell disease

Optometrists encounter patients at risk for stroke every day. We must acquaint ourselves with the first symptoms and signs, including ocular complications. Our next column will address optometry's role in post-stroke care. ■

1. Centers for Disease Control and Prevention. Stroke Facts. Accessed June 5, 2015. www.cdc.gov/stroke/facts.htm.
2. Mozzafarian D, Benjamin EJ, Go AS, et al. Heart disease and stroke statistics—2015 update: a report from the American Heart Association. Circulation. 2015:e29–322.
3. Purvin V. Cerebrovascular disease and the visual system. Ophthalmol Clin N Am. 2004;17:329-55.
4. Lambert M. Practice Guidelines: AHA/ASA guidelines on prevention of recurrent stroke. Am Fam Physician. 2011;83(8):993–1001.
5. National Institute of Neurological Disorders and Stroke. Brain Basics: Preventing Stroke. Accessed December 4, 2013.
6. National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. N Engl J Med. 1995;333:1581–7.
7. Marler JR, Tilley BC, Lu M, et al. Early stroke treatment associated with better stroke outcome: the NINDS rt-PA stroke study. Neurology. 2000;55:1649–55.
8. National Institute of Neurological Disorders and Stroke. Stroke: Challenges, Progress, and Promise. Bethesda, MD: National Institutes of Health; 2009.
9. American Heart Association/American Stroke Association. Stroke Treatments. 2013.

The Rare Case of Uneven Steven

Using fundus and SD-OCT images, what can you say about this patient's optic nerve?

By Mark T. Dunbar, OD, and Sherrol Reynolds, OD

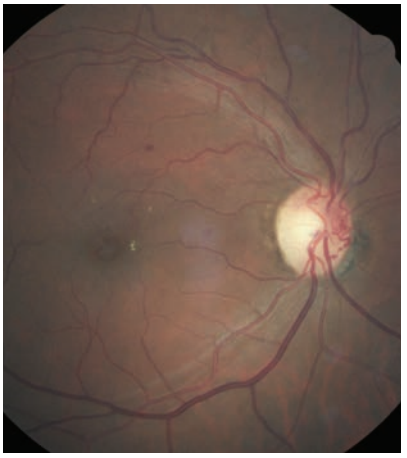
A 62-year-old black male presented for a comprehensive eye examination with compliant of blurry vision at distance for a year. Medical history included Type 2 diabetes mellitus for five years, hypertension and hypercholesterolemia, for which he was on oral medications. His reported glycosylated hemoglobin (HbA1c) was 8.0.

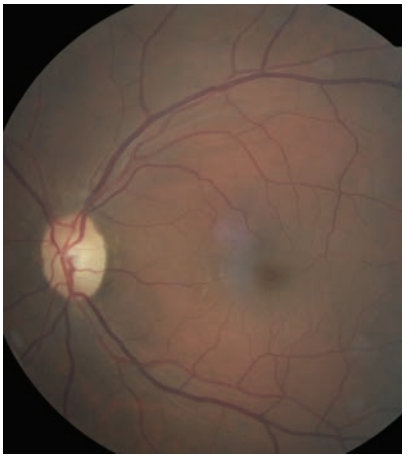
Ocular examination revealed best-corrected visual acuities of 20/30 OD and 20/25 OS. Pupils, ocular motilities and confrontation visual field were unremarkable. Mild nuclear sclerotic cataracts were observed on slit-lamp evaluation in both eyes. Of note was an area of vascular abnormality on his right optic disc. Mild retinal hemorrhages, arteriovenous nicking and macular changes were noted in his right eye (*Figures 1 and 3*). Dilated examination of his left eye revealed a healthy optic disc and arteriovenous nicking (*Figure 2*). SD-OCT was performed as well (*Figure 4*).

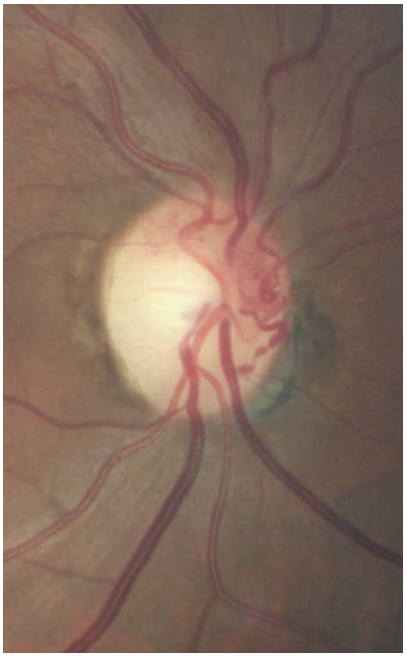
Take the Quiz

1. What do the changes seen on the optic nerve photo represent?
   a. Optic disc hemorrhage.
   b. Neovascularization.
   c. Collateral retinochoroidal vessels.
   d. Optic disc capillary hemangioma.

2. What does the SD-OCT finding of the right eye reveal?
   a. Clinically significant macular edema (CSME).
   b. Vitreomacular traction (VMT).
   c. Non-center involved diabetic macular edma.
   d. Epiretinal membrane.







Figs. 1 and 2. Above, can you diagnose this 62-year-old black male who presented with blurry vision at distance using these fundus photos of the right and left eyes?
Fig. 3. At left, look carefully at the optic nerve of the right eye.

3. Based on the clinical findings, what is the correct diagnosis?
   a. Resolving central retinal vein occlusion (CRVO).
   b. Asymmetric diabetic retinopathy.
   c. Ocular ischemic syndrome.
   d. VMT.

4. What is the likely etiology?
   a. Carotid artery stenosis.
   b. Hypertension.
   c. Sickle cell disease.
   d. Idiopathic.

5. How should the ocular findings be managed?
   a. Intravitreal anti-VEGF injection.
   b. Topical steroid/NSAID.
   c. Pars plana vitrectomy.

