Therapeutic Review: A New Way to Treat GCA?, *p. 85*

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December 15, 2016

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22ND ANNUAL SURGERY REPORT

COMANAGING CATARACT SURGERY COMPLICATIONS

Know how to differentiate between conditions that may arise after extraction. ***p. 36***

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Sam desires the replacement cycle and affordability of monthly wear but wants to avoid declining comfort over the month, which may be caused by a change in lens hydration.

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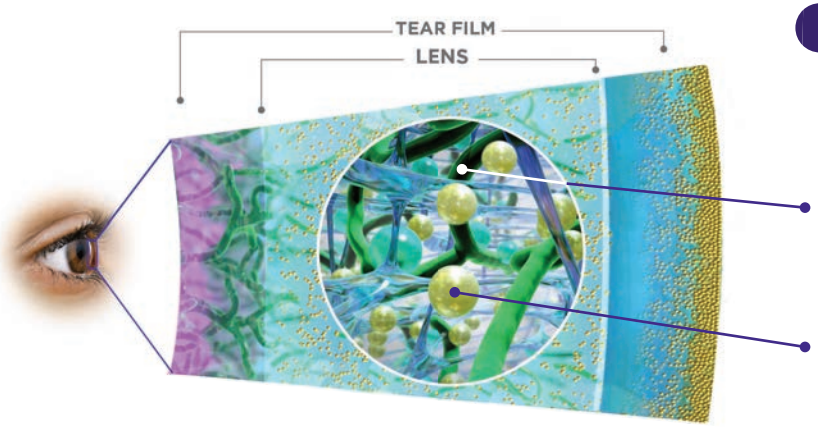

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VOL. 153 NO. 12 ■ DECEMBER 15, 2016

IN THE NEWS

A recently published phase II study suggests treating **wet age-related macular degeneration** (AMD) with a platelet-derived growth factor (PDGF) antagonist in combination with ranibizumab could provide **significant improvement in visual outcomes**. Researchers noted a 62% relative benefit from baseline in study participants treated with 1.5mg anti-PDGF Fovista (pegpleranib, Ophthotech) in combination with Lucentis (ranibizumab 0.5mg, Roche) compared with monotherapy.

Jaffe GJ, Ciulla TA, Ciardella AP, et al. Dual antagonism of PDGF and VEGF in neovascular age-related macular degeneration. Ophthalmol. October 28, 2016. [Epub ahead of print].

A new study shows an **iPad game is more effective than patching for amblyopia treatment**. Half of the pediatric study participants were asked to play the game 10 hours total over two weeks, and the other half were assigned patching therapy. When BCVA was measured at two weeks, 39% of those who used the iPad game reached 20/32 or better compared with 7% in the patching group.

Li SL, Reynaud A, Hess RF, et al. Binocular iPad game vs patching for treatment of amblyopia in children. JAMA Ophthalmol. November 10, 2016. [Epub ahead of print].

A new study compared laser iridotomy (LI) and lens extraction with intraocular lens (IOL) implantation for the **treatment of glaucoma**. By looking at the outcomes of 211 patients who received LI and 208 who received lens extraction with IOL implantation, researchers found **clear lens extraction was more effective** regarding patients' vision and eye pressure levels at three years post-procedure.

Azuara-Blanco A, Burr J, Ramsay C, et al. Effectiveness of early lens extraction for the treatment of primary angle-closure glaucoma (EAGLE): a randomised controlled trial. The Lancet. 2016;388:1389-97.

Optometrists Shouldn't Butt Out

Encouraging smoking cessation is the OD's job, and a new study shows it can work.

By Bill Kekevian, Senior Editor

Optometrists don't simply care for patients' eyes. As research reveals more ocular manifestations of systemic disease, and the profession's scope of practice expands, optometrists are finding themselves making health recommendations, including advice on diet, exercise and tobacco cessation. But as ODs approach these new avenues of patient care, a question lingers: Will patients even listen to them on these issues?

According to new evidence, the American Academy of Optometry says they will. The study, presented at the Academy's meeting in Anaheim last month, looked at 193 patients who were counseled on smoking cessation. The research, conducted by Stanley W. Hatch, OD, found 14.4% of patients ceased tobacco use after counseling by an optometrist.[1] By way of comparison, a 2012 study found the cessation rate of patients counseled by a primary care physician is 15%.[2]

"Other than age, smoking is the biggest risk factor for these conditions, and it can be changed," Dr. Hatch says. Chances are, you know someone who has beaten smoking. Dr. Hatch suggests referring to those stories to help reach out to patients. However you approach it, Dr. Hatch wants to make it clear that it's within the OD's purview. "To the optometrist who feels discussing smoking cessation is outside the scope of practice: Is the management of cataract, macular degeneration, retinal vein and artery occlusion, and uveal melanoma outside their scope of practice? Absolutely not. If you quit smoking, you reduce your risk of blindness and death," he says.

In addition to degrading systemic health, studies show smoking directly affects ocular health and is associated with diseases such as macular degeneration.[3,4]

Dr. Hatch suggests researching cessation programs and finding one you're willing to share with patients. Then, relay this script: "Is there anything I can do to help? Research has shown that solo quitting is rarely successful, but those who participate in a smoking cessation program, see their primary care provider for medication and get counseling have much better success rates. Can I refer you to this local program?"

1. Hatch SW. Effect of tobacco cessation counseling in an eye care practice. Available at www.aaopt.org/sites/default/files/userfiles/2016/Education_Web.compressed.pdf. Accessed November 28, 2016.
2. Pierce J, Cummins S, White M, et al. Quitlines and nicotine replacement for smoking cessation: do we need to change policy? Annual Review of Public Health. 2012 April;33:341-56.
3. Asfar T, Lam B, Lee D. Smoking causes blindness: time for eye care professionals to join the fight against tobacco. Invest Ophthalmol Vis Sci. 2015;56:1120-1.
4. Swanson MW. Smoking deception and age-related macular degeneration. Optom Vis Sci. 2014;91(8):865-71.

NEUROSTIMULATION: A NEW APPROACH TO TEAR PRODUCTION

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1-800-Contacts Making Headway with FTC?

After reviewing comments received in September 2015 about the costs, benefits and impact of the Contact Lens Rule, the FTC has proposed changes, including mandating eye care providers (ECPs) maintain a signed agreement of prescription dispensation for contact lens (CL) patients—for three years.[1]

Such a rule change comes as a hefty blow to optometrists fighting against the selling practices of online giants such as 1-800-Contacts. The FTC's "Notice of Proposed Rulemaking (NPRM)" outlines the review process and the reasons behind the proposed changes.[2]

Although the American Academy of Optometry provided a comment that included peer-reviewed research to support its claim that alternative supply chains for the sale of CLs is an identifiable risk factor for ocular morbidity in CL patients, the FTC concluded "they are not sufficient to reliably demonstrate that purchasing lenses online is a risk factor, or that online purchasers are at a higher risk of developing microbial keratitis or any other ocular complication."[2]

Other organizations and ECPs also provided comments suggesting the Contact Lens Rule creates a mechanism for renewal of expired prescriptions, putting patients' eye health at risk. The FTC claims these comments "did not include any empirical evidence showing that the passive verification mechanism has actually resulted in the renewal of expired prescriptions."[2] Additionally, "other examples of patient harm identified by commenters were either hypothetical or anecdotal."[2]

Yet, the NPRM declares other evidence submitted by commenters *is* informative for the purposes of the rule change. The NPRM provides statistics from 1-800-Contact's comment, pulled from surveys conducted on the company's behalf. The surveys found only 35% of CL wearers reported receiving a copy of their prescription without asking for it, 28% received it after asking and 36% never received it at all.[2] The NPRM claims that, even though these surveys cannot provide a definitive answer regarding automatic release compliance, they, along with "the high number of verifications, the ongoing pattern of consumer complaints and anecdotal reports, and the industry's long history of failing to provide prescriptions" suggest compliance with the automatic prescription release provision needs improvement.[2]

With such evidence, it will be an uphill battle to keep these new rules from being enacted. The FTC is accepting public comments until January 30, 2017.

"If the FTC rule changes become finalized and implemented, I see an adverse potential to disrupt the patient-physician relationship, which places the patient at risk for vision loss," says Greg Caldwell, OD, diplomate of the American Board of Optometry. "These rule changes will also create additional administrative burdens, especially for small businesses. I encourage colleagues to help the AOA and state associations fight back and educate the FTC about why its proposal is rooted in inaccurate information about our role as physicians committed to the health and well-being of our patients."

1. Federal Trade Commission. FTC Seeks Comment on Proposed Changes to Contact Lens Rule. News Release. November 10, 2016. Available at www.ftc.gov/news-events/press-releases/2016/11/ftc-seeks-comment-proposed-changes-contact-lens-rule. Accessed November 18, 2016.
2. Federal Trade Commission. Notice of Proposed Rulemaking. Available at www.ftc.gov/system/files/documents/federal_register_notices/2016/11/contact_lens_rule_nprm111416.pdf. Accessed November 18, 2016.

Brien Holden Humanitarian Award: Good Work and its Accolades

In honor of the memory and efforts of Professor Brien Holden, PhD, DSc, FAAO, the American Academy of Optometry recently announced the establishment of the Brien Holden Humanitarian Award. This new accolade aims to recognize an individual or organization whose humanitarian work in non-profit sectors strives to create or improve sustainable eye care systems in developing communities.

Professor Holden, founder of the Brien Holden Vision Institute, was an internationally renowned scientist, humanitarian and professor at the School of Optometry and Vision Science at the University of New South Wales in Australia. His inspiring life of work was informed by a dream of universal access to vision care and a passion to provide sight for communities where disparities existed.

"The awards given at the Academy of Optometry annual meeting represent our values and our history," says Don Mutti, OD, PhD, incoming awards committee chair. "Each award honors an exceptional life while affirming our progress by celebrating the achievement of each new recipient. Dr. Holden devoted so much of himself to humanitarian work. This new award means the values of improving the vision of all are alive and well in our profession and the Academy." Beginning in 2017, the award will be given annually. Nominations are due by April 1, 2017 to Helenv@aaoptom.org and should include two letters of nomination from Academy Fellows as well as the nominee's CV.

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Study: Most Diabetes Patients Skip Eye Exams

Nearly 60% of Americans with diabetes skip their regular eye exams, according to a study presented at the American Academy of Ophthalmology meeting in October. Researchers reviewed approximately 2,000 patients with diabetes over a four-year stretch. They found that 58% of patients with diabetes did not obtain regular follow-up exams. Subsets within that group show even more troubling numbers, as patients with diabetes who are also smokers were more likely than nonsmokers to neglect eye exams. Patients with less severe diabetes and no resultant eye problems were also skipping out on eye exams. However, patients who have already developed diabetic retinopathy were 30% more likely to follow up, according to the research.

Nonadherence to eye care in people with diabetes. Presented at the 120th annual meeting of the American Academy of Ophthalmology, October 14-18, 2016; Chicago.

Thyroid Eye Disease Discovery

A new study, recently published in *The American Journal of Pathology*, took a closer look at the underlying molecular pathways that lead to scarring in thyroid eye disease (TED) and found that activating the aryl hydrocarbon receptor (AHR) pathway by its ligands blocks collagen production and myofibroblast proliferation.

"Thyroid eye disease is the most common extra-thyroidal manifestation of Graves' disease, an automimmune disorder, and is characterized by myofibroblast accumulation, tissue remodeling and scarring within the orbit," says Joseph Pizzimenti, OD. "Unfortunately, current therapies do not target or prevent the excessive tissue remodeling caused by myofibroblast formation and activation."[1,2]

But now, investigators from the Flaum Eye Institute of the School of Medicine and Dentistry of the University of Rochester believe AHRs may be the key to controlling or preventing tissue remodeling or destruction associated with TED. The researchers compared human orbital fibroblasts from TED patient tissue with tissue from patients without TED, and found the TED orbital fibroblasts expressed higher levels of AHRs than non-TED orbital fibroblasts. They also discovered the AHR ligands turned on AHR-dependent genes, blocking the transforming growth factor (TGF)-β -driven conversion of orbital fibroblasts to scar-forming myofibroblasts.

"TGF-β is a cytokine that induces myofibroblast formation," Dr. Pizzimenti explains. "Because the TGF-β signaling pathway is influenced by AHR signaling pathways, AHR agonists may have the potential to prevent myofibroblast formation in patients with TED, resulting in a therapeutic benefit."[1,2]

"The study looks ahead to AHR and AHR ligands as future 'anti-scarring' therapeutic options for eye diseases and possibly also for other scarring conditions," Dr. Pizzimenti says. "In severe cases of TED, treatment should be customized for each patient in close collaboration with the endocrinologist."

But as promising as these study findings may be, the journey from lab to approval can be a long one, Dr. Pizzimenti cautions. ■

1. Woeller CF, Roztocil E, Hammond CL, et al. The aryl hydrocarbon receptor and its ligands inhibit myofibroblast formation and activation. Am J Pathol. November 11, 2016. [Epub ahead of print].
2. Pelino CJ, Pizzimenti JJ. Axis of activity. Rev Optom. 2014;151(1):67-9.