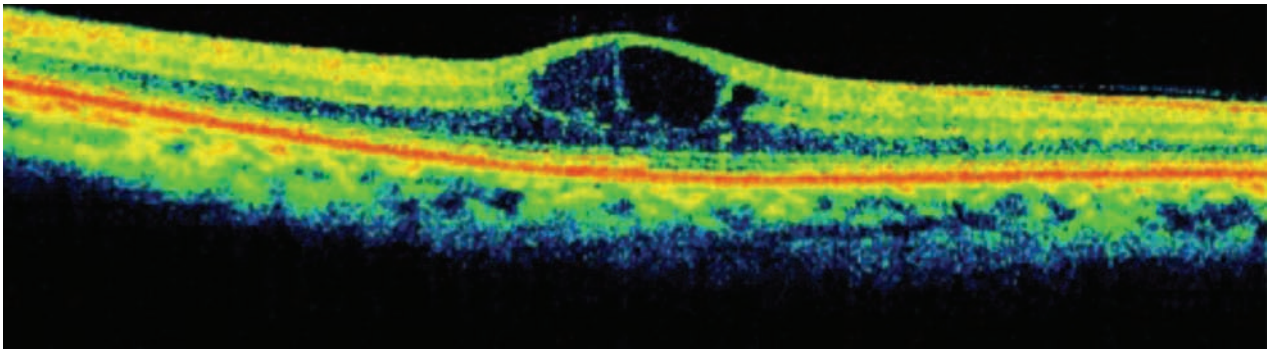


Fig. 4. SD-OCT images of the right and left macula—what does the OCT reveal?

d. Intravitreal ocriplasmin injection.

For answers, see page 106.

Discussion

The changes involving the optic nerve of the right eye represent neovascularization. On close inspection, fine wispy vessels are visible from 1:00 to 3:00 on the disc. xIn addition, there were scattered blot hemorrhages, and the SD-OCT revealed cystoid macular edema. This appears to be early proliferative diabetic retinopathy (PDR). We expected to see similar changes in the left eye, but upon close inspection, the left eye was essentially normal with the exception of mild A/V nicking. Our patient appears to have asymmetric diabetic retinopathy with PDR and CSME in the right eye and no diabetic retinopathy in the left eye. So, what is going on?

Diagnoses

Asymmetric DR is defined as proliferative diabetic retinopathy in one eye and nonproliferative diabetic retinopathy or no retinopathy in the fellow eye.[1,2] It is a rare disease, occurring in 5% to 10% of patients with diabetes.[3] Several factors have been linked to asymmetric DR, including vitreous loss from cataract surgery, trauma, uveitis, optic atrophy, branch retinal vein occlusion, PVD, chorioretinal atrophy, amblyopia and high myopia.[1-3] A predominant factor inducing asymmetric DR is carotid artery disease, which has been found to occur in four of 20 patients with asymmetric PDR.[1]

The disease presents with no mild retinopathy on the same "ipsilateral" side as the more stenotic carotid artery, which is thought to be "protective" against the development of PDR. Although not clearly understood, researchers speculate that the protective effect results from a reduction of retinal arterial perfusion pressure.[4] Asymmetric DR should also raise suspicion of venous stasis retinopathy or ocular ischemic syndrome (OIS), which is characterized by uveitis, iris neovascularization and mid-peripheral retinal hemorrhages and microaneurysms. OIS develops on the ipsilateral side as the severe stenosis and is associated with a five-year mortality of 40%.[5]

Additional Testing

Given these concerns, we listened for a bruit by performing carotid artery auscultation on our patient and were surprised to hear one on the left side. We then referred our patient for carotid ultrasound and Doppler testing, which revealed 50% stenosis of the right carotid and 65% stenosis of the left carotid. Due to lack of symptoms, such as transient ischemic attack (TIAs) or dizziness, carotid artery endarterectomy was deferred and the patient was placed on aspirin (Ecotrin) 81mg daily therapy. His retinopathy was treated with anti-VEGF therapy and the cystoid macular edema resolved.

This case underscores the importance of recognizing this condition and its association with severe carotid artery disease. Prompt medical testing and intervention is essential with this condition to prevent not only vision loss, but also life-threatening complications or early death. ■

This case was written and provided by Sherrol Reynolds, OD, associate professor of optometry, Nova Southeastern University College of Optometry.

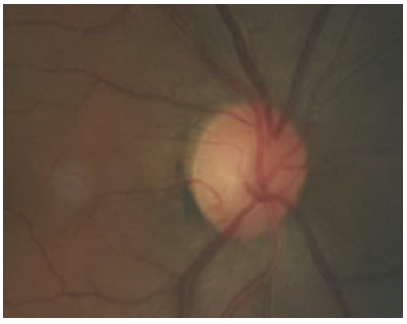
1. Duker J, Brown G, Bosley T, et al. Asymmetric proliferative diabetic retinopathy and carotid artery disease. Ophthalmology. 1990;97:869–74.
2. Valone J, McMeel J, Franks E. Unilateral proliferative diabetic retinopathy. Initial findings. Arch Ophthalmol. 1981;99:1357–61.
3. Valone J, McMeel J, Franks E. Unilateral proliferative diabetic retinopathy. II. Clinical course. Arch Ophthalmol. 1981;99:1362-6.
4. Brown G, Magargal L, Simeone F, et al. Arterial obstruction and ocular neovascularization. Ophthalmology. 1982;89:139-44.
5. Sivalingam A, Brown G, Magargal L, Menduke H. The ocular ischemic syndrome. II. Mortality and systemic morbidity. International Ophthalmology. 1989 May;13(3):15-20.


The EAGLE Has (Not Yet) Landed

Optometry may be on the brink of discovering new treatments for chronic angle closure glaucoma. **By Joseph W. Sowka, OD and Alan G. Kabat, OD**

A 58-year-old asymptomatic man was referred for a glaucoma consultation. He was seen several weeks earlier when his intraocular pressure (IOP) was measured at 36mm Hg OD and 20mm Hg OS. His uncorrected visual acuity was 20/20 OD and 20/400 OS; a large macular toxoplasmotic scar accounted for the vision reduction in that eye. His biomicroscopic examination revealed healthy anterior segment structures in each eye. A crystalline lens evaluation showed minimal nuclear sclerosis in each eye. His anterior chamber was shallow, but there was no iris bombé in either eye. Gonioscopic evaluation revealed only anterior trabecular meshwork, or Schwalbe's line, throughout his anterior chamber angles in each eye. Gonioscopic indentation was successful in opening his angles to pigmented trabecular meshwork at most, save for several areas of peripheral anterior synechiae (PAS). Optic disc evaluation showed bilateral superior and inferior neuroretinal rim damage, with threshold perimetry demonstrating corresponding visual field defects.

The patient was diagnosed with bilateral primary chronic angle closure glaucoma (PCACG). The therapeutic goals were to change the anatomic status of his closed angles and lower his IOP. Angle closure glaucoma in any form is far less common than open angle glaucoma, making management sometimes confusing. Additionally, new theories are challenging the way we address this condition. This month, we will review the management of patients with chronic angle closure glaucoma.



This fundus image shows a patient with primary chronic angle closure glaucoma.