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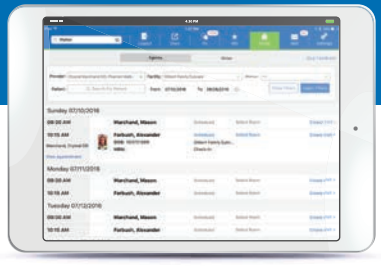

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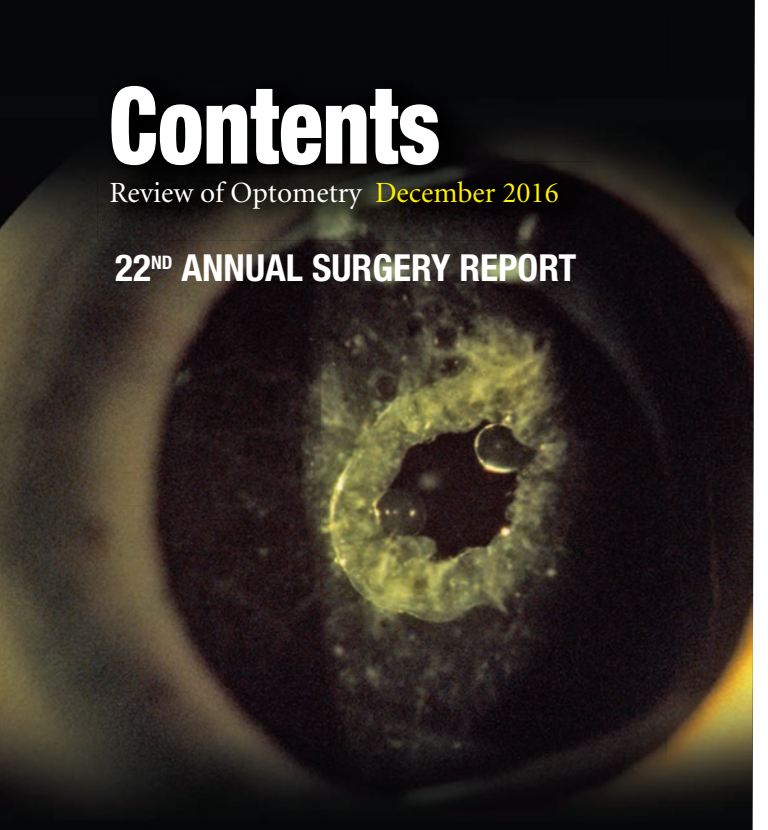

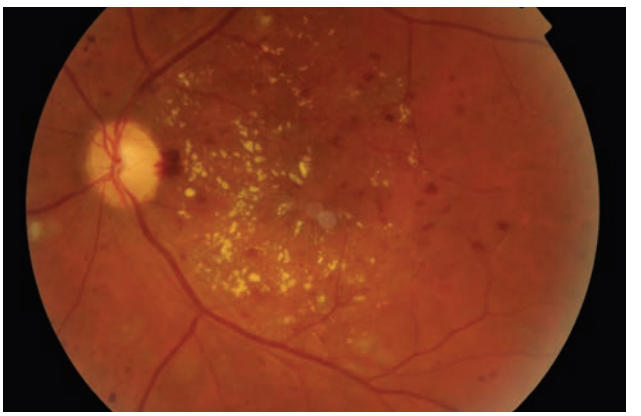

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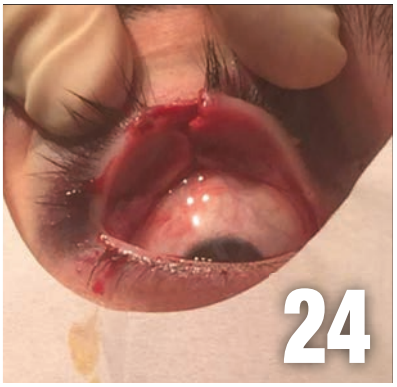

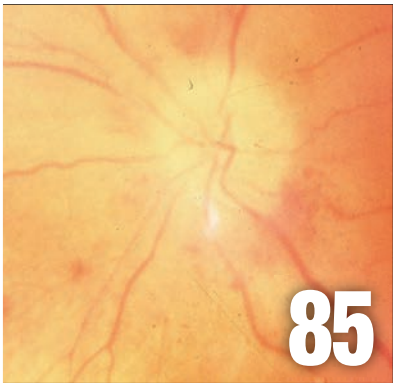

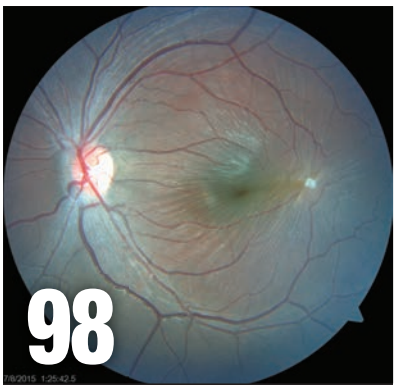

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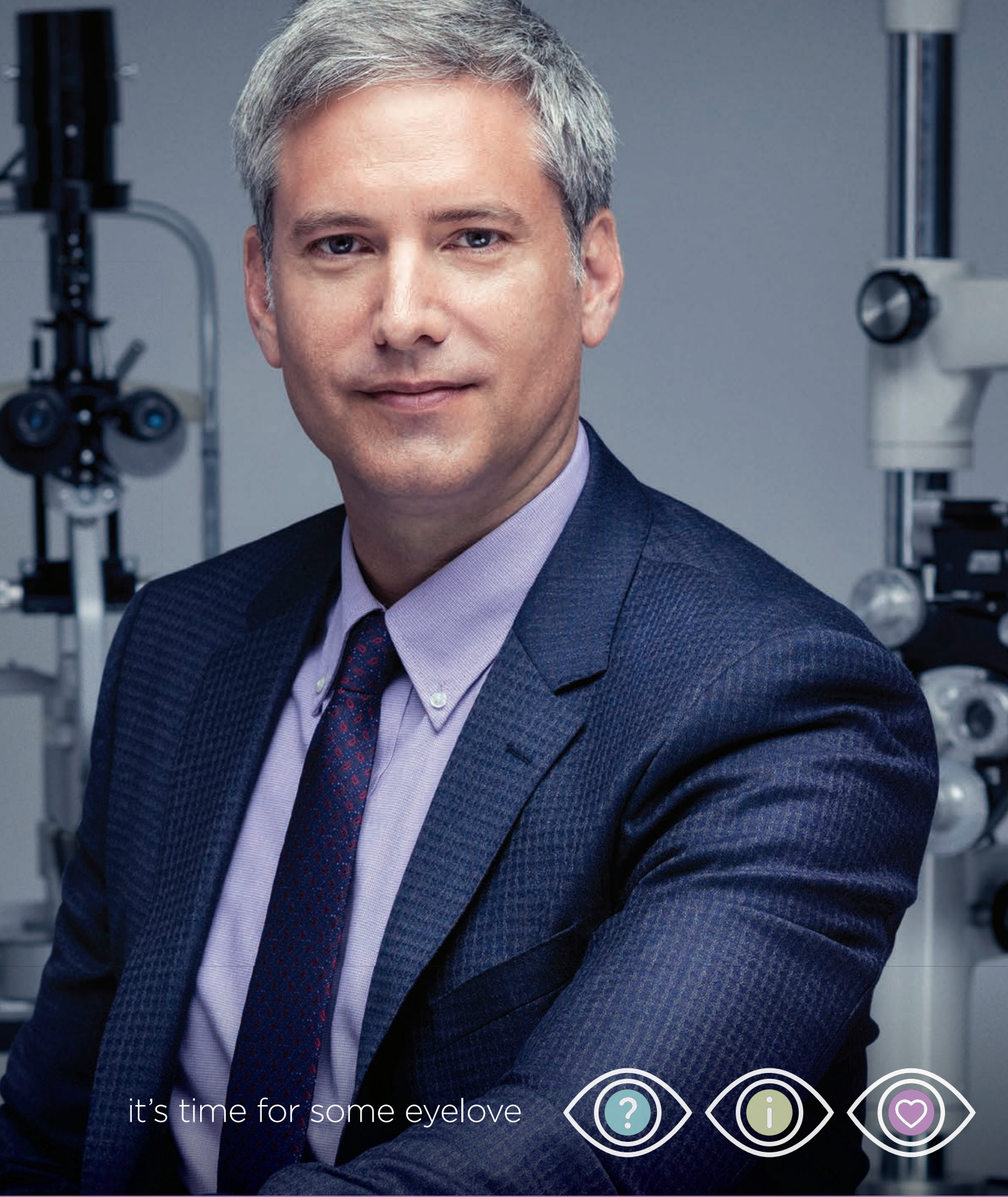
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Outlook

By Jack Persico, Editor-in-Chief

The Best is Yet to Come

Our 125th anniversary year ends with a farewell to the past and a bold embrace of the future.

We at *Review of Optometry* began 2016 by reflecting on what had come before—in this publication and in the profession as a whole—as part of our retrospective to honor the 125th anniversary of our debut. Back then, this journal was called *The Optician,* and optometry itself didn't even have a name. Though its founders had a clear vision of what they hoped to accomplish, anything was possible.

It seems fitting that we end 2016 with this month's focus on surgical comanagement. That's about as far away as you can imagine from what optometry's early pioneers aspired to back in the mid to late 19th century. Most, in fact, might have been incredulous if a time traveler from today showed up at the optical shops and jewelry stores where "refracting opticians" began to separate from traditional opticianry and create the early origins of what we now call optometry.

Younger ODs may not fully realize just how hard it was to get from there to here. It started with the profession's forefathers staking a claim to expertise in vision assessment and correction, raising the bar above the often dubious, or at least well-intentioned but imprecise, methods that came before. But even that seemingly uncontroversial goal required enormous effort; a bitter legislative slog took 23 years to pass optometry licensure laws in every US state. Toss in a few more decades' worth of toil for the passage of DPA and TPA laws (roughly 1968 to 1998) and you begin to see that optometry's advance has met stiff resistance at every turn. Even today, Massachusetts optometrists are still prevented from prescribing glaucoma drugs, a battle long finished in the rest of the country.

Despite a century of successes for optometry, modern threats and potential disruptors require ongoing vigilance. The recent FTC proposal on contact lens prescription release seems positively gift-wrapped for the likes of 1-800-CONTACTS. Online refraction and dispensing services have the potential to completely upend a traditional pillar of practice. And with the Affordable Care Act vulnerable and likely to be rebooted, optometry's role in the broader healthcare landscape could be subject to seismic shifts in response.

The mantra in the early years—"organization, education, legislation"—bears repeating. Optometry won more battles than it lost but never took success for granted. Everyone with a stake in its future needs to participate, especially today. A chaotic political environment underscores the importance of organization and legislation. And as this month's special supplement on 2017 optometric CE events makes plain, professional education has never been stronger, with more than 220 meetings planned next year and an increasingly global footprint.

Optometry on the edge of '17 may not be what founding fathers Charles Prentice and Andrew Cross expected, but its success is a testament to the adventurous spirit they imbued in it. And its future looks bright. ■

PATIENT AND PRACTICE SUCCESS: BEING PROACTIVE

Melissa Barnett, OD, FAAO, FSLS
UC Davis Eye Center, Sacramento, CA

Dr. Barnett was compensated by Alcon for her participation in this advertorial.

As doctors we have received advanced training in a variety of clinical areas, from dry eye disease and meibomian gland dysfunction to astigmatism and presbyopia. Many of us have also spent our professional lives striving to find the best treatment approaches for patients with diverse ocular needs so they can see, look, and feel their best. How do we know what those needs are? As busy practitioners, do we always take the time to really communicate and listen to our patients? And if our patients tell us that "everything is ok" with their current lenses, do we learn how to probe a little deeper? I have found that many of my patients are "silent sufferers" who may not be open to discuss their ocular problems without my encouragement; others tell me that their contact lenses are "fine" because they do not know that any superior options exist.

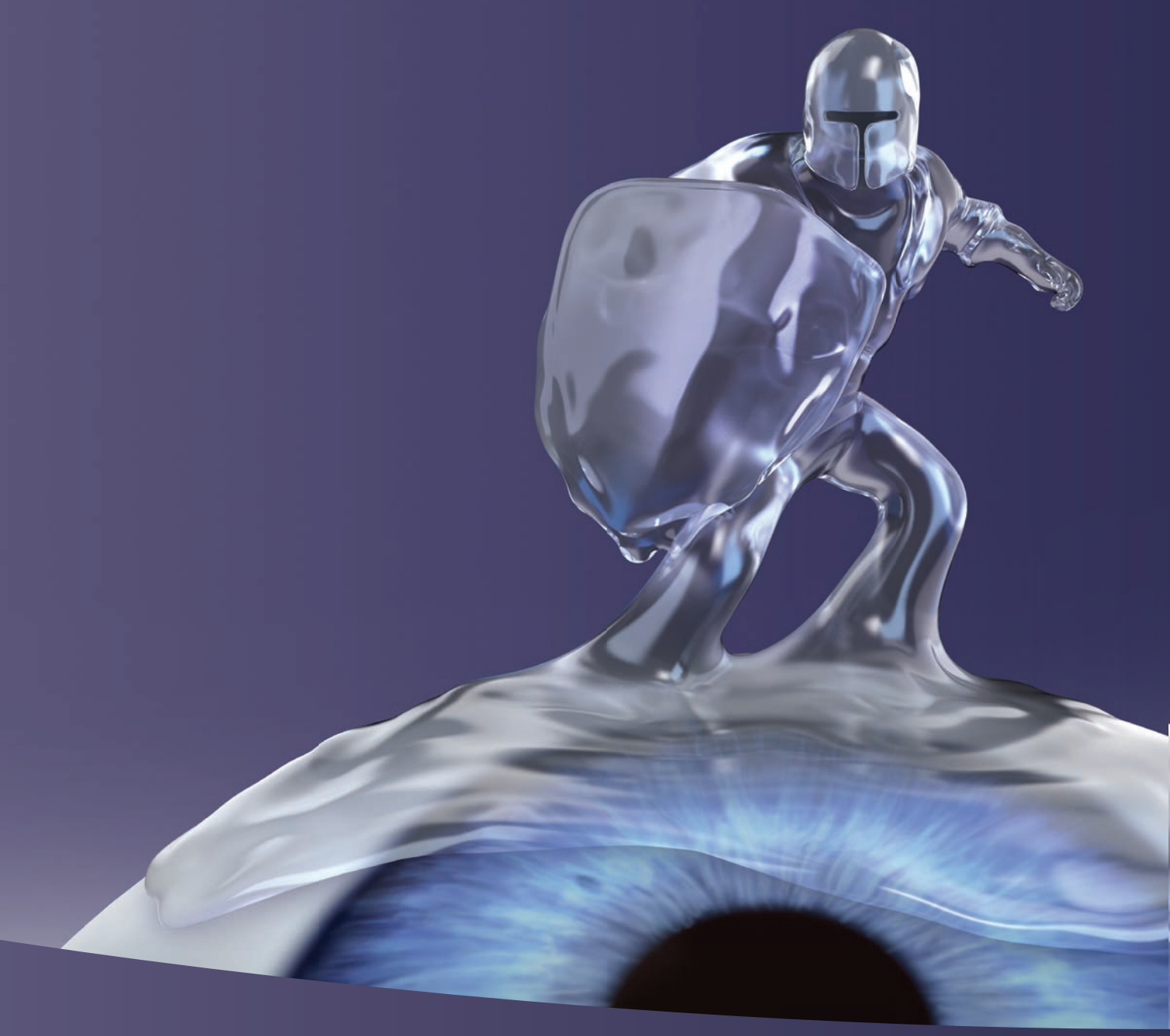
In my practice, I know that listening and discussing various options with my patients lead to good results. However, the patient experience is much more valuable than the discussion. This is why I encourage my patients to try DAILIES TOTAL1® contact lenses. These lenses incorporate the most advanced technology available today and feature a surface water content approaching 100%, providing exceptional lubricity and comfort.[1] This is made possible by Alcon's proprietary water-gradient technology, which not only allows for high water content on the surface of the lens, but also a lower water content at the core (about 33%) that permits more oxygen to pass through to the eye.[2-4] In fact, DAILIES TOTAL1® contact lenses are the highest breathable daily disposable lenses available for white, healthy-looking eyes.[3,5]

Many of my patients in northern California suffer from allergies and/or contact lens-related dryness, so I first ask about these symptoms. I proactively ask them if they are experiencing any end of day dryness, tiredness or discomfort with their current contact lenses. If so, they are more prone to discontinue wearing contact lenses entirely,[6] and I may be the last doctor to have the opportunity to offer a more comfortable lens-wearing experience. To keep patients satisfied and returning to my practice, I need to offer them innovative, high-performance products like DAILIES TOTAL1® contact lenses that have the potential to significantly improve my patients' vision and comfort—and consequently, their quality of life.

I proactively ask my patients if they are experiencing any end of day dryness, tiredness or discomfort with their current contact lenses. If so, they are more prone to discontinue wearing contact lenses entirely,[5] and I may be the last doctor to have the opportunity to offer a more comfortable lens-wearing experience. To keep patients satisfied and returning to my practice, I need to offer them innovative, high-performance products like DAILIES TOTAL1® contact lenses that have the potential to significantly improve my patients' vision and comfort—and consequently, their quality of life.

DAILIES TOTAL1® contact lenses offer the ultimate contact lens-wearing experience for many of my patients, including those who want the best technology and a high-performing option, those with end-of-day discomfort, and the silent sufferers who just require a little encouragement to talk about their visual and ocular needs. One survey from the DAILIES® Difference Program found that 70% of patients chose Alcon daily disposable lenses over their previous lenses, and of those patients 60% chose DAILIES TOTAL1® contact lenses, regardless of their income level.[7] Findings such as these, along with the recently expanded parameters of DAILIES TOTAL1®, helped make these lenses my top recommendation for patients seeking a high-performing solution to their visual needs.

References **1.** Angelini T, Nixon R, Dunn A, et al. Viscoelasticity and mesh-size at the surface of hydrogels characterized with microrheology. *Invest Ophthalmol Vis Sci*. 2013; 54: E-abstract 500. **2.** Alcon data on file, 2011. Based on laboratory measurement of unworn lenses. **3.** Based on the ratio of lens oxygen transmissibilities; Alcon data on file 2010. **4.** Thekveli S, Qui Y, Kapoor Y, et al. Structure-property relationship of delefilcon A lenses. *Cont Lens Anterior Eye*. 2012; 35(suppl 1): e14. **5.** Based on published manufacturer-provided Dk values in *Tyler's Quarterly Soft Contact Lens Parameter Guide*, June 2016. **6.** Dumbleton K, Woods CA, Jones LW, Fonn D. The impact of contemporary contact lenses on contact lens discontinuation. *Eye & Contact Lens*. 2013;39:93-99. **7.** Alcon data on file, 2014. Based on a survey of 1,339 participants through October 2014.



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

Important Safety Information

- **Slow or Delayed Healing:** All topical nonsteroidal anti-inflammatory drugs (NSAIDs), including BromSite (bromfenac ophthalmic solution) 0.075%, may slow or delay healing. Topical corticosteroids are also known to slow or delay healing. Concomitant use of topical NSAIDs and topical steroids may increase the potential for healing problems.
- **Potential for Cross-Sensitivity:** There is the potential for cross-sensitivity to acetylsalicylic acid, phenylacetic acid derivatives, and other NSAIDs, including BromSite (bromfenac ophthalmic solution) 0.075%. Therefore, caution should be used when treating individuals who have previously exhibited sensitivities to these drugs.
- **Increased Bleeding Time of Ocular Tissue:** With some NSAIDs, including BromSite (bromfenac ophthalmic solution) 0.075%, there exists the potential for increased bleeding time due to interference with platelet aggregation. There have been reports that ocularly applied NSAIDs may cause increased bleeding of ocular tissues (including hyphemas) in conjunction with ocular surgery.

  It is recommended that BromSite be used with caution in patients with known bleeding tendencies or who are receiving other medications which may prolong bleeding time.
- Use of topical NSAIDs may result in keratitis. Patients with evidence of corneal epithelial breakdown should immediately discontinue use of topical NSAIDs, including BromSite (bromfenac ophthalmic solution) 0.075%, and should be closely monitored for corneal health. Patients with complicated ocular surgeries, corneal denervation, corneal epithelial defects, diabetes mellitus, ocular

A DROP OF PREVENTION

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

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

- DuraSite increases retention time on the ocular surface and absorption of bromfenac[2-5]
  - Allows for increased aqueous humor concentrations
- Ensures complete coverage throughout the day with BID dosing[1]

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

surface diseases (e.g., dry eye syndrome), rheumatoid arthritis, or repeat ocular surgeries within a short period of time may be at increased risk for corneal adverse events which may become sight threatening. Topical NSAIDs should be used with caution in these patients. Post-marketing experience with topical NSAIDs also suggests that use more than 24 hours prior to surgery or use beyond 14 days postsurgery may increase patient risk for the occurrence and severity of corneal adverse events.

- BromSite should not be administered while wearing contact lenses. The preservative in BromSite, benzalkonium chloride, may be absorbed by soft contact lenses.
- The most commonly reported adverse reactions in 1% to 8% of patients were anterior chamber inflammation, headache, vitreous floaters, iritis, eye pain, and ocular hypertension.

You are encouraged to report negative side effects of prescription drugs to the FDA. Visit www.fda.gov/medwatch or call 1-800-FDA-1088.

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

NSAID=nonsteroidal anti-inflammatory drug.

References: 1. BromSite [package insert]. Cranbury, NJ: Sun Pharmaceutical Industries, Inc.; 2016. **2.** Hosseini K, Hutcheson J, Bowman L. Aqueous humor concentration of bromfenac 0.09% (Bromday™) compared with bromfenac in DuraSite® 0.075% (BromSite™) in cataract patients undergoing phacoemulsification after 3 days dosing. Poster presented at: ARVO Annual Meeting; May 5-9, 2013; Seattle, Washington. **3.** Bowman LM, Si E, Pang J, et al. Development of a topical polymeric mucoadhesive ocular delivery system for azithromycin. *J Ocul Pharmacol Ther.* 2009;25(2):133-139. **4.** ClinicalTrials.gov. Aqueous humor concentration of InSite Vision (ISV) 303 (bromfenac in DuraSite) to Bromday once daily (QD) prior to cataract surgery. https://clinicaltrials.gov/ct2/show/results/NCT01387464?sect=X70156&term=insite+vision&rank=1. Accessed July 18, 2016. **5.** Si EC, Bowman LM, Hosseini K. Pharmacokinetic comparisons of bromfenac in DuraSite and Xibrom. *J Ocul Pharmacol Ther.* 2011;27(1):61-66.

SUN OPHTHALMICS

BromSite™ (bromfenac ophthalmic solution) 0.075% Brief Summary

INDICATIONS AND USAGE

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

DOSAGE AND ADMINISTRATION

Recommended Dosing

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

Use with Other Topical Ophthalmic Medications

BromSite should be administered at least 5 minutes after instillation of other topical medications.

Dosage Forms and Strengths

Topical ophthalmic solution: bromfenac 0.075%.

CONTRAINDICATIONS

None

WARNINGS AND PRECAUTIONS

Slow or Delayed Healing

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

Potential for Cross-Sensitivity

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

Increased Bleeding Time of Ocular Tissue

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

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

Keratitis and Corneal Reactions

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

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

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

Contact Lens Wear

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

ADVERSE REACTIONS

Clinical Trial Experience

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

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

USE IN SPECIFIC POPULATIONS

Pregnancy

Risk Summary

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

Clinical Considerations

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

Data

Animal Data

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

Lactation

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

Pediatric Use

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

Geriatric Use

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

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis and Impairment of Fertility

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

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

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

PATIENT COUNSELING INFORMATION

Slow or Delayed Healing

Advise patients of the possibility that slow or delayed healing may occur while using NSAIDs.

Concomitant Topical Ocular Therapy

If more than one topical ophthalmic medication is being used, advise patients to administer BromSite at least 5 minutes after instillation of other topical medications.

Concomitant Use of Contact Lenses

Advise patients not to wear contact lenses during administration of BromSite. The preservative in this product, benzalkonium chloride, may be absorbed by soft contact lenses.

Sterility of Dropper Tip/Product Use

Advise patients to replace the bottle cap after use and do not touch the dropper tip to any surface as this may contaminate the contents.

Advise patients to thoroughly wash hands prior to using BromSite.

Rx Only

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SUN-OPH-BRO-017 09/2016

SUN OPHTHALMICS

Get Online or Get Lost

Even your grandma has a Facebook account these days. Isn't it time you finally became a real doctor, according to the internet? **By Montgomery Vickers, OD**

Not a day goes by that I don't have a colleague ask me to help straighten out their crappy online presence. Sometimes even a patient hates your website so much, they want my intervention. As always, I am here to take your online life off life support.

Facebook

Let's start with your photo. Is that really the face you want your patient to find? Oh, I know. Only your friends will see this one, right? Please, spare me. Some myopic teenager is already spreading that picture of you with the Annual Beer Pong Trophy from 15 years ago. Prospective patients think that's how you always look. That *is* how you always look? Nevermind.

While you are at it, peruse the photos your friends have shared with you. I totally believe you just happened to be in Denver when that anti-glaucoma medication was legalized and you and your buddy photo bombed the crowd outside Billy's Bong Emporium, but your presbyopic grandmas may feel differently.

And those political postings? No particular candidate really wants the world to burn to ashes, or believes online contact lens providers should be given the Congressional Medal of Freedom. OK, maybe one, but keep all of them off your home page!

LinkedIn

This is for businesspeople. You are an optometrist, which means you know nothing about business. But, let's pretend you actually do know about business. How is a LinkedIn relationship with a solo practitioner in Angoon, Alaska, going to help your business grow? Do a search for permafrost if you are that curious. (If you practice in Alaska, substitute Mississippi for Alaska and James Cotton for permafrost.)

Your Email Address

I have always used my real email address. Many think this is really dumb, but I cannot name three patients who have abused their right to email me in the past 37 years. They just wanted my account number so they could send me my inheritance from my recently deceased relative from Nigeria. (That money should be here any day.)

And what, exactly, is your email address? A doctor has to sound at least a little doctor-ly, so drop any references to funk, the Kardashians, beer goggles, a horse's hindquarters, spring break, Jack Black, Jack White, Jack Daniels, football teams, love, hate, apathy, atrophy, parole, pancreatitis or assorted lesions.

Website

We are redoing our website, and it's not as easy or cheap as you think. You can use a template and create one for nothing, but these types of websites are, well, stupid looking. If you are a Millennial, maybe you 'get' computers, so, you can try it, I guess. But if you are a Millennial, you're not reading this—you're too busy washing your socks at mom's.

Once you determine what to do about your website, spare everyone the generic bullet points such as, "all types of insurance accepted," "contact lens specialist" or "family eye care." Instead, show them a video of a cute cat or your baby laughing. You DO want them to call, right?

Your online presence should tell patients something important about you and should influence them to call for an appointment. If you play your cards right, they won't call just to tell you what a digital loser you seem to be. ■

A Cut Above the Rest

Distinguish yourself by mastering the evaluation and management techniques of eyelid lacerations. **By Sarah Krein, OD, Matthew Krein, OD, and Richard Mangan, OD**

Eyelid lacerations may not be the most common walk-in appointment, but it when it is in your chair, it really doesn't matter whether it is common or not. These wounds can be messy, and optometrists are tasked with evaluating, and sometimes treating, them.

A thorough understanding of the eyelid anatomy and lacrimal system is necessary when evaluating an eyelid laceration. You'll need it to accurately assess whether an immediate referral to an oculoplastic lid surgeon is called for.

Anatomy

Beginning with the exterior upper eyelid, just under the epidermis lies the orbicularis muscle, which closes the eyelid. Below the orbicularis muscle is the orbital septum. This fibrous tissue is the anatomical division separating the preseptal and postseptal portions of the eye. Behind the orbital septum is orbital fat, the levator aponeurosis and Müller's muscle.

Finally, you will see the palpebral conjunctiva lining the interior of the upper eyelid.

The lacrimal drainage apparatus is located in the medial canthus. Superior and inferior puncta on the lid margin drain through the canaliculus (approximately 8mm to 10mm) to the nasolacrimal sac. This sac lies in a fossa in the anterior portion of the medial orbital wall. The nasolacrimal sac drains via the nasolacrimal duct into the nasal cavity.

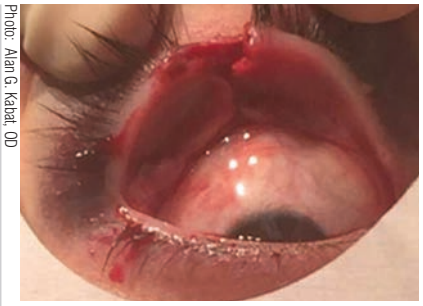


Photo: Alan G. Kabat, OD

This lid laceration was the result of a mishap during a basketball game.

Triage Care

When a patient with a recent ocular trauma arrives, gather a thorough history to determine the origin of the trauma and whether the patient lost consciousness with the incident. Next, an examination of the eye and orbit will determine the severity and rule out other traumatic complications, such as an orbital fracture, foreign bodies, corneal lacerations or abrasions, hyphema, an open globe or trauma to the retina. When a penetrating foreign body, ruptured globe, severe blunt trauma or a loss of consciousness is suspected, a CT of the brain and orbits is warranted.

Once the trauma evaluation is complete and you determine that the patient has no other sight-threatening injuries, address the lid laceration itself. Eyelid lacerations are classified into two categories—complicated and simple.

When to Refer

Complicated lid lacerations should be referred to an oculoplastic surgeon for repair, along with any patients who may require sedation.

Full-thickness eyelid lacerations pass through the entire eyelid. If your patient has a full-thickness laceration, pay special attention to the underlying bulbar conjunctiva and be extra careful to rule out a penetrating ocular foreign body.

A laceration involving the eyelid margin requires a referral because of the complexity of the anatomy at the eyelid margin. These cases need to be referred out to decrease the chances of scarring or notching of the eyelid. If orbital fat is visible, the laceration has breached the orbital septum. This is an alarming finding because it indicates the postseptal orbital space is at risk for infection.

If your patient presents with a ptosis, it is possible the levator muscle could be damaged. This can lead to chronic ptosis or permanent levator damage. When eyelid lesions are medial, it is important to determine whether or not the lacrimal system is involved. Any involvement to the structures of the lacrimal system, such as the puncta, canaliculi, nasolacrimal sac and nasolacrimal duct, should be managed by an oculoplastic lid surgeon. Lacerations with poor alignment can lead to scarring, disfigurement of the eyelid and poor eyelid function, so these are best managed with a referral as well.