PCACG Patients

Patients with PCACG are typically older and asymptomatic, and women are more commonly affected than men.[1] Biomicroscopically, there will be a shallow anterior chamber, though typically deeper than in primary acute angle closure glaucoma, where iris bombé is characteristically seen. The anterior chamber angle may be appositionally closed and opened upon manual pressure using a four-mirror gonio lens, or the angle may be closed with broad areas of PAS. The superior and temporal quadrants of the anterior angle may be the earliest sites of the synechial angle closure, with gradual extension on the nasal quadrant, until the angle closes at the inferior quadrant.[2] While in most cases there is asymmetric closure first involving the superior angle, there can be an even, circumferential process that slowly progresses to symmetrical closure. This is called "creeping angle closure" and appears as an angle that becomes progressively more shallow over time.[3]

Symptoms

Anatomical features act in concert to cause shallowing of the anterior chamber. As a patient ages, thickening of the crystalline lens leads to a relative pupil block, putting the iris into apposition with the trabecular meshwork. Because the closure is slow, symptoms you would typically see with acute angle closure, such as pain, nausea and vision loss, are absent and patients remain unaware of the elevating IOP.[4,5]

Treatments

Conventional thinking has long held that all cases of primary angle closure resulting from relative pupil block need to undergo laser peripheral iridotomy (LPI) as soon as possible after diagnosis. This allows aqueous flow from the posterior chamber to the anterior chamber to bypass any pupil blockage. This can stimulate the backward relaxation of the iris, with a resultant deepening of the chamber and opening of the angle.[6,7]

However, a significant number of these PCACG eyes will manifest residual angle closure even after LPI.[6] Additionally, there often will be elevated IOP due to damage to the trabecular meshwork

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from appositional and synechial closure.[8,9] Most eyes with PCACG require further treatment to control IOP, including trabeculectomy and medical therapy.[10]

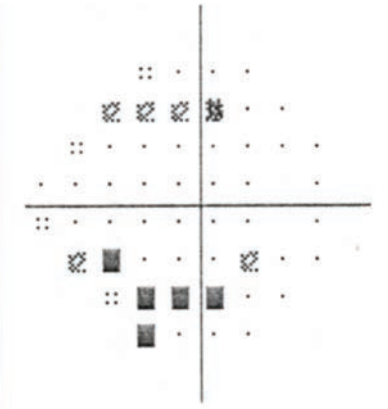
Pharmaceuticals

Medical therapy that has been successful in reducing IOP in eyes with PCACG include beta blockers, miotics, alpha-2 adrenergic agonists, and prostaglandin analogs.[11,12] Research shows that the prostaglandins work especially well in eyes with PCACG that need IOP reduction both before and after LPI, though the mechanism remains unclear.[13-16]

Lensectomy

Because the crystalline lens can contribute to the development of PCACG, lensectomy remains a viable option for some eyes. The exact mechanism by which cataract surgery lowers IOP in glaucomatous eyes remains unknown. Evidence suggests that in eyes with narrow and closed angles, the level of IOP lowering after cataract surgery is proportional to the resultant widening of the angle.[17] Phacoemulsification and intraocular lens implantation can lower IOP, reduce or remove the critical anatomical characteristics that produce pupillary block, and subsequently increase angle width.[17] Research shows, in eyes with PCACG and coexisting cataract, that phacoemulsification alone can significantly reduce both IOP and the need for topical therapy, supporting cataract removal as a primary treatment rather than LPI and stepped-medical therapy.[18,19]

The role of clear lensectomy (i.e., extraction of the non-cataractous lens) in patients with PCACG is unclear. The Effectiveness in Angle Closure Glaucoma of Lens Extraction (EAGLE) study, a prospective, randomized clinical trial now underway, will compare the safety and effectiveness of LPI and medical therapy to clear lens extraction for patients with newly diagnosed PCACG.[20] With any luck, the results of the EAGLE study will help guide management of these challenging patients.



Visual field damage in a patient with primary chronic angle closure glaucoma.

We educated our patient about his condition and the ultimate need for surgical intervention. Because his lenses were relatively clear with poor visual potential in his left eye (and perhaps partially due to his lack of insurance) he opted to initially undergo a less invasive and less expensive LPI. After the procedure, his anterior chamber angles opened to at least posterior pigmented trabecular meshwork (with minimal PAS) with an immediate reduction in IOP. However, in the course of several months, his IOP did elevate due to trabecular compromise induced by the appositional closure, necessitating use of a topical glaucoma medication.

While this patient was successfully managed with conventional methods, the EAGLE study may ultimately change our therapeutic algorithm from LPI and medical therapy to clear lens extraction. ■