When to Treat

A simple lid laceration that is small, superficial and horizontal can be managed in-office. If the laceration

is small, it may heal without anything more than a topical antibiotic ointment. Lacerations more than a quarter of the length of the eye can be treated with surgical or butterfly tape, or with a tissue adhesive.[1] In some cases, sutures may be the best option. If you determine sutures are necessary, follow these steps:

Irrigate the wound. Use a sterile saline and check the eyelid laceration for any retained foreign body.

Prepare the wound. Apply betadine in a circular motion starting at the laceration edge and working away from the laceration. Ensure adequate hemostasis, if needed light pressure or possibly a cotton tip applicator soaked in phenylephrine can help to stop the bleeding.

Apply surgical tape. This can be applied perpendicular to the laceration. The surgical or butterfly tape should be placed on one side of the wound and gently pulled across the laceration so that the edges of the wound come as close together as possible without overlapping. Multiple butterfly strips may be necessary to bring the edges of the wound together the entire length of the laceration.

Tissue adhesive. A cyanoacrylate tissue adhesive can be a particularly effective way of closing a simple eyelid laceration. Bringing the edges of the laceration in apposition, without overlap, them apply the glue along the full length of the laceration and allow it to dry. Take special care not to get any of the adhesive in the eye.[2]

Prophylactic care. A tetanus booster is recommended at minimum every 10 years. Additionally, consider broad-spectrum antibiotics (cephalexin 250mg to 500mg PO QID) based on the likelihood of wound contamination. Follow-up evaluation can be scheduled in five to seven days to ensure appropriate healing and no complications.

The OD's role is to assess the nature of the laceration to determine if the patient has a complicated laceration in need of a surgical consult or if it is a simple laceration to be managed in office. Applying basic first aid skills and infection prophylaxis will keep you a cut above the rest. ■

Drs. Krein and Krein are assistant professors at Northeastern State University in Oklahoma.

1. Handschel JG, Depprich RA, Dirksen D, et al. A prospective comparison of octl-2-cyanoacrylate and suture in standardized facial wounds. International Journal of Oral Maxillofacial Surgery. 2006;35(4):318-23.
2. Bruns TB, Worthington JM. Using Tissue Adhesive for Wound Repair: A Practical Guide to Dermabond. Am Fam Physician. 2000 Mar 1;61(5):1383-8.

Haunted By History

A patient who sought a second opinion reveals telltale findings and a history of scleral buckle. **Edited by Paul C. Ajamian, OD**

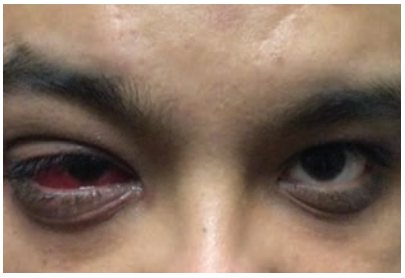
Q I saw a 24-year-old Hispanic male for a second opinion regarding a red, painful right eye, which was managed by a local MD. Four months of treatment for conjunctivitis with ophthalmic moxifloxacin, ophthalmic prednisolone acetate and, most recently, oral penicillin, has provided no relief. Could a retinal detachment (RD) in the right eye with scleral buckle repair from 2012 have anything to do with this?

A "Two key pieces of this patient's ocular history are pertinent in solving this quandary," says Chelsea Miller, OD, who is in a disease-oriented practice in Racine, Wis. First is the patient's retinal detachment with vitrectomy and scleral buckle repair, she says, and the second is the acute onset of symptoms that did not resolve with treatment over several months.

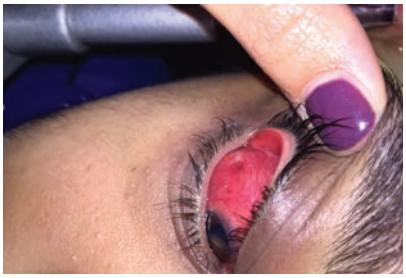
An important finding is a granulomatous lesion on the sclera at the center of the conjunctival injection. "Many times, these cases will present with a complaint of generalized pain without ocular findings, so getting a good ocular history is very important," according to Dr. Miller.[1] It's also important to look for buckle exposure in patients with a history of buckle repair, she says, because "typically, buckle infections will be associated with some degree of buckle exposure, especially in cases that present several months to years post-RD repair."[1,2]

Treating the Infection

Management is time sensitive, according to Dr. Miller, and this patient should be referred back to his retina specialist for a suspected scleral buckle infection. "Without proper care, preceptal cellulitis, endophthalmitis or panophthalmitis can occur."[2,3] Rare cases can be managed with systemic intravenous antibiotic and corticosteroid treatment; but in most cases, the buckle needs to be removed.[2]



The patient presented with significant lid swelling.



A 4mm round lesion was noted on the inferior temporal conjunctiva with a central umbilication, which had white discharge when expressed.

"Though a wide microbial spectrum causes these infections, *Staphylococcus epidermidis* and *Staphylococcus aureus* are the most common organisms," explains Dr. Miller.[1,3] Vancomycin can be an effective treatment option, and ciprofloxacin can be effective in treating other gram-positive and some gram-negative buckle bacterial infections, according to Dr. Miller.

Weighing the Risks

Removing the buckle is often necessary, but is not without risks, Dr. Miller says. "The biggest problems associated with scleral buckle removal are scleral perforation (~13%) and recurrence of a retinal detachment (~7%)," Dr. Miller says.[4] "Retinal detachment usually occurs within five days to 50 months of removal of the buckle, with 70% to 80% of these being within 90 to 180 days of buckle removal."[1,3]

Other indications for buckle removal include long-standing diplopia, ocular pain, optic nerve injury, anterior segment ischemia and macular distortion, according to Dr. Miller. "Although buckle exposure is a significant risk factor for scleral buckle infection, this is not necessarily an indication for removal if the patient is asymptomatic.[1] However, careful patient education regarding signs and symptoms of scleral buckle infection is a must with these patients." Your awareness and knowledge can save them from incorrect diagnosis and treatment. ■

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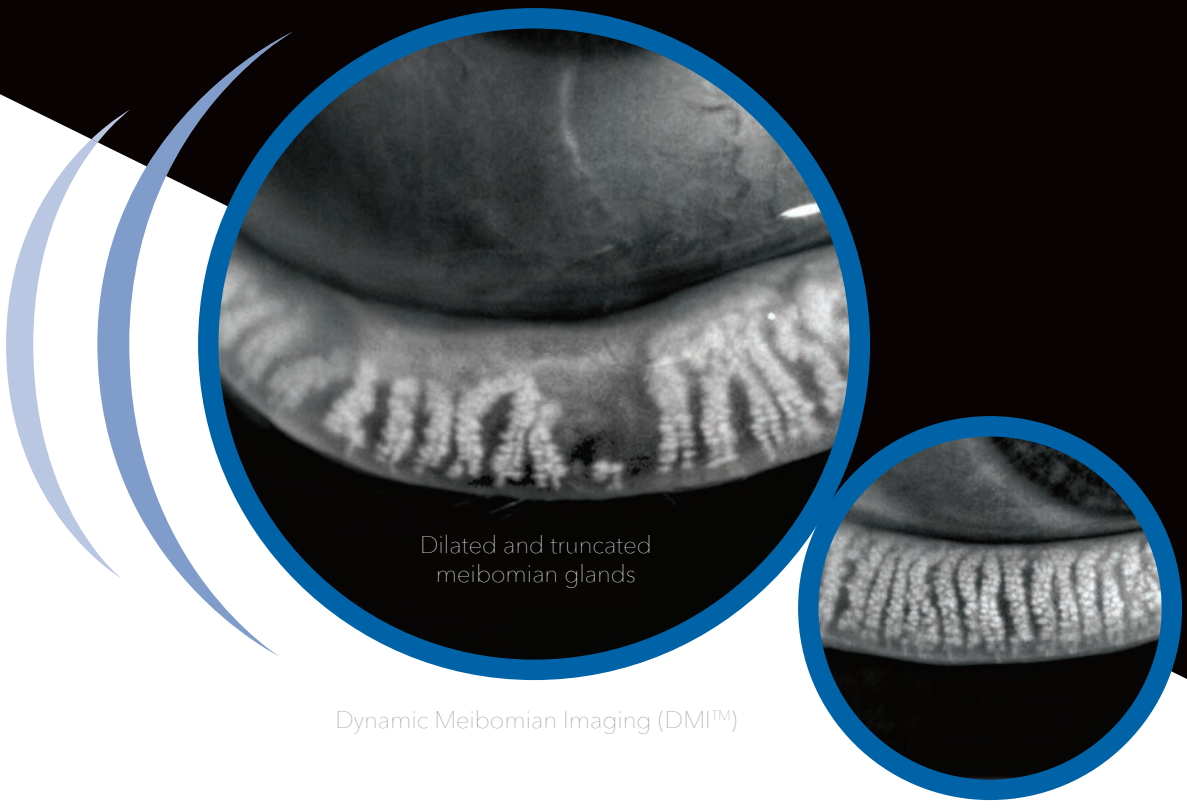

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What The MIPS is Going On?

ACA or no ACA, a reimbursement system based on quality is on its way.

By John Rumpakis, OD, MBA, Clinical Coding Editor

So here we are—post-election America. Some are rejoicing, some are rebelling and many, if not most, are confused about how our current and future politics will affect the healthcare landscape.

As we enter into a critical time with respect to the Affordable Care Act (ACA) and the potential to "repeal and replace" looming ahead, it is important to understand that the imposed changes to our payment system—moving from quantity to quality—is unrelated to the ACA.

What's What

The Patient Protection and Affordable Care Act (PPACA), commonly called the ACA or Obamacare, is a federal statute enacted on March 23, 2010. It introduced mandates, subsidies and insurance exchanges intended to reduce the costs of healthcare, increase health insurance quality and affordability and lower the uninsured rate by expanding coverage. The law requires insurers accept all applicants, cover a specific list of conditions and charge the same rates regardless of pre-existing conditions or sex.[1]

The Medicare Access and CHIP Reauthorization Act of 2015 (MACRA) is federal legislation signed into law on April 16, 2015. The law does many things, but most importantly it establishes new ways to pay physicians for caring for Medicare beneficiaries. The Merit Incentive Based Payment System (MIPS) is a critical component of MACRA, as it is transforming the way physicians in the United States are going to get paid moving forward.

So even if the change in government results in a change in the ACA, the conversion to a compensation system based upon quality rather than quantity will likely remain in place—and that means your practice needs to prepare because the changes happening in 2017 will start to affect how and how much you get paid in 2019 and beyond.

Preparation is Key

Every major medical carrier in the United States is migrating to a quality-based payment system, so this has the potential to greatly impact every practice, not just those who care for Medicare patients.

Luckily, CMS has made it very easy to comply with the MIPS reporting in 2017. Most importantly, *if you don't do anything, you will receive a -4% downward adjustment on your Medicare payments*. So what does it take to comply? Let's examine the various components.

There are four categories of physician performance contributing to a MIPS Composite Performance Score (CPS) of up to 100 points, based on these relative weights:

- Quality (formerly PQRS): 50%
- Advancing care information (ACI, formerly MU): 25%
- Clinical practice improvement activities (CPIA): 15%
- Resource use (based on claims data, calculated by CMS): 10%

You have four options for 2017 if you would like to avoid the -4% adjustment:

1. Report some data to avoid a negative payment adjustment (awaiting Final Rule). Even if you report on one measure on a single patient on one day, you can avoid the downward adjustment.
2. Submit full performance data (ACI, Quality and CPIA) for a reduced number of days, meaning your first performance period could begin later than January 1, and your payment could be smaller (awaiting Final Rule).
3. Move forward with a full year of reporting for maximum reimbursement potential.
4. Participate in an advanced alternative payment model, such as a Medicare Shared Savings Track 2 or 3 ACO. This has both positive and negative financial risk and is exempt from MIPS, but most likely is not applicable to optometry, as far as we know.

There will be much more to come in the New Year, and as we know change is inevitable, I will do my best to keep you ahead of the curve; but you have to promise me one thing: don't ignore this. In this case, ignorance is not bliss. ■

Send questions and comments to ROcodingconnection@gmail.com.

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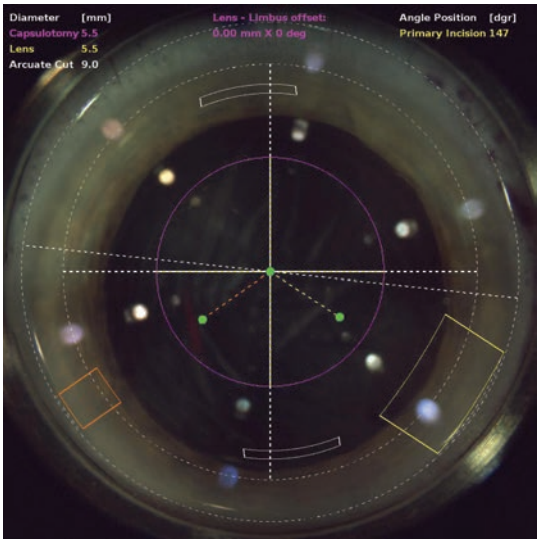
22nd Annual Surgery Report

The Evolution of Cataract Surgery

Femtosecond lasers and premium IOLs are just a few innovations clinicians are excited to discuss with cataract patients. **By Justin Schweitzer, OD**

Technology in cataract surgery continues to evolve to meet the needs of both doctors and patients. More than ever, patients expect exceptional outcomes, including less dependence on spectacles. The precision of pairing femtosecond laser-assisted cataract surgery (FLACS) with a multitude of premium intraocular lens (IOL) options, for example, is one way doctors are incorporating new technology to deliver the outcomes their patients expect. Many new options are available that are improving the cataract surgery experience for all involved, and understanding them is key to proper patient education and comanagement.

This article reviews FLACS, premium IOL technology, future IOL designs, postoperative advances and current controversies in cataract surgery to keep you up-to-date on the evolving landscape of cataract surgery.



OCT during laser cataract surgery allows the cataract surgeon to plan the location and depth of incisions.