1. Bonomi L, Marchini G, Marraffa M, et al. Epidemiology of angle-closure glaucoma: prevalence, clinical types, and association with peripheral anterior chamber depth in the Egna-Neumarket Glaucoma Study. Ophthalmology. 2000;107(5):998-1003.
2. Mok K, Lee V. Synechial angle closure pattern in Chinese chronic primary angle-closure glaucoma patients. J Glaucoma. 2001;10(5):427-8.
3. Lowe RF. Primary creeping angle closure glaucoma. Br J Ophthalmol 1964;48:544.
4. Wang T, Liu L, Li Z, et al. Studies of mechanism of primary angle closure glaucoma using ultrasound biomicroscope. Zhonghua Yan Ke Za Zhi. 1998;34(5):365-8.
5. Tarongoy P, Ho CL, Walton DS. Angle-closure glaucoma: the role of the lens in the pathogenesis, prevention, and treatment. Surv Ophthalmol. 2009;54(2):211-25.
6. He M, Friedman DS, Ge J, et al. Laser peripheral iridotomy in primary angle-closure suspects: biometric and gonioscopic outcomes: the Liwan Eye Study. Ophthalmology. 2007;114(3):494-500.
7. Hsiao CH, Hsu CT, Shen SC, et al. Mid-term follow-up of Nd:YAG laser iridotomy in Asian eyes. Ophthalmic Surg Lasers Imaging. 2003;34(4):291-8.
8. Chen MJ, Cheng CY, Chou CK, et al. The long-term effect of Nd:YAG laser iridotomy on intraocular pressure in Taiwanese eyes with primary angle-closure glaucoma. J Chin Med Assoc. 2008;71(6):300-4.
9. Sihota R, Lakshmaiah NC, Walia KB, et al. The trabecular meshwork in acute and chronic angle closure glaucoma. Indian J Ophthalmol. 2001;49(4):255-9.
10. Rosman M, Aung T, Ang LP, et al. Chronic angle-closure with glaucomatous damage: long-term clinical course in a North American population and comparison with an Asian population. Ophthalmology. 2002;109(12):2227-31.
11. Ruangvaravate N, Kitnarong N, Metheetrairut A, et al. Efficacy of brimonidine 0.2 per cent as adjunctive therapy to beta-blockers: a comparative study between POAG and CACG in Asian eyes. J Med Assoc Thai. 2002;85(8):894-900.
12. Aung T, Wong HT, Yip CC, et al. Comparison of the intraocular pressure-lowering effect of latanoprost and timolol in patients with chronic angle closure glaucoma: a preliminary study. Ophthalmology. 2000;107(6):1178-83.
13. How AC, Kumar RS, Chen YM, et al. A randomised crossover study comparing bimatoprost and latanoprost in subjects with primary angle closure glaucoma. Br J Ophthalmol. 2009;93(6):782-6.
14. Chen MJ, Chen YC, Chou CK, et al. Comparison of the effects of latanoprost and bimatoprost on intraocular pressure in chronic angle-closure glaucoma. J Ocul Pharmacol Ther. 2007;23(6):559-66.
15. Chen MJ, Chen YC, Chou CK, et al. Comparison of the effects of latanoprost and travoprost on intraocular pressure in chronic angle-closure glaucoma. J Ocul Pharmacol Ther. 2006;22(6):449-54.
16. Gupta V, Srinivasan G, Sharma A, et al. Comparative evaluation of bimatoprost monotherapy in primary chronic angle closure and primary open angle glaucoma eyes: a three-year study. J Ocul Pharmacol Ther. 2007;23(4):351-8.
17. Pachimkul P, Intajak Y. Effect of lens extraction on primary angle closure in a Thai population. J Med Assoc Thai. 2008;91(3):303-8.
18. Lai JS, Tham CC, Chan JC. The clinical outcomes of cataract extraction by phacoemulsification in eyes with primary angle-closure glaucoma (PACG) and co-existing cataract: a prospective case series. J Glaucoma. 2006;15(1):47-52.
19. Hata H, Yamane S, Hata S, et al. Preliminary outcomes of primary phacoemulsification plus intraocular lens implantation for primary angle-closure glaucoma. J Med Invest. 2008;55(3-4):287-91.
20. Azuara-Blanco A, Burr JM, Cochran C, et al. Effectiveness in Angle-closure Glaucoma of Lens Extraction (EAGLE) Study Group. The effectiveness of early lens extraction with intraocular lens implantation for the treatment of primary angle-closure glaucoma (EAGLE): study protocol for a randomized controlled trial. Trials. 2011;12:133.

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By Derek N. Cunningham, OD, and Walter O. Whitley, OD, MBA

Postoperative String of Pearls

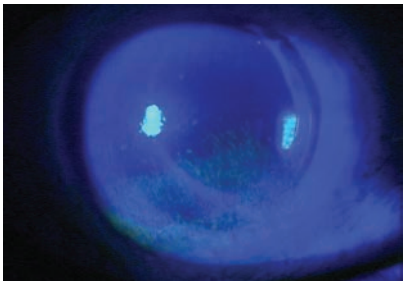
Addressing these issues could make the difference between 20/happy and 20/unhappy.

In our estimation, only about 30% to 40% of optometrists actively comanage cataract surgery postoperatively. Numerous factors contribute to this number, such as insurance, practice modality, patient demographics and possibly a lack of surgeons willing to "share care" (keep looking, because many will). But optometry needs to play a bigger role in pre- and postoperative care to meet patient demands and ensure best outcomes.

Risks of Surgery

As safe as cataract surgery is, it still carries risk. The preoperative evaluation and patient education are keys to ensuring patients have realistic expectations. By addressing issues preoperatively we can minimize postoperative concerns. Here are four of the most common conditions that affect our patients' vision postoperatively:

Cystoid Macular Edema (CME) is the most common cause of visual decline following uncomplicated cataract surgery. The development of CME is due in part to prostaglandin-mediated breach of blood-retinal barrier. It is estimated to occur in 1% to 3% of low-risk cataract cases with an onset of four to six weeks postoperatively.[1] CME should be considered in patients who present with best corrected visual acuity (BCVA) worse than the previous examination. For patients at higher risk for CME, topical NSAIDs are prescribed one week prior to surgery. Treatment includes topical NSAIDs, oral steroids, periocular steroid injections, and pars plana vitrectomy.



Keep any eye out for dry eye, which is commonly seen perioperatively.

The development of ***Posterior Capsular Opacification (PCO)*** is due to a proliferation of equatorial lens epithelium along the posterior capsule, which appears as a cloudy membrane. Symptoms include decreased BCVA, foggy vision and glare. The incidence of PCO is 10% to 25% and may present weeks, months or even years after the procedure.[2] It is easily treated with a YAG laser capsulotomy.

Ocular Surface Disease (OSD) is the most common reason for decreased vision after surgery. We cannot overemphasize the importance of aggressively treating OSD preoperatively. Studies show the incidence of dry eye is 80% of patients preoperatively and 87% of patients postoperatively.[3-5] Consider OSD in patients who complain of fluctuating vision, burning or irritation after surgery. Signs include decreased tear film break-up time, corneal staining, increase OSD index scores, MGD and increased osmolarity and MMP-9 levels. Contributing factors include topical anesthesia, multiple eye drops with preservatives, light exposure and corneal incisions. Treatment includes artificial tears, nutraceuticals, topical anti-inflammatories and punctal occlusion.

Patient expectations are at an all-time high for cataract surgery, especially elective procedures such as femtosecond laser surgery coupled with premium IOL technology. Education on realistic outcomes is important—and that includes the possibility of ***residual refractive error***. Patients may need a spectacle prescription for full-time wear or for certain activities. Discuss with your surgeon how to manage patients with residual refractive error after toric or multifocal implants. Many offer enhancements with laser refractive surgery or limbal relaxing incisions, which is included in the elective surgical fee.

Wrapping it All Up

With more than three million cataract procedures performed annually, optometrists play a key role in preparing patients for surgery and managing their care perioperatively. All complications, no matter the issue, should be discussed with your surgical team. Communication is key both pre- and postoperatively, and having everyone on the same page will help ensure the best possible outcomes. ■

1. Samiy N, Foster CS. The role of nonsteroidal antiinflammatory drugs in ocular inflammation. Int Ophthalmol Clin. 1996;36(1):195-206.
2. Schaumberg D,. et al. A systematic overview of the incidence of posterior capsule opacification. Ophthalmology. 1998;105(7):1213-21.
3. Trattler WB, Reilly CD, Goldberg DF, et al. Clinical study report: Cataract and dry eye: prospective health assessment of cataract patients ocular surface study. Poster, ASCRS/ASOA Symposium & Congress. San Diego; March 2011.
4. Roberts CW, Elie ER. Dry eye symptoms following cataract surgery. Insight. 2007;32(1):14-23.
5. X-M Li, et al. Investigation of dry eye disease and analysis of the pathogenic factors in patients after cataract surgery. Cornea. 2007;26(9)S1:S16-20.

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Product Review

Contact Lenses

Contact Lens Two-Packs

For a limited time, patients interested in color contact lenses can purchase two-packs of Air Optix Colors to try a new color without committing to a full supply. The two-count packages will be available until Sept. 30, 2015, or while supplies last, according to Alcon.

The lenses are available with an 8.6mm base curve and a 14.2mm diameter. The contact lens power ranges from +6.00D to -6.00D in 0.25D steps (including plano) and -6.50D to -8.00D in 0.50D steps.

Visit www.alcon.com.

Intraocular lenses

Multifocal IOL to Treat Cataract Patients

A new multifocal intraocular lens will soon be available for cataract surgery patients. The FDA recently approved Alcon's AcrySof IQ Restor +2.5D IOL, which is indicated for adult patients with and without presbyopia undergoing cataract surgery, the company says.

The Centers for Medicare and Medicaid Services (CMS) added the new IOL to the list of CMS recognized presbyopia-correcting IOLs, confirming that AcrySof IQ Restor +2.5D IOLs will be eligible for reimbursement as both a Medicare-covered service and as a non-covered service.

The product will be available in the United States in the near future.

Visit www.novartis.com.

Eyelid Care

Lash Advance Trial Cards

Practitioners can offer their patients the opportunity to try MediNiche's Lash Advance with low cost trial coupons. The company is offering coupons that allow doctors, their staff or their patients to purchase a three-month supply of Lash Advance at one-third the regular retail price.

The program will run through 2015 and is intended to increase professional and patient awareness and trial use of Lash Advance, without practitioners stocking the product in their dispensary, the company says.

Visit www.mediniche.com.

Retinal Imaging

New Digital Retinal Camera

Practitioners can consider upgrading their retinal imaging technology with the new CR-2 AF digital non-mydriatic retinal camera, which offers a unique contrast enhancement technology to correct for reduced contrast, Canon says. The camera's contrast enhancement feature provides increased image clarity from previous models by emphasizing the difference in 'redness' and 'brightness' of blood vessel structures relative to their surroundings. By expanding the range of brightness values in a fundus image, the practitioner is able to better discriminate minute retinal details between areas initially having small differences in density, Canon says.

The CR-2 AF features low-flash intensity to minimize pupil constriction and shorten the time needed to take multiple pictures. Additionally, its lightweight design makes it ideal for offices with limited space or where telemedicine portability is required, Canon says.

Visit www.usa.canon.com/eye-care.

Dispensary

Updated Generator Model

A new generation of surface generator can help clinicians improve dry-cut milling technology for processing Trivex (PPG Optical Products), CR39 (PPG Optical Products), polycarbonate and high index lenses, according to Coburn Technologies. The SGX Pro operates on Windows 7 and includes touch screen and USB capabilities.

SGX Pro has one of the widest curve ranges and provides the same dependability and accuracy of earlier SGX models, the company says. Although Coburn has discontinued production of its SGX Plus generator, it will continue to provide support and service to existing SGX Plus generators.

Visit www.coburntechnologies.com. ■

A New Multifocal: Built from the Ground Up

Kurt Moody, OD, FAAO

1-DAY ACUVUE® MOIST Brand MULTIFOCAL Contact Lens

1-DAY ACUVUE® MOIST Brand MULTIFOCAL Contact Lenses are an exciting new option for your presbyopic patients.

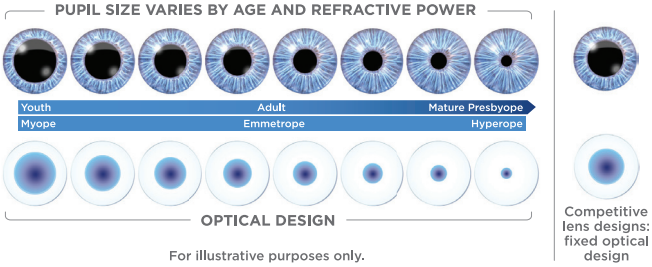
In developing this new aspheric center-near design, the engineers at Johnson & Johnson Vision Care, Inc., drew on lessons from more than 15 years of experience with presbyopic lenses, combined with data from thousands of lens fits in practices across the world, to arrive at a concept that is simple yet revolutionary: There are anatomical differences in presbyopic patients that could drive differences in fit success, thus no one lens design will work for every presbyope. So, we made 183 lens designs targeted to address these differences.

The uniquely optimized optic designs of this lens have been created to address the natural variation in pupil size according to age and refractive power. And, the unique hybrid-back curve design was developed to help maintain centration over the pupil, and preserve the integrity of the front surface optics.

In a clinical study, 94% of subjects were successfully fit in four or fewer total lenses: Three in five patients on the very first pair recommended by the fit guide, an additional 20% with just one adjustment, and another ~15% with only two adjustments (see sidebar).

Pupil optimization

It is well known that pupil size decreases with age. Less well known is the fact that there is a very consistent, natural variation of pupil size by refractive status, with myopes having larger pupils than hyperopes, and high myopes having larger pupils than low myopes.



For illustrative purposes only.

Because of their complex optics, the success of multifocal lenses is greatly influenced by pupil size. If the pupil is larger or smaller than the optical design of the lens, for example, the image quality will be reduced. That's why a single optical design just won't work for every presbyope.

Based on extensive data to validate this phenomenon, we've created 183 unique optical designs. This sounds like an intimidating number, but it actually means that the work of figuring out the best combination has been done already. For every power/add combination, from +6.00 D to -9.00 D in 0.25 D steps, the lenses are designed to optimize visual performance across ~95% of the range of pupil sizes expected for any given refractive error and add power.

Hybrid back-curve design

Lens centration also becomes a critical factor with more complex multifocal optics. The hybrid back-curve design of 1-DAY ACUVUE® MOIST Brand

MULTIFOCAL relies on an aspheric central back curve, as well as low modulus, to help it drape well on the cornea, preserving the integrity of the front-surface optics. Meanwhile, the spherical periphery of the back curve helps maintain centration.

The lens has a base curve of 8.4 with a 14.3-mm diameter, but the unique design gives it a very flexible fit profile. In clinical studies, normal eyes with Ks between 38.75 D and 48.50 D could be successfully fit with a single base curve.