Laser Cataract Surgery

FLACS is still a developing technology, and while research continues to evaluate its effectiveness, current findings only suggest it is noninferior relative to manual cataract surgery.[1] Femtosecond laser technology in contrast to manual methods takes certain parts of the cataract procedure out of the surgeon's hands, such as corneal incisions, the capsulotomy and softening of the lens. Potential advantages, still being debated, include a precise shape and size of the capsulotomy, custom lens fragmentation patterns, a reduction in endothelial cell loss, customized and precisely placed corneal incisions and improved refractive stability and predictability.[1] Currently there are five FLACS platforms on the market: LenSx (Alcon), LensAR (LensAR), Catalys (Abbott), Victus (Bausch + Lomb) and LDV Z8 (Ziemer).[2]

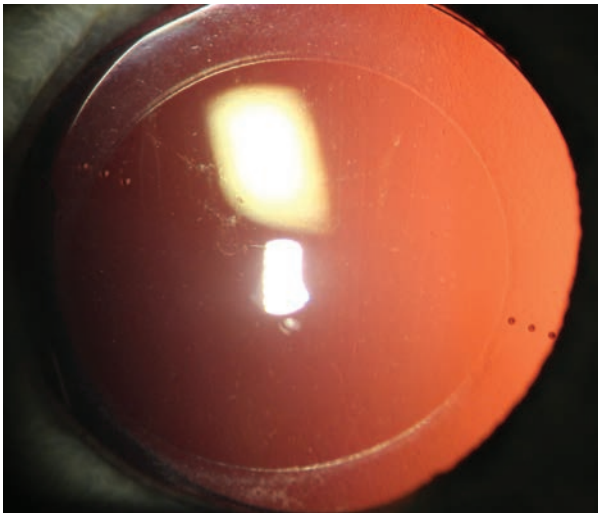
The capsulotomy is arguably the most important step in the cataract surgery procedure, as the size of the capsulotomy is key to optimizing the position and performance of an IOL. A capsulotomy that is too small has the risk of anterior capsule fibrosis and a hyperopic shift, while a too large capsulotomy can increase rate of tilt, decentration and posterior capsular opacification.[3] All of these issues can lead to a less than desirable outcome for patients, and in some cases the need for a lens exchange.[4-6] In addition, the capsulotomy plays a crucial role in predicting the effective lens position, which is important in IOL power calculations.[7] A difference of only

1mm in lens position can lead to a 1.25D change in refractive error.[3,8] With toric and multifocal IOLs, the margin of error is even smaller. A tilted, decentered or rotated IOL can cause a significant deviation from the desired refractive outcome, and can make it difficult to tolerate visual aberrations such as halo and induced coma.[9,10]

Can FLACS minimize many of these issues with a laser-incised capsulorhexis? One study found no difference in predictable lens position error when comparing traditional phacoemulsification with FLACS, but it did find higher refractive stability and IOL centration.[11]

A femtosecond laser can also be used to segment the nucleus and place pattern cuts on this structure to soften harder cataracts using less ultrasound energy. These treatments theoretically decrease complications relative to the manual technique, but debate exists. Researchers compared corneal endothelial cell loss after fluid-based vs. ultrasound phacoemulsification and concluded there was significantly lower endothelial cell loss after phaco with a fluid-based vs. ultrasound system.[12] Others have recently compared endothelial cell loss rates between phacoemulsification and FLACS and found similar results of no difference between both modalities.[13,14] FLACS has been shown to have less day-one edema, which is always something eye care providers watch for.[15]

Although FLACS has many potential advantages, many studies bring into question the benefit of the technology. A recent study that assessed the visual outcomes of 988 eye that underwent FLACS and 888 eyes that underwent standard phacoemulsification cataract surgery found that, six months postoperatively, there was no clinically meaningful visual benefit to FLACS over standard phacoemulsification.[16] Best-corrected visual acuity was slightly better with FLACS than standard phacoemulsification (20/24.5 vs. 20/26.4), and the researchers concluded that, given the refractive outcomes, FLACS is not currently cost-effective.[16]



A toric IOL with the axis markings visible.

The same study also looked at safety issues and found that in the FLACS group there were 15 anterior capsule tears compared with three in the standard phacoemulsification group and 11 posterior capsule tears vs. two in the standard phaco group.[16]

Premium IOL Options

Many new options exist, providing better visual outcomes for wide array of patients, including those with astigmatism and presbyopia:

Toric IOLs

Emmetropia can be achieved for patients with myopic or hyperopic refractive errors by selecting the appropriate spherical IOL lens power. However, nearly 20% to 30% of patients who undergo cataract surgery have corneal astigmatism of 1.25D or higher, and nearly 10% of patients have 2.00D or higher.[17] Toric IOLs offer patients the opportunity to correct corneal astigmatism at the time of cataract surgery and increase the likelihood of spectacle independence for distance vision. Multiple toric IOL models are available, including the AcrySof IQ Toric (Alcon), Tecnis Toric (Abbott) and Staar Toric IOL (Staar Surgical).

New multifocal toric IOLs take the correction of astigmatism a step further by not only correcting distance vision but also near and intermediate vision. The AcrySof IQ Restor Toric multifocal (Alcon) is an example of this type of technology, although not yet FDA approved in the United States. In one study, patients with 1.00D or higher astigmatism with a multifocal IOL showed a compromise to both distance and near visual acuities.[18] Such results highlight the importance of optimal astigmatism correction in patients with low amounts of corneal toricity.

Although minimal, complications are consistent with those found with monofocal IOLs, except alignment is significantly more crucial with a toric IOL. The efficacy of toric IOLs is dependent on the position of the IOL relative to the intended alignment axis. Residual astigmatism is induced for every degree of misalignment from the intended alignment axis. For every degree that the lens is off, the patient loses 3.3% of astigmatism correction. If a toric lens is off by

Controversies in Cataract Surgery

In addition to the lack of consensus on the proper role of FLACS—some surgeons perform it routinely, some reserve it only for more challenging cases and a sizable contingent still favor manual techniques—other debates have captured the attention of both ODs and MDs.

• ***Dropless Cataract Surgery***. One of the biggest concerns for patients and eye care providers is postoperative eye drops. Compliance issues remain a major concern, and patients' lack of adherence to their medication regimens can lead to complications. One study found that 50% of patients took less than half, and 20% took less than a quarter, of their prescribed medications after cataract surgery.[24] With recent advances in intracameral or intravitreal injections at the end of cataract surgery, there is hope for increased compliance while decreasing the number and cost of postoperative drops and the incidence of complications.

Intracameral injections after cataract surgery are well accepted in Europe, with nearly 74% of European ophthalmologists adopting the use.[25] The injection consists of a broad-spectrum antibiotic, typically moxifloxacin, and a steroid such as triamcinolone. The medication is injected directly behind the IOL, through the zonules and into the vitreous cavity.

Injections minimize the most important risk factor in cataract surgery, endophthalmitis. A large study compared intracameral antibiotics vs. topical antibiotics after cataract surgery and found with the addition of intracameral antibiotics there was a 22-fold decrease in endophthalmitis rates.[26] The study concluded at five years, and the participating surgeons' use of intracameral antibiotics climbed from 11% to 100%.[26]

A second benefit is cost-effectiveness, as the patient does not have to purchase both a topical steroid and topical antibiotic. Finally, it minimizes the instillation of topical drops, which is particularly helpful for patients with physical limitations that make instilling drops difficult.

Common concerns regarding the use of an intraoperative injection include the risk of intraocular pressure (IOP) spikes and the efficacy of controlling surgically-induced inflammation and cystoid macular edema. A study of 1,575 eyes that received an injection of antibiotic and steroid found that the mean IOP was 21.8mm Hg on the day of surgery and 14.5mm Hg at three weeks post-procedure.[5] No eyes required ocular hypotensive treatment due to a steroid response, the rate of CME was 2%, inflammation rate was 2.5% and, in those eyes, the use of topical steroids was required to reduce the inflammation.[26]

• ***Bilateral Cataract Surgery***. Although most surgeons delay surgeries between eyes to avoid potentially blinding a patient with a bilateral complication, there is a growing number of surgeons who believe simultaneous bilateral cataract surgery is better for patients because of rapid patient rehabilitation and the economy of a one-stop approach.

Studies show various economic and quality of life arguments that favor bilateral cataract surgery.[27] This procedure is not well accepted by health insurers or providers despite these studies, mainly due to safety concerns.[28] A large multicenter randomized clinical trial reported a very low rate of intraoperative and postoperative complications, including a low rate of CME.[29]

The counterarguments revolve around safety. Concerns over endophthalmitis and toxic anterior segment syndrome can't be ignored. A second counterargument is the ability to consider the refractive outcome. By delaying surgeries, the surgeon can consider the refractive outcome in the first eye and modify the IOL power in the second eye.[30]

The debate will continue, but it is hard to argue with the compelling evidence of economic and quality of life advantages for patients.

30 degrees, it has no cylindrical effect and acts similar to a spherical IOL. Misalignment will affect a higher-powered toric IOL more significantly than a lower-powered IOL. For example, if an AcrySof T3 (Alcon) that corrects 1.03D of astigmatism is misaligned by 15 degrees, there will be a loss of 0.51D or roughly 50% of its astigmatism correction. In contrast, if an AcrySof T9 (Alcon) that corrects 4.11D of astigmatism is misaligned by 15 degrees, there will still be a 50% loss of astigmatism correction, but the magnitude will be much higher at 2.05D. A misalignment of more than 10 degrees is generally regarded as an indication for surgical repositioning.[19] The two main factors accounting for misalignment are inaccurate alignment of the IOL during surgery and postoperative rotation of the IOL.[19]

Determining how to correct the residual astigmatism can be challenging. Generally, it can be corrected by rotating the IOL, laser vision correction, IOL exchange or limbal relaxing incisions. However, cross cylinder effects complicate matters, and in these situations it can be helpful to use an online toric IOL calculator (e.g., astigmatismfix.com) to help decide if lens rotation is necessary.

Multifocal IOLs

All patients older than 40 know presbyopia can be a frustrating process, and those who want to ditch their spectacles for both distance and near now have many multifocal IOL options from which to choose. Multifocal IOLs allow multiple focal distances independent of ciliary body function and capsular mechanics by using diffractive optics that split light between distance, intermediate and near. Once placed in the capsular bag, the function of the multifocal lens will not change over time.

Current multifocal IOL technology (e.g., AcrySof Restor IQ, Alcon; Tecnis Multifocal, Abbott) is available in high-add and low-add configurations. Practitioners can provide patients with a large range of vision and decreased dependence on readers by using a low add in

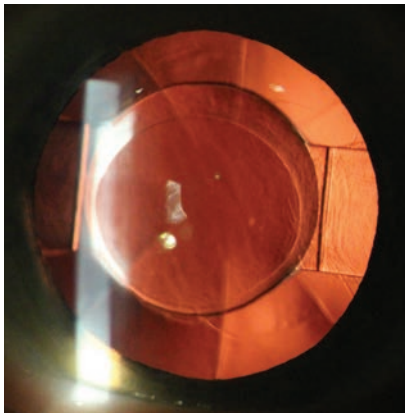
one eye paired with a high add in the contralateral eye.

The challenge of multifocality remains with preserving optical quality for patients. An overall healthy eye is required for multifocal IOLs to work best. Macular function and tear film quality must be examined and treated, if necessary, to achieve optimal outcomes with multifocal IOLs. In situations where contrast sensitivity is reduced permanently, as can be the case with patients with moderate to severe glaucoma, multifocal IOLs should be used with caution.

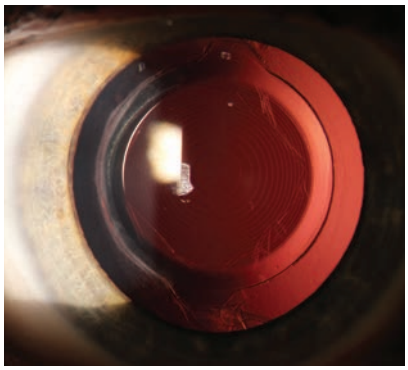
Another major challenge for multifocal technology is reducing or eliminating phenomena such as glare and halos. Patient selection can be challenging in these situations, as it is more of an art than a science. Practitioners should remain cautious with demanding patients, especially those expecting perfect visual performance in low light conditions. Fully counseling patients on the chances of needing part-time spectacle correction for night-time vision or reading in dim illumination is imperative. If the patient is unwilling to accept this as a reality, offering monfocal options may be the best way to proceed.

Clinicians should also consider lifestyle and visual expectations when deciding if a patient is suitable for a multifocal IOL. This involves a detailed history regarding the patient's work and leisure activities, the amount of time spent doing each and a ranking of how important each is to the patient.

Eye care providers must also understand the functional benefits and limitations of the IOL. Specific multifocal IOLs have certain strengths and weaknesses concerning their ability to function adequately at certain distances, with residual amounts of corneal



With this Crystalens IOL, note the modified plate haptic with the hinges to allow backward and forward movement.



Note the concentric rings common with multifocal IOLs.

astigmatism or when there is IOL decentration.[20]

Accommodative IOLs

These lenses are intriguing because the technology addresses presbyopia—and astigmatism, in certain IOLs—by attempting to mimic the eye's natural method of focusing. Clinical studies indicate that restoration of accommodation may be achieved, to some extent, with axial movement of the lens optic within the capsular bag.[21,22] The IOL needs to have forward-backward axial movement or flexibility in its shape or thickness to effect change in focal point from distance to near vision. Examples of these types of IOLs include the Crystalens AO (Bausch + Lomb) and the Trulign Toric IOL (Bausch + Lomb), which merges presbyopia and astigmatism correction.

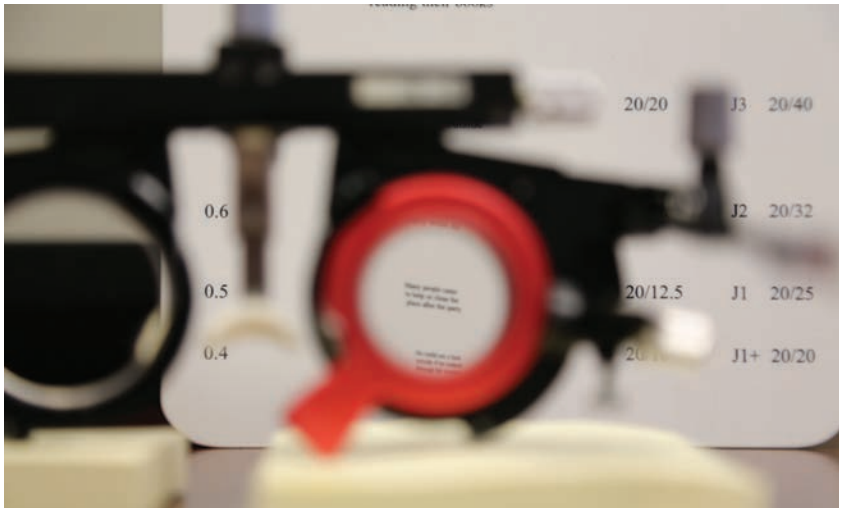
One major advantage of accommodative IOLs is a greatly decreased risk of visual aberrations such as halos or contrast sensitivity loss compared with multifocal IOLs because the brain is not forced to choose between different images, as is the case with multifocal lenses.