Proven platform

The etafilcon A material has a 30-year track record of success in delivering comfortable lens wear and crisp vision to millions of patients around the world. The proven material of the 1-DAY ACUVUE® MOIST Family of Lenses uses dual-action technologies to keep moisture in and irritation out, which helps to address the essential needs of the aging eye:

- LACREON® Technology to lock moisture in
- An INFINITY EDGE™ Design and low modulus to minimize mechanical irritation
- A unique ability to attract and maintain the enzyme lysozyme in its beneficial natural state*
- Class 2 UV blocking†**

This comfortable daily disposable platform is ideal for presbyopic patients, who are far more likely to struggle with irritation and symptoms of contact lens-related dryness as they age.

Clinical Experience Pearls

Drew Dayton, OD

As an unhappy presbyope, I have personally tried every multifocal contact lens on the market. Not only is 1-DAY ACUVUE® MOIST Brand MULTIFOCAL the first one I've been able to wear, but it has been wildly successful with my patients, too. Of the first 15 I tried it on, 11 purchased lenses – a previously unheard of percentage for me. The difference is that with 1-DAY ACUVUE® MOIST MULTIFOCAL, my patients don't have to compromise distance or comfort to get the near vision they want.

Four steps to a good fit:

1. Perform a good functional refraction for distance, being careful not to over-minus.
2. Determine neurosensory eye dominance by testing a +1.0 lens over each eye, with the patient looking binocularly, to see which one blurs their vision more.
3. Determine the best functional add (LOW, MID, or HIGH), based on their age, history, and the visual tasks that are a high priority for that patient.
4. Use the fit guide. I promise you, it will help you avoid mistakes and reduce chair time! Let the lens settle for 10 minutes and let the patient experience real-life distance and near tasks (ideally for several days at home) before making any changes.

In my experience, most patients are successful on that very first fit. If you need to refine, try these quick tips from the fit guide:

- To improve DISTANCE performance, reduce the add power in the dominant eye
- To improve NEAR performance, add +0.25 D to the non-dominant eye
- NOTE: Refer to the Fitting Tips for patients requiring a HIGH add, as small differences to the directions above for fitting these patients.

*Data on file, 2014. Based on in-vitro data; clinical studies have not been done directly linking differences in lysozyme profile with specific clinical benefits.
†Helps protect against transmission of harmful UV radiation through the cornea and into the eye.
**WARNING: UV-absorbing contact lenses are NOT substitutes for protective UV-absorbing eyewear, such as UV-absorbing goggles or sunglasses, because they do not completely cover the eye and surrounding area. You should continue to use UV-absorbing eyewear as directed.
NOTE: Long-term exposure to UV radiation is one of the risk factors associated with cataracts. Exposure is based on a number of factors such as environmental conditions (altitude, geography, cloud cover) and personal factors (extent and nature of outdoor activities). UV-blocking contact lenses help provide protection against harmful UV radiation. However, clinical studies have not yet been done to demonstrate that wearing UV-blocking contact lenses reduces the risk of developing cataracts or other ocular disorders. Consult your eye care practitioner for more information.

Meetings + Conferences

July 2015

■ **16-19.** *2015 Victoria Conference*. Inn at Laurel Point, Victoria, British Columbia, Canada. Hosted by: Pacific University. Key faculty: Terry Burris, Danica Marelli, Curtis Baxstrom, Tad Buckingham. CE hours: 20. To register, go to www.pacificu.edu.

■ **16-19.** *Florida Optometric Association Annual Convention*. The Breakers, Palm Beach, FL. Hosted by: Florida Optometric Association. Key faculty: William Marcolini, Ian Gaddie, Mark Dunbar, Christian Guier, Paul Palmber, April Jasper. CE hours: 30 Total; 22 per OD. To register, call Jessica Brewton at (805) 877-4697 or go to www.floridaeyes.org.

■ **17-18.** *OOPA Summer CE Event*. The Resort at the Mountain, Welches, OR. Hosted by: Oregon Optometric Physicians Association. Key faculty: Gordon Johns, Beth Kinoshita, Lorne Yudcovitch, Rebecca Uhlig, Robert Egan, Stan Teplick. CE hours: 13. To register, email Lynne Olson at lynne@oregonoptometry.org or go to www.oregonoptometry.org.

■ **22-25.** *Northern Rockies Optometric Conference*. Snow King Hotel, Jackson, WY. Hosted by: Northern Rockies Optometric Conference. Key faculty: Ian Ben Gaddie, Mark Dunbar, Rebecca Wartman. CE hours: 16. To register, go to www.nrocmeeting.com.

■ **23-26.** *New Technologies and Treatments in Vision Care*. Wailea Beach Marriott Resort & Spa, Wailea, HI. Hosted by: Review of Optometry. Key faculty: Paul Karpecki, Brad Sutton, Randall Thomas, Ron Melton. CE hours: 14. To register, email Lois DiDomenico at ReviewMeetings@jobson.com, call (866) 658-1772 or visit www.reviewofoptometry.com.

■ **23-26.** *CE in the Rockies*. Rocky Mountain Park Inn, Estes Park, CO. Hosted by: University of Houston College of Optometry. Key faculty: Danica Marrelli. CE hours: 21. To register, email optce@uh.edu or go to www.ce.opt.uh.edu/.

■ **26-Aug. 2.** *Getting Comfortable with Retinal Care: An Optometric View*. Alaska Glacier Bay Cruise, departs Seattle, WA. Hosted by: Dr. Travel Seminars/The New Jersey Society of Optometric Physicians. Key faculty: Diana Shechtman. CE hours: 16. To register, email Robert Pascal at info@DrTravel.com or go to DrTravel.com.

■ **31-Aug. 2.** *Southwest Florida Educational Retreat*. South Seas Island Resort, Ft. Myers, FL. Hosted by: Southwest Florida Optometric Association. Key faculty: Jimmy Bartlett, Tammy Than, Ron Foreman. CE hours: 18. To register, email Brad Middaugh at swfoa@att.net or go to www.swfoa.com.

■ **31-Aug. 2.** *Colorado Vision Summit*. Crown Plaza DIA, Denver. Hosted by: Colorado Vision Summit. Key faculty: John Neal, John Winton, Doug Devries, Dominick Maino. CE hours: 40 Total; 17 per OD. To register, email Lindsay Wright at lwright@visioncare.org or go to www.visioncare.org.

August 2015

■ **3-10.** *AEA Cruises Baltic Cruise Seminar*. Silversea Silver Whisper, departs Copenhagen. Hosted by: AEA Cruises. Key faculty: Louise Sclafani. CE hours: 10. To register, email Marge McGrath at aeacruises@aol.com or go to www.optometriccruiseseminars.com.

■ **6-10.** *Art & Science of Optometric Care–A Behavioral Perspective*. Michigan College of Optometry, Big Rapids, MI. Hosted by: OEP Foundation. Key faculty: Robert A. Hohendorf. CE hours: 35. To register, email Theresa Krejci at TheresaKrejciOEP@verizon.net or go to www.oepf.org.

■ **14-16.** *1st World Congress of Optometry*. Plaza Mayor Convention and Exhibition Centre, Medellin, Columbia. Hosted by: The World Council of Optometry and La Federación Colombiana de Optómetras. To register, go to www.worldcongressofoptometry.org.

■ **15-16.** *IU Cornea & Contact Lens Conference*. IU School of Optometry. Bloomington, IN. Hosted by: IU School of Optometry. Key faculty: Jason Jedlicka, Pete Kollbaum, Sue Kovacich, Tony Van Alstine, Carolyn Begley. CE hours: 14. To register, email Cheryl Oldfield at coldfiel@indiana.edu or go to www.opt.indiana.edu/ce/seminars.htm.

■ **16-17.** *Glaucoma: Grand Rounds*. Marshall B. Ketchum University, Fullerton, CA. Hosted by: SCCO at MBKU. Key faculty: George Comer, John Nishimoto, Mark Sawamura, Judy Tong. CE hours: 16. To register, go to www.ketchum.edu/ce.

■ **19.** *AAO-NJ Conference*. Jumping Brook Country Club, Neptune, NJ. Hosted by: American Academy of Optometry New Jersey Chapter. CE hours: 6. To register, email Dennis Lyons at Dhl2020@aol.com or call (732) 920-0110.

■ **20-23.** *108th SCOPA Annual Meeting*. Westin Hilton Head Island Resort and Spa, Hilton Head Island, SC. Hosted by: SC Optometric Physicians Association. CE hours: 21. To register, email Jackie Rivers at jrivers@sceyedoctors.com, call (803) 799-6721 or go to www.sceyedoctors.com.

■ **27-29.** *International Vision Conference*. Hyatt Manchester, San Diego. Hosted by: OD Excellence and PFO Global. Key faculty: John McGreal, Jim Grue, Bob Schultz, Jim Riverson, Nathan Lighthizer. CE hours: 17. To register, go to www.ivisionconf.org.

■ **28-30.** *Alumni Weekend*. UAB School of Optometry, Birmingham, AL. Hosted by: UAB School of Optometry. Key faculty: Ian Gaddie, Marie Bodack, Diana Shechtman, Scot Morris, Sunny Sanders. CE hours: 18. To register, go to www.uab.edu/optometry.

September 2015

■ **2-13.** *Adventure CE Italy*. Siena/Sorrento/Rome, Italy. Hosted by: Tropical CE. Key faculty: Jill Autry, Ian Ben

Gaddie. CE Hours: 20. To register, email Stuart Autry at sautry@tropicalce.com or go to www.tropicalce.com.

■ **9-12.** *Envision Conference 2015*. Grand Hyatt Denver. Hosted by: Envision University. CE hours: 90+; 23 per OD. To register, email Bonnie Harrell at bonnie.harrell@envisionus.com or go to www.envisionuniversity.org.

■ **10-13.** *GWCO Congress 2015*. Oregon Convention Center, Portland, OR. Hosted by: Great Western Council on Optometry. Key faculty: Paul Karpecki, April Jasper, Mile Brujic. CE hours: Total: 71; 26 per OD. To register, email Tracy Oman at gwco@gwco.org or go to www.gwco.org.

■ **16-19.** *International Vision Expo West*. Sands Expo & Convention Center, Las Vegas. Hosted by: International Vision Expo & Conference. CE hours: 390+; 31 per OD. To register, email Rachel Spencer at Rachel@visitaccess.com, call (540) 344-8499 or visit www.visionexpowest.com.

■ **17-20.** *EyeFlyFish 2015*. Allenberry Resort on the Yellow Breeches, Boiling Springs, PA. Hosted by: Charles Griffen and Mark Boas. CE hours: 6. To register, email Mark Boas at mboas56852@aol.com or visit www.eyeflyfish.com.

■ **17-20.** *2015 IOA Annual Convention*. Westin Chicago Northwest Hotel, Itasca, IL. Hosted by: Illinois Optometric Association. CE hours: Total: 24; 15 per OD. To register, visit www.ioaweb.org.

■ **17-21**. *VT/Visual Dysfunctions*. The Holiday Inn, Baltimore, MD. Hosted by: OEP Foundation. Key faculty: Paul Harris. CE hours: 35. To register, email Theresa Krejci at TheresaKrejciOEP@verizon.net or visit www.oepf.org.

■ **18-20.** *Colorado Vision Training Conference*. YMCA of the Rockies, Estes Park, CO. Hosted by: OEP Foundation. Key faculty: Paul Harris. CE hours: 12. To register, email Jamie Anderson at drjamieanderson@gmail.com, call (720) 870-2828 ext. 151 or visit www.oepf.org.

■ **18-20.** *Vermont Optometric Association Fall Conference*. Woodstock Inn and Resort, Woodstock, VT. Hosted by: Vermont Optometric Association. CE hours: 16. To register, email Rebecca Hogan at vtcecoordinator.@gmail.com or visit vtoptometrists.org.

■ **23-25.** *CE in Italy*. Hotel Silla, Florence, Italy. Hosted by: James Fanelli. Key faculty: James Fanelli, Joseph Pizzimenti. CE hours: 12. To register, email James Fanelli at jamesfanelli@CEinItaly.com or visit www.CEinItaly.com.

To list your meeting, please send the details to:

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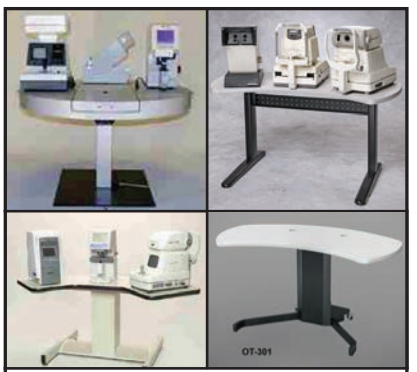

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REVIEW OF OPTOMETRY

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Diagnostic **Quiz**

Season of the Itch

By Andrew S. Gurwood, OD

A 27-year-old female reported to the office with a chief complaint of red, itchy eyes for three months. She explained that she had been placed on Pataday QD PRN by her internal medicine doctor, but did not acheive sustained relief.