Challenges do exist, the main one being achieving more near power. So far, no accommodative device can perfectly duplicate the eye's accommodative mechanism, so patient education on near point ability with these lenses is imperative. Studies also show accommodative lenses are associated with more posterior capsular opacification, which can induce asymmetric vaulting, leading to lens tilt and ultimately a decrease in visual acuity.[22]

Extended Depth of Focus (EDOF) Lenses

Another emerging technology, EDOF IOLs (e.g., Tecnis Symfony, Abbott; IC-8, AcuFocus; WIOL-CF, Medicem), use new designs to improve the range of vision without splitting light rays. Much like accommodative and multifocal IOLs, this technology is designed with the presbyopic patient in mind. Instead of the single focal point of accommodative lenses or two distinct foci of multifocal lenses, the EDOF IOL creates one elongated focal point, intended to smooth out the dips in the defocus curve common with other presbyopia-correcting IOLs. Since EDOF IOLs don't split light, patients experience less glare and halos, but the intensity of near vision does not match that provided by multifocal lenses.

According to one study conducted on the Symfony, 99.6% of



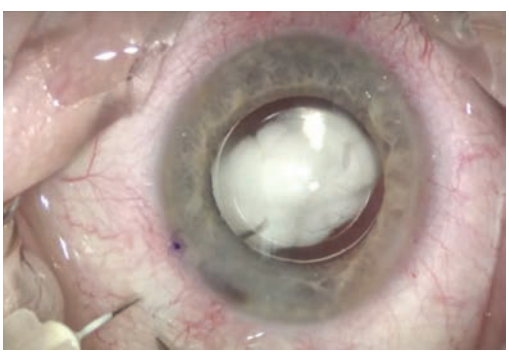
A misalignment of 15 degrees with a toric IOL is simulated in this image. With 15 degrees of misalignment, the IOL loses 50% of its astigmatic correcting power.

patients had binocular distance UCVA of 20/40 or better at distance, 96.6% had binocular intermediate UCVA of 20/25 or better and 95.9% of patients had binocular near UCVA of 20/40 or better.[20] In this study, 97% of 31 subjects indicated they would elect to have the lens implanted again.[23] While the Symfony IOL is approved for use in the US and includes a toric design—the Tecnis Symfony Toric IOL (Abbott)—the IC-8 and WIOL-CF have yet to receive approval.

The Future

The future is bright for IOL technology, particularly for two categories: devices with a modular multicomponent category and those where the optic is adjusted postoperatively with a secondary device. Both of these allow customized prescriptions for the patient at the time of the primary surgery or at a time in the postoperative period.

Multicomponent IOLs have a base unit—that serves as a holding device to secure the optic—which is placed within the capsular bag similar to a standard cataract procedure. The design is intended to allow a safe and easy exchange of the optic component to reduce residual postoperative refractive error at the time of surgery or postoperatively, potentially minimizing the need for secondary touchups and offering adjustability postoperatively.



A transzonluar injection of TriMoxi after cataract surgery.

IOLs that provide adjustments postoperatively with a secondary device have many advantages, such as the decreased risk of infection because the surgeon does not have to re-enter the eye to adjust or exchange the implant. The design also eliminates secondary adjustments using a corneal refractive procedure, leaving the anatomy of the cornea untouched and eliminating the recovery process that comes with those procedures.

The Light Adjustable Lens (Calhoun Vision) is one example of this type of IOL technology, which is currently going through FDA clinical trials in the United States. A patient undergoes traditional cataract surgery and has the monofocal IOL implanted into the capsular bag. The IOL's silicone macromers, which are distributed evenly throughout the lens, are photosensitive to near-ultraviolet wavelength of energy. If the patient has residual refractive error in the postoperative period, a secondary device can apply the near-ultraviolet wavelength of energy to the IOL to change the distribution of the macromers and correct the refractive power. A myopic adjustment occurs when the pattern puts more energy in the periphery of the lens, and vice versa for a hyperopic adjustment. Cylindrical adjustments are also possible. The surgeon can adjust the IOL multiple times until the lens power is accurate. Once this has been achieved and the patient's refraction is stable, a final irradiation step locks in the power change and IOL power.

Patients will continue to present to ODs asking about what new technologies are available in cataract surgery and the benefits they may provide. It is our role as eye care providers to educate them on surgical options such as FLACS and the many new IOL options, including the current research that suggests they have safe, efficient and reproducible track records thus far. We must guide patients with appropriate information to help them make an informed decision on what correction will suit them best. ■

Dr. Schweitzer is a cornea, glaucoma, cataract and refractive surgery specialist at Vance Thompson Vision in Sioux Falls, SD.

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22nd Annual Surgery Report

Comanaging Cataract Surgery Complications

Know how to differentiate between conditions that may arise after extraction.

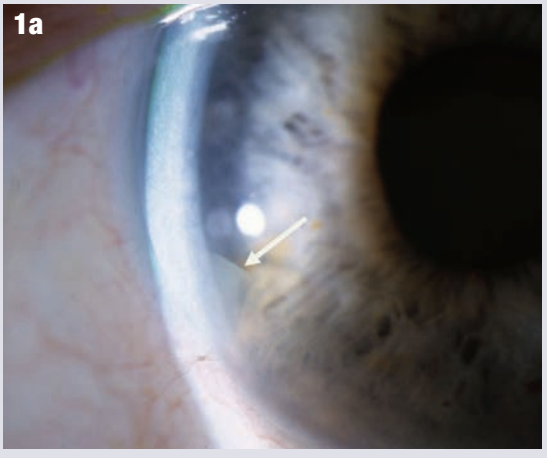
By Marta Fabrykowski, OD. Cases by Matthew Garston, OD

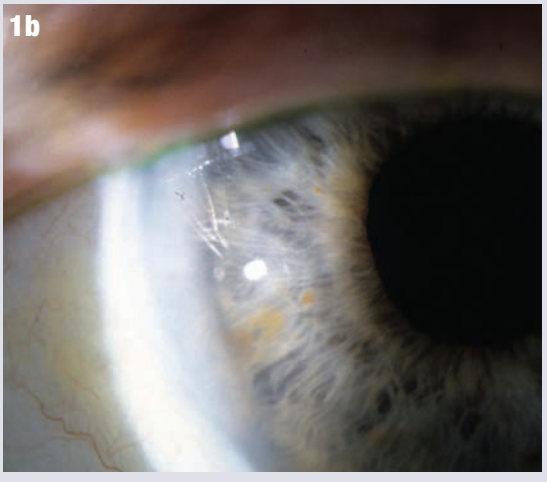
Cataract surgery has become one of the most frequently performed and most successful surgical procedures, with overwhelmingly positive outcomes.[1] While the complication rate after cataract surgery is relatively low, the sheer number of procedures coupled with a large group of comanaging optometrists merits a review of potential postoperative complications.[1] Most postoperative problems may be adequately treated by the comanaging OD, though some may require referral back to the surgeon. Postoperative complications are best characterized by the timeframe when they occur and by frequency.

Early Common Complications

Though the official postoperative period begins immediately after surgery concludes, we will consider 'early' complications as roughly spanning the first four weeks. Generally the first postoperative check occurs within 24 hours of surgery. At that appointment, vision will often be described as qualitatively blurry. During this early period, topical steroids and antibiotics are routinely started four times a day,

Case 1. Retained Nuclear Fragment





Although this 77-year-old male's uncomplicated bilateral cataract surgery was performed in 2007, the fragment in his right eye wasn't seen until almost two years later. A nuclear fragment in the anterior chamber (*Figure 1a*) after cataract surgery is a rare, but well-reported event. *Figure 1b* shows the eye after it was removed. Iritis may occur years after surgery. If it occurs in a patient who has had phacoemulsification, that should prompt a careful examination for nucleus fragments in the anterior chamber. Corneal edema may be associated, but was not in this case.

with or without a non-steroidal anti-inflammatory drug (NSAID)—dosed as the examining doctor deems necessary. The frequency and timing of subsequent postoperative checks depends on how the patient heals, essentially on these aspects:

Cell/flare. After surgery, at least a small amount of cell and flare will always be present in the anterior chamber. This decreases the clarity of the initial refractive outcome. This is not truly considered a complication of cataract surgery, but rather an expected result. Its sequelae often include complaints of blurry vision or light sensitivity. Topical steroids are prescribed and tailored for this.

Corneal edema. Virtually all patients will present with some level of corneal edema. This is commonly localized around the corneal incisions, but certainly can present diffusely anywhere on the cornea. This can range from relatively superficial epithelial swelling to full-thickness edema with Descemet's folds, especially when the surgery is longer in duration or the cataract is more dense. While most postoperative corneal edema will resolve over time, most physicians prefer to treat corneal edema with topical steroids, customizing the dosage to the level of corneal edema present. This can be done by either raising the frequency of drop administration or by changing to a more potent steroid (i.e., prednisolone to difluprednate). Raised intraocular pressure (IOP) can also manifest as diffuse microcystic epithelial edema.[2] One reason an NSAID is often given is for discomfort related to the incisions or corneal damage.

Elevated IOP. Elevated intraocular pressure is not an uncommon problem following cataract surgery. It is the most frequent postoperative complication that demands treatment.[3] Statistics vary, but as many as 18% to 45% of patients experience a pressure greater than 28mm Hg initially, but one that often returns to their baseline by 24 hours postoperatively either with or without treatment.[3] When IOP approaches 40mm Hg, sometimes the paracentesis wound may need to be 'burped', which necessitates some previous training or a trip back to the surgeon. When the corneal edema coincides with some level of raised IOP, a delicate balance of ocular hypotensive and steroid may be required. For example, a highly edematous, 3+ to 4+ fold cornea with an IOP of 35mm Hg may demand sustained prednisolone dosage with an added beta blocker or carbonic anhydrase inhibitor, especially if the nerve is suspicious for glaucoma. Frequent pressure and cornea checks may be needed to adjust the drops to the proper dosage. Causes for elevated IOP within 24 hours of surgery generally include retained viscoelastic or pre-existing glaucoma problems.

It may be challenging to determine the correct balance in the treatment drop regimen in patients who present with high IOP due to steroid response, though this condition is usually elucidated at the one-week follow up after the steroid has been used for some time, not the 24-hour check-up.[4] Those patients with steroid response, assuming normalization of the cornea and

Case 2. Positioning Problems

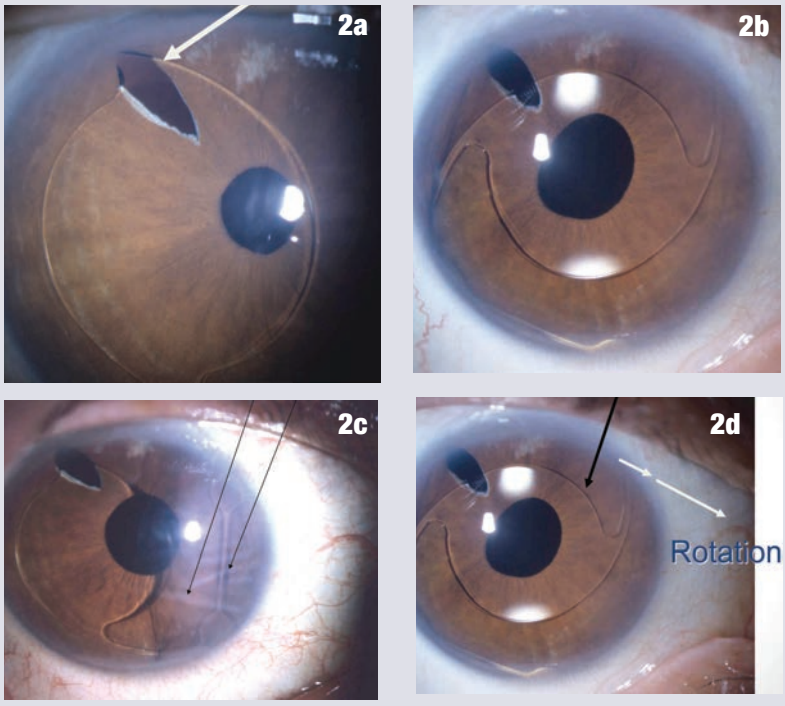
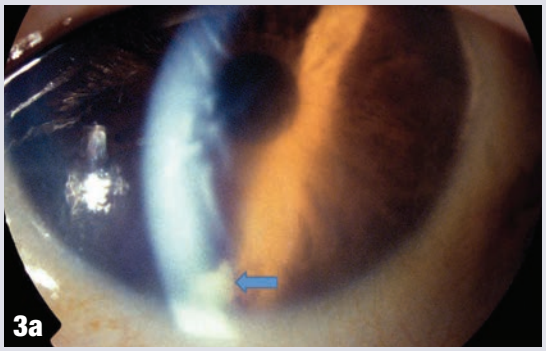
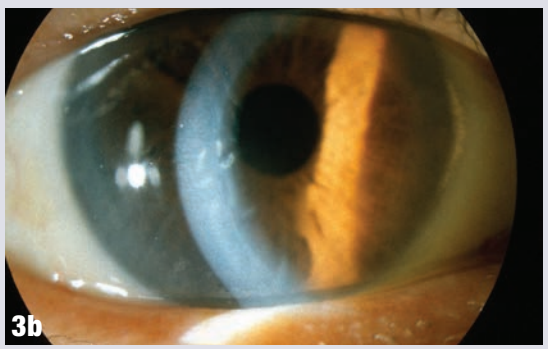


Figure 2a shows a patient with an IOL out of position and in the peripheral iridectomy. *Figure 2b* shows the same eye after the IOL was restored to its proper position. *Figure 2c* shows how this IOL, out of position, caused corneal folds. The arrows in *Figure 2d* show how the lens was rotated out of the iridectomy.