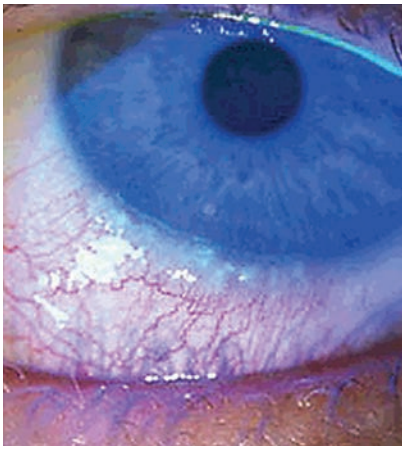
Her ocular history was noncontributory. Her systemic disease history was positive for rheumatoid arthritis, for which she medicated with Enbrel (etanercept, Immunex) 50mg/week IM. She denied allergies to medications and foods.

Diagnostic Data

Her best-corrected entering visual acuities were 20/15 OU at distance and near. Her external examination was normal with no evidence of afferent pupillary defect. The pertinent biomicroscopic examination of the anterior segment is demonstrated in the photograph. Goldmann applanation tonometry measured 14mm Hg OU. The dilated fundus examination revealed no peripheral pathologies in either eye.

Your Diagnosis

Does this case require any additional tests? What is your diagnosis? How would you manage this patient? What's the likely prognosis? To find out, please visit *Review of Optometry* online at www.reviewofoptometry.com.



A 27-year-old female patient who was previously diagnosed with rheumatoid arthritis presented with a chief complaint of itchy eyes for three months. Can you identify the underlying cause?

Retina Quiz Answers *(from page 88):* 1) b; 2) a; 3) b; 4) a; 5) a.

Next Month in the Mag

August will be *Review of Optometry*'s annual "Contact Lens Report," which will focus on issues involving contact lenses and comfort. Topics will include:

- *Yes, Dry Eye Patients Can Wear Contact Lenses*
Though ocular surface comprimise is technically a contraindication, it is possible to offer lenses and ensure comfort.

- *Anatomy of an OCT Scan*
Optical coherence tomography images are now vital for visualizing disease progression. Bone up on how to analyze them using this helpful guide.

- *Real-World Factors that Affect Contact Lens Success*
Environmental and lifestyle factors can influence choice of lens material and other decisions.

- *Ocular Manifestations of Neurofibromatosis*
An overview of a newly identified biomarker of this tumor type.

- *Systemic Infectious Disorders of the Anterior Segment*
(earn 2 CE credits)
Could your patient's red eye indicate an underlying disease such as herpes, lyme disease, syphilis, chlamydia or HIV? This article reviews the protocol for diagnosis and treatment.

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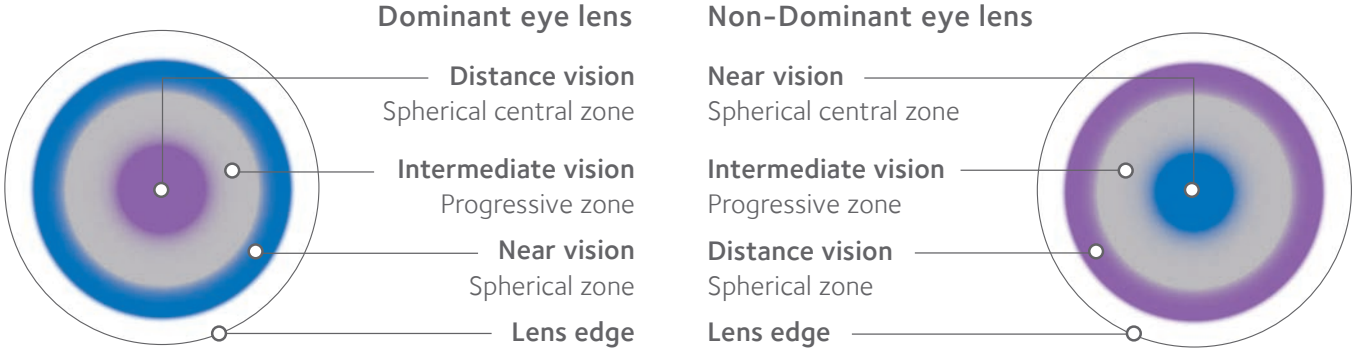

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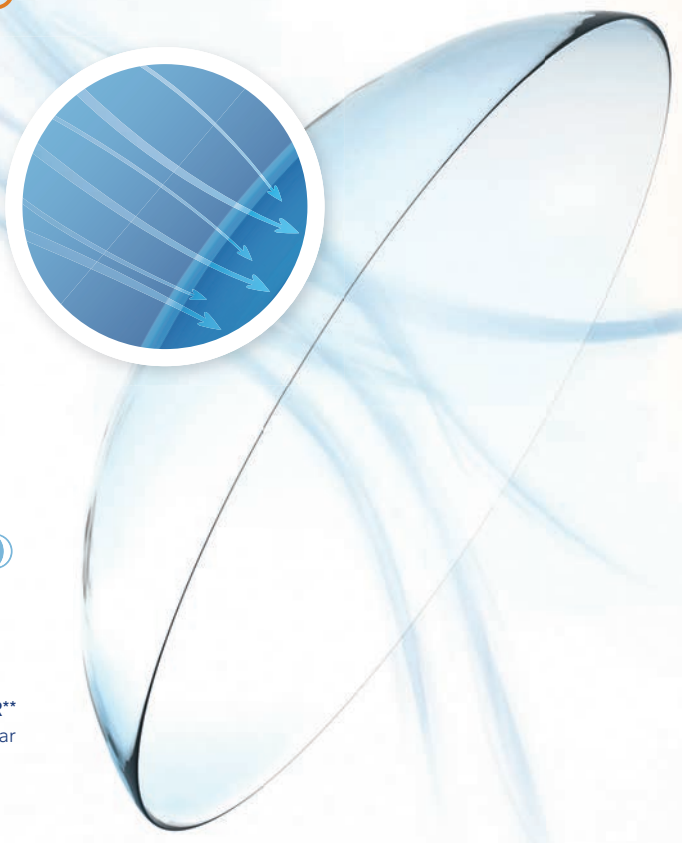

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Reference: 1. In a survey of 2,115 daily and extended wear contact lens patients. Alcon data on file, 2012. **2.** In a survey of 302 optometrists in the U.S.; Alcon data on file, 2012. **3.** Based on the ratio of lens oxygen transmissibilities; Alcon data on file, 2009, 2010. **4.** Nash W, Gabriel M, Mowrey-Mckee M. A comparison of various silicone hydrogel lenses; lipid and protein deposition as a result of daily wear. Optom Vis Sci. 2010;87: E-abstract 105110. **5.** Nash W, Gabriel M. Ex vivo analysis of cholesterol deposition for commercially available silicone hydrogel contact lenses using a fluorometric enzymatic assay. Eye Contact Lens. 2014;40(5):277-282.

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