Case 3. Retained Cortical Fragment





This 75-year-old male patient presented one week after having a cataract removed from his right eye, with a complaint of blurry vision in that eye only. The blue arrow in *Figure 3a* shows a cortical fragment in the anterior segment. After he returned to the surgeon to have the fragment removed, his vision cleared up. *Figure 3b* shows the patient's right eye following the removal.

anterior chamber, may need to taper the steroid quicker or be switched to a less potent steroid (e.g., from difluprednate 0.05% to prednisolone acetate 1% or loteprednol etabonate 0.5%), or with the addition of a topical hypotensive, such as a beta blocker or carbonic anhydrase inhibitor or alpha-2 agonist. If the IOP is greater than 30mm Hg, another 24-hour pressure check may be required. A modified paracentesis procedure or a burping of the wound should be considered if the initial postoperative IOP is 40mm HG or greater. If not high, the next check may take place a few days later—at which point the steroid and hypotensive regimen can be revised.[5]

Early Rare Complications

Iris prolapse. A truly rare early postoperative complication, iris prolapse most commonly from inadequate wound closure, accidental trauma or raised IOP.[4] If the iris tissue is seen less than 48 hours post surgery, its tissue can be repositioned by the cataract surgeon.[4] If the iris prolapse is of longer duration, the prolapsed section may need to be excised, a procedure left for the operating room.[6]

Wound leaks. These are also rare, and can present in a number of different ways. Signs include poor vision, IOP less than 8mm Hg, complaints of epiphora and shallow anterior chamber. The easiest way to clinically identify a wound leak is with the instillation of fluorescein dye. Close inspection of the fluorescein-surrounded wound under cobalt blue filter light will show a dark band of negatively dyed fluid, or aqueous, running from the wound. Management of the leak depends on the severity, cause and timing of the leak—usually small wound leaks resolve within 24 hours to 48 hours, with the only intervention being decreasing the steroid dosage or strength.[5] Moderate leaks may require the use of a bandage contact lens, or an added cycloplegic or aqueous inhibitor to tamponade the flow. In either mild or moderate case, diligent follow up every 24 hours is recommended. If the leak is significant and the chamber appears shallow with a low IOP, the patient should be sent back to the surgeon for repair.[7]

Toxic anterior segment syndrome (TASS). Thankfully, this is a very rare early occurrence usually appearing 12 hours to 72 hours after cataract surgery.[2] Anterior segment inflammation in this case is quite severe, often with hypopyon, fibrinous uveitis and significant corneal swelling due to endothelial cell damage.[2] It is sterile, minimally painful and, as the name implies, does not present with vitreous involvement—it is localized to the anterior segment. When it does occur, it's almost always after uneventful cataract or anterior segment surgery.[8] The culprits can involve contaminated surgical equipment, inadequately sterile drops or solutions introduced into the eye during the procedure, or other foreign substances such as talc from surgical gloves.[9]

Management of TASS varies in the literature but generally requires high doses of topical steroids every hour; some studies advocate frequent NSAIDs or even oral steroids.[2,10] It is vital to differentiate TASS from infectious endophthalmitis, also rare, and whose incidence is from 0.05% to 0.4% in different studies.[2] From an anterior perspective, the two appear fairly similar.

Early acute endophthalmitis. Though TASS appears roughly 24

to 72 hours after surgery, endophthalmitis occurs three to seven days post surgery.[2] Both will present with blurred vision, corneal edema and anterior chamber reaction and likely hypopyon. In endophthalmitis, the corneal edema will be greater, accompanied by larger hypopyon—though depending on the duration and severity, the anterior segment findings may be confounding.[2] The main difference is the presentation of the vitreous, where endophthalmitis will present with some level of vitritis, but TASS will have a clear vitreous. In terms of treatment, unlike TASS, endophthalmitis will not respond to steroids and will require referral to the surgeon or retinal specialist for either intravitreal broad spectrum antibiotics with likely vitreous sample taken, or pars plana vitrectomy. Systemic or periocular antibiotics may be used as well.[2,11]

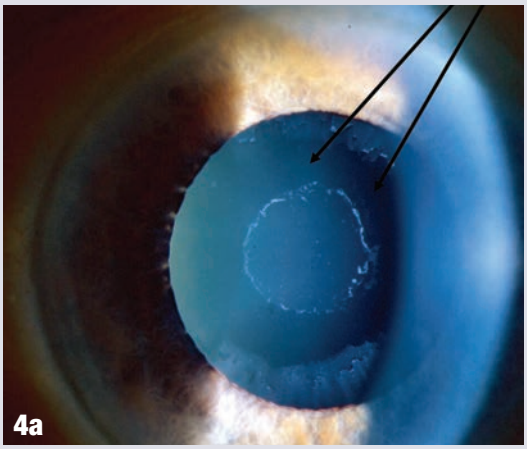
Retained lens fragments. Retained lens fragments can appear any time in the postoperative period, from day one through months to even years later. They can present anywhere in the eye—anteriorly if the capsule is intact, posteriorly if the capsule has ruptured. The fragments are not always easy to see, as they may be very small and may hide within the angle or behind the iris and cause symptoms such as blurred vision, photophobia, tearing and redness.

If the fragment is in the anterior chamber, the patient may present with symptoms of blur with a sectorally edematous cornea and an anterior chamber reaction. If possible, gonioscopy should be completed to visualize the angle and trabecular meshwork in search of the fragment.

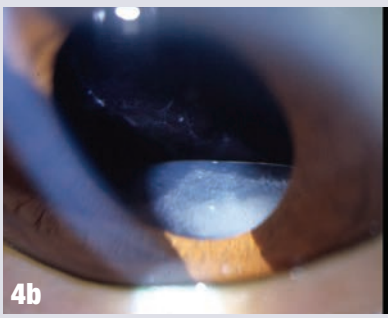
If the retained material is cortical, it may appear light yellow to white in color and translucent. If the fragment is nuclear, it may appear darker yellow/brown and more opaque. Small amounts of cortical material often can be followed closely with topical anti-inflammatory drops without surgical intervention.[12] When the fragment is nuclear, anterior to the lens and there is a considerable amount of inflammation, the particle should be removed via surgical intervention by the original operating ophthalmologist.

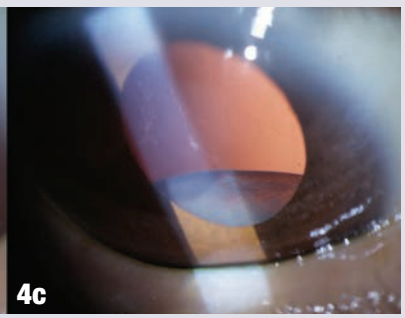
A large posterior fragment may need removal with pars plana vitrectomy. If the patient remains to be followed by the OD, they should be followed closely for the development of cystoid macular edema (CME), retinal detachment (RD), ocular hypertension or corneal decompensation.[12]

Case 4. Pseudoexfoliation



The arrows in *Figure 4a* shows where deposits have been rubbed off the lens by the iris. *Figures 4b and 4c* show a case where pseudoexfoliation caused complete IOL dislocation seven years after cataract surgery.

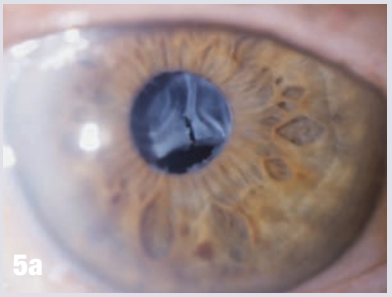




Late Common Complications

Posterior capsular opacification (PCO). Sometimes associated with Elschnig's pearls, this is the most frequent late complication from cataract surgery.[13] Incidence of PCO varies in the literature from 14% to 60%, and may be more frequent in younger patients, myopes, those with diabetes, those with previous ocular surgeries and those with greater amounts of posterior subcapsular or cortical cataracts.[13]

The severity of PCO is evaluated in many ways using simple observation under the slit lamp light, either under direct slit beam or via retroillumination. The most important grading scale is subjective—determining if the patient's best corrected vision has become problematic. One grading system uses a 0 to 3 scale,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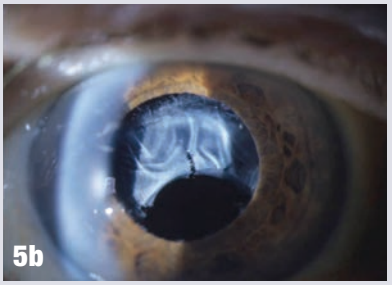
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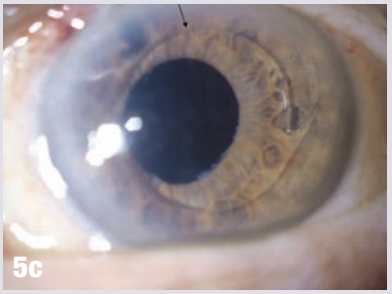
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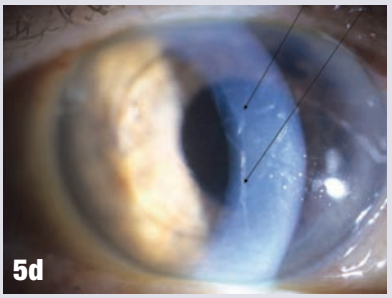
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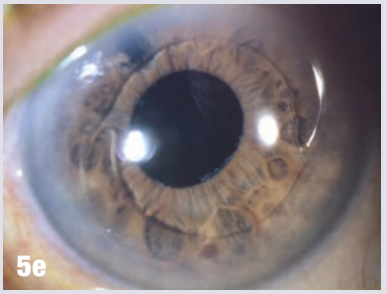
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Case 5. IOL Dislocation and Corneal Trauma

This 84-year-old female patient underwent cataract extraction four years prior and a YAG capsulotomy one and a half years ago in her left eye. She had a visual acuity of 20/30 OS, but reported seeing a "film" over her eye (*Figure 5a*). The dilated view (*Figure 5b*) reveals the problem: her superior zonules ruptured and her posterior chamber IOL and capsular bag came loose. You can better see the opaque anterior capsule in this view.

The patient had two surgical options. She could have the dislocated posterior chamber IOL sewn back into to place, risking erosion of the sutures, or she could have an anterior chamber IOL installed. The second option would put her at risk for glaucoma and iritis, but has a shorter recovery time. She was fitted for an anterior chamber IOL. *Figure 5c* shows her dilated eye two weeks after the IOL was placed. Unfortunately, she also developed endothelial striae as a result of surgical trauma (*Figure 5d*), which was later resolved after a short course of steroid drops (*Figures 5e*).

where 0=absent, 3=dense white.[14] A more commonly used method is a four point scale, where 1=no or slight PCO without reduced red reflex and 4=severe fibrosis covering the visual axis.[14]

The mechanism of PCO development is usually secondary to a proliferation and migration, or growth of residual lens epithelial cells left on the anterior capsule after surgery.[13] Though these residual cells themselves are not dangerous to the implant or the eye, they can scatter light and decrease the patient's best corrected vision. They often cause glare and hamper the OD's view of the fundus.[14] Treatment for PCO, should it become visually significant, is Nd:YAG laser posterior capsulotomy, commonly referred to as a YAG. Though the laser procedure is quick and painless for the patient, it is not without risk. Retinal complication concerns such as lattice, holes and weak spots should be addressed prior to proceeding with laser due to risk of retinal detachment.[15]

Cystoid macular edema. Pseudophakic CME, or Irvine-Gass syndrome, is less common in recent years and it can vary from 1% to 30%, and generally peaking at six weeks postoperatively.[16-18] Patients may present with best corrected vision that has worsened from previous visits. Dilated exam and OCT can confirm parafoveal cystic spaces.[18] On fluorescein angiogram, the macula may show a characteristic petalloid pattern of leakage.[16] Factors that are predictive in its development are previously compromised maculae—via diabetes, epiretinal membrane, other vascular event or preoperative use of prostaglandin analogs—as well as ruptured posterior capsule.[18]

As CME is thought to be an inflammatory process, the most common treatment is a regimen of topical nonsteroidals with or without the addition of topical steroids.[18] The mean resolution of patients treated topically with an NSAID is approximately two months, though it can take longer.[18] Rarely, CME requires intravitreal injection with corticosteroids or pars plana vitrectomy.[18]

Late Rare Complications

IOL dislocations. With improvements in IOL design and surgical technique, the incidence of subluxation has become more rare, estimated from 0.2% to 3%.[19,20] Previously, IOL dislocations happened more often in the early postoperative period, but with the advent of intraoperative adjunctive

devices such as capsular tension rings and iris hooks, progressive zonular dehiscence, and late 'in-the-bag' dislocation incidents have been increasingly reported, its risk increasing with the presence of pseudoexfoliation.[19,20] The patient may or may not be symptomatic to the dislocation—most often with a sudden, painless loss of some level of vision.

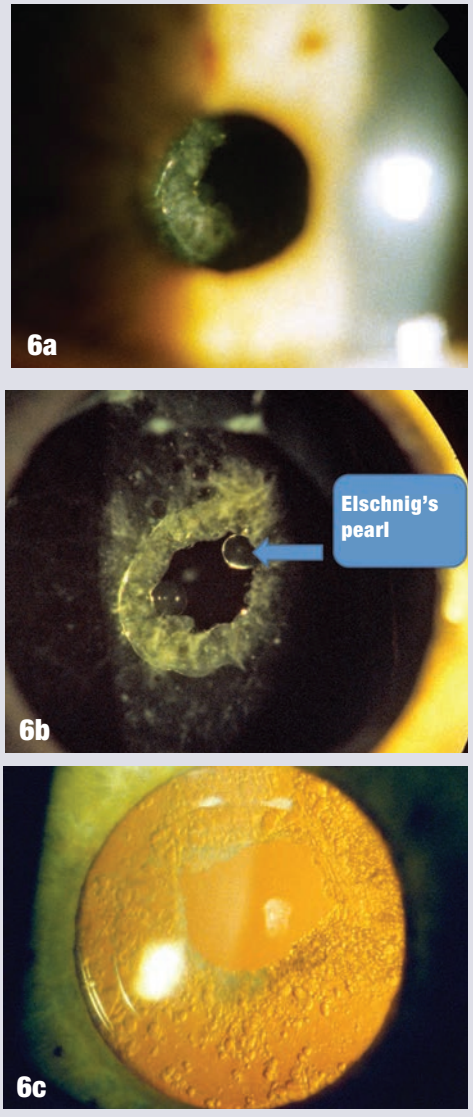
If the complaint of blur is mild, and the lens is not posing a threat to the eye, many patients will use refractive means to treat—via contact lens or spectacle prescription. Surgical treatment for IOL dislocation, if intervention is warranted and wanted by the patient, is multifaceted. Depending on the type of lens originally implanted, sometimes it can be intraoperatively sutured to the iris or sclera. If the lens must be explanted, a sulcus, anterior chamber IOL, or sutured IOL may be implanted.[19,20]

Retinal detachments, most commonly in younger patients without a posterior vitreous detachment, are an important late comorbidity associated with dislocated IOLs. Up to 6% of these patients will present with a concurrent RD, though many of those involved trauma.[21]

Capsular contraction syndrome. Anterior capsular phimosis (ACP), also known as capsular contraction syndrome or anterior capsule contraction syndrome, is quite uncommon with only a few cases reported in the literature. One study of 801 eyes had an incidence of 0.004%.[22] ACP is due to remaining lens epithelial cells that lead to fibrous metaplasia and contraction, causing a reduction of the capsular opening.[23] Vision can be impaired not only because of the fibrous nature of the opacification, but also due to the potential tilting, decentration or folding forces placed on the lens by the contracting capsule.[23] The treatment for ACP, should the lens warpage merit intervention, is YAG laser 'relaxing incisions' at the edge of the capsulorhexis.[22]

Chronic corneal edema. Pseudophakic bullous keratopathy (PBK), or the development of irreversible corneal edema following cataract surgery, has decreased dramatically since the 1980s.[24] The current incidence, as reported by the FDA, is 0.1%, though it was once as high as 1.5%.[24] The risk of its occurrence depends on a multitude of factors; the endothelial cell integrity and presence of Fuchs' greatly increases the chance. Lenses other than a posterior chamber IOL, such as

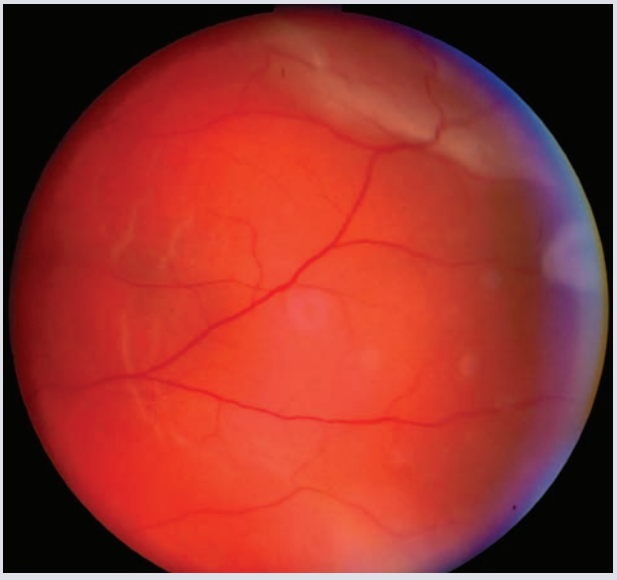
Case 6. Elschnig's Pearls After YAG Capsulotomy



Epithelial cells may continue to grow on the posterior capsule, forming what are known as Elschnig's pearls. The epithelium secretes a clear material that gets trapped within the epithelial sheets.

In the case presented here, after the first YAG procedure, cells grew into the visual axis and decreased acuity in the right eye. *Figure 6a* shows the undilated initial presentation of a 69-year-old female patient's right eye one year after the YAG capsulotomy. The dilated view in *Figure 6b* clearly shows Elschnig's pearls, for which the patient underwent repeat capsulotomy. *Figure 6c* offers a retroview, exposing multiple pearls that remain in the posterior opacification capsule. Note how the second YAG procedure created an enlarged clear central area without pearls. The patient's vision was restored to 20/20.

Case 7. Retinal Detachment



This 57-year-old male's retinal detachment occurred seven months after cataract surgery in his left eye. Detachment issues are uncommon in general, but are more common in patients younger than 60 years who have not yet had a posterior vitreous detachment or in patients whose axial length is 26mm or longer. Trauma is also associated with retinal detachment. This patient underwent a scleral buckle procedure and achieved a visual acuity of 20/20.

iris-fixated and anterior chamber IOLs, also raise the risk.[24,25]

The prime feature of PBK is persistent stromal corneal edema with associated loss of endothelial cells, which progresses towards the intercellular epithelial region and forms characteristic bullae.[24] Its treatment includes topical hyperosmotic agents, 2% and 5% sodium chloride solution and ointment, dosed based on the level of corneal edema.[24] Sometimes, topical hypotensives such as beta blockers and alpha agonists decrease corneal edema; however, clinicians should avoid carbonic anhydrase inhibitors for the risk of increased endothelial toxicity, and both prostaglandin analogs and miotics can worsen the ocular inflammation.[24] In cases of painful epithelial bullae, bandage contact lenses may be used. In terms of surgical care, penetrating keratoplasties were once the treatment of choice, but the newer lamellar endothelial keratoplasties (such as DSAEK and DMEK) have gained popularity and are now considered first line treatments.

Attentive postoperative cataract care is vital for the patient's success, both short and long term. Crucial for this care is having a firm understanding of expected and unexpected complications, and their subsequent management. Be sure to contact the cataract surgeon for any sight-threatening medical complications or if there is any doubt as to treatment regimen. ■

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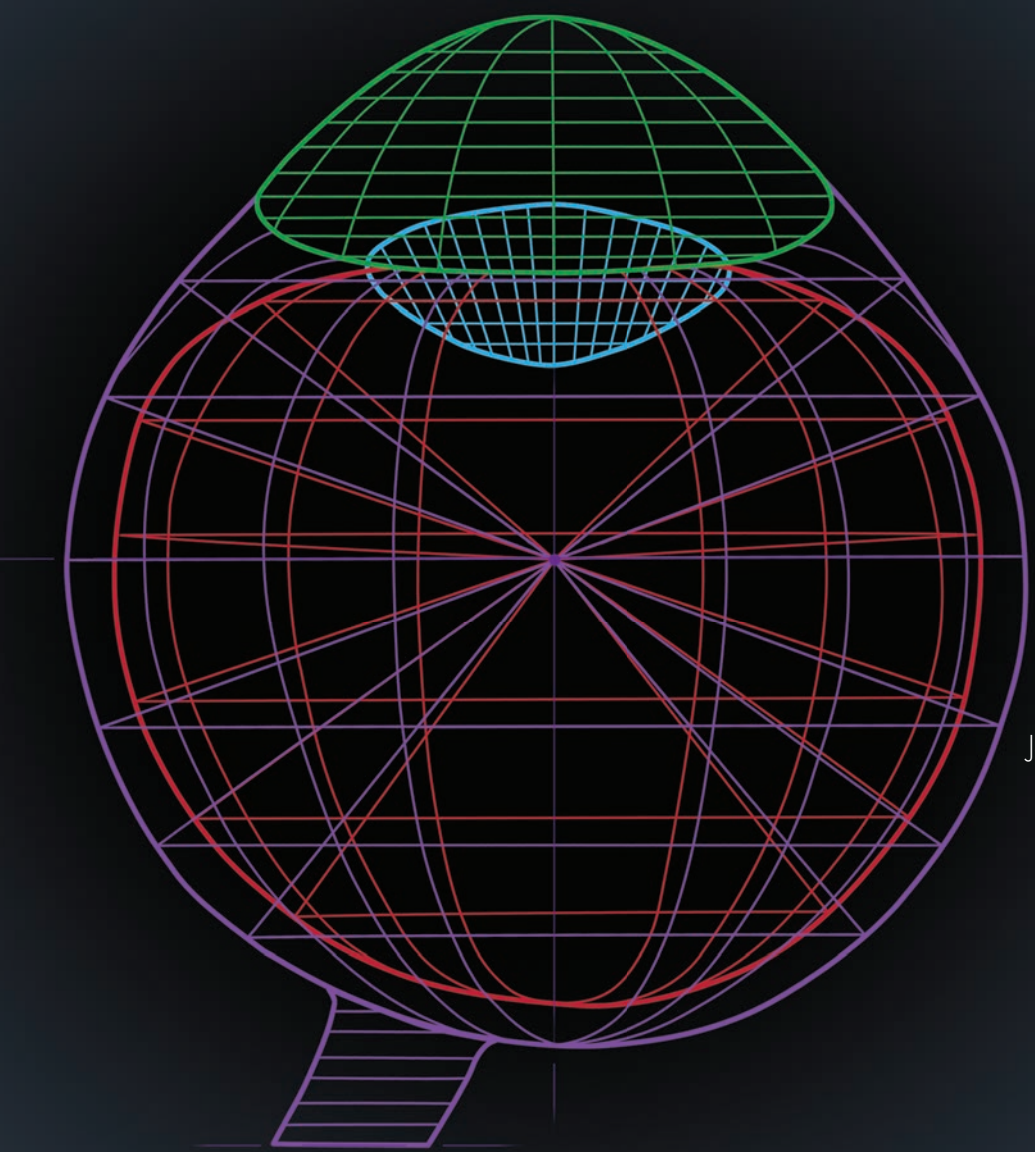
Taking Glaucoma Risk Assessment to the Next Level: The Role of CORNEAL HYSTERESIS



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Jonathan S. Myers, MD
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Jeffrey M. Liebmann, MD
Anne L. Coleman, MD, PhD
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John Flanagan, MCOptom, PhD

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REVIEW OF OPTOMETRY

FACULTY

Robert N. Weinreb, MD: Dr. Weinreb is the chairman and distinguished professor of ophthalmology, Morris Gleich Chair, and the director of the Shiley Eye Institute at the University of California, San Diego. A graduate of Harvard Medical School, Dr. Weinreb has trained more than 140 post-doctoral fellows in glaucoma, many of whom hold distinguished academic and leadership positions throughout the world. In April 2015, his h-impact factor was 95.

James D. Brandt, MD: Dr. Brandt is professor and vice-chair of ophthalmology and vision science and director of the Glaucoma Service at the University of California, Davis. Dr. Brandt has served as the principal investigator of numerous clinical trials, including the Ocular Hypertension Treatment Study (OHTS). Dr. Brandt serves on the editorial boards of Ophthalmology and Journal of Glaucoma.

Nathan M. Radcliffe, MD: Dr. Radcliffe is the director, glaucoma service and clinical assistant professor, New York University Langone Ophthalmology Associates and a cataract and glaucoma surgeon at the New York Eye Surgery Center.

Felipe A. Medeiros, MD, PhD: Dr. Medeiros is Professor of Ophthalmology and the Ben and Wanda Hildyard Chair for Diseases of the Eye at the UCSD School of Medicine. He is also Medical Director of the Hamilton Glaucoma Center, University of California San Diego and Director of the Visual Performance Laboratory at the Shiley Eye Institute.

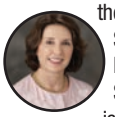
Jonathan S. Myers, MD: Dr. Myers is an associate attending surgeon on the glaucoma service of Wills Eye Hospital. He serves on the editorial boards of Glaucoma Today and the Journal of Clinical and Experimental Ophthalmology. Current research interests include perimetry and novel glaucoma surgeries.

Tony Realini, MD, MPH: Dr. Realini is an associate professor of ophthalmology at West Virginia University. Dr. Realini previously worked in the Department of Ophthalmology at the University of Arkansas for Medical Sciences. He has received numerous research grants, including two from the National Eye Institute, and has published widely in ophthalmic medical journals.

Ronald L. Gross, MD: Dr. Gross recently joined West Virginia University (WVU) as professor and chair of the Department of Ophthalmology and is the director of the WVU Eye Institute in Morgantown, West Virginia. He holds the Jane McDermott Shott Chair in Ophthalmology. He previously worked at the Cullen Eye Institute of Baylor College of Medicine in Houston, Texas, where he held the Clifton R. McMichael Chair and was a professor of ophthalmology.

Jeffrey M. Liebmann, MD: Dr. Liebmann is professor of ophthalmology at Columbia University Medical Center, New York, NY. Dr. Liebmann currently serves as president of the World Glaucoma Association, is past-president of the American Glaucoma Society, secretary-treasurer of the New York Glaucoma Society, and co-editor of Journal of Glaucoma and is a member of the board of governors of the World Glaucoma Association and boards of directors of The Glaucoma Foundation and the American Glaucoma Society Foundation.

Anne L. Coleman, MD, PhD: Dr. Coleman is a professor of epidemiology at UCLA's Jonathon and Karin Fielding School of Public Health and the David Geffen School of Medicine. She is also the Fran and Ray Stark Professor of Ophthalmology at the Stein Eye Institute. Dr. Coleman is the past chair of a 14-member panel of experts overseeing the National Eye Health Educational Program of the National Eye Institute, the prior Secretary of Quality of Care for the American Academy of Ophthalmology and the founding director of the AAO H. Dunbar Hoskins Center for Quality Care.



Murray Fingeret, OD: Dr. Fingeret, a graduate of the New England College of Optometry, completed a residency at the Joseph C. Wilson Health Center in Rochester, New York. Dr. Fingeret is chief of the Optometry Section, Brooklyn/St. Albans Campus, Department of Veterans Administration New York Harbor Health Care System. Dr. Fingeret is also a clinical professor at the State University of New York, College of Optometry.

John Flanagan, MCOptom, PhD: Dr. Flanagan is the dean and a professor at the School of Optometry and Vision Science Program, University of California, Berkeley. Until May 2014, he was professor at the School of Optometry and Vision Science, University of Waterloo and in the Department of Ophthalmology and Vision Sciences, University of Toronto. He was director of the glaucoma research unit, Toronto Western Research Institute and a senior scientist at the Toronto Western Hospital, University Health Network.

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The Role of CORNEAL HYSTERESIS

INTRODUCTION

Glaucoma is a complex, multifactorial disorder that affects the optic nerve and can lead to functional vision loss or blindness if not treated. Reduction of intraocular pressure remains the only established form of therapy to slow or halt the progression of glaucoma. The aggressiveness of therapy is often based on a global risk assessment. Risk factors for glaucoma are well established and include intraocular pressure, age, central corneal thickness, and ethnicity, among others. Corneal hysteresis—a measure of the viscoelastic biomechanical properties of the eye—is emerging as an additional important risk factor for glaucoma progression. Corneal hysteresis is easily measured in a noninvasive fashion in the office, and emerging data support its importance in the process of global risk assessment for glaucoma. In 2015, hysteresis was given a reimbursable CPT code.

Recently, a group of glaucoma specialists gathered in San Francisco to review and interpret the data supporting the role of corneal hysteresis in glaucoma risk assessment. This gathering was supported by Reichert—manufacturer of the Ocular Response Analyzer, the only device that measures corneal hysteresis.

This monograph is intended to share the key take-home messages derived from that meeting. These include a basic understanding of corneal hysteresis and its relationship to ocular biomechanics, familiarity with the data supporting the importance of hysteresis in glaucoma risk assessment, and guidance on incorporating hysteresis in the clinical management of glaucoma patients.

What is Corneal Hysteresis? Historical Perspectives of Central Corneal Thickness and Corneal Hysteresis as Risk Factors for Glaucoma

Robert N. Weinreb: Corneal hysteresis (CH) has been of great interest in glaucoma for more than ten years. There now are several hundred publications, many of which validate and support its use in glaucoma care. In clinical research studies, there is compelling evidence that CH is a powerful tool for predicting the development of glaucoma and its progression as well. Today's discussion discusses the use of CH in clinical glaucoma care.

Dr. Brandt: The emergence of CH as a risk factor for glaucoma is reminiscent of the path that central corneal thickness (CCT) followed in becoming a validated risk factor for glaucoma. The influence of CCT in IOP measurement had been recognized since the 1950s. Its widespread acceptance and use in risk modeling did not occur until the Ocular Hypertension Treatment Study (OHTS) provided strong evidence of its importance and practical guidance on how to incorporate it into the risk assessment process. Many of us were surprised that CCT was such a strong risk factor in OHTS, and it was helpful in establishing CCT's credibility that the European Glaucoma Prevention Study (EGPS) confirmed this finding.

Dr. Weinreb: We began to evaluate the role of CCT in glaucoma in the Diagnostic Innovations in Glaucoma Study (DIGS), which began in 1986. One early analysis from DIGS involved 98 patients with suspected preperimetric glaucoma—their optic nerves looked suspicious but their visual fields were full. After a follow-up period of about eight years, 60% had converted to glaucoma. But when the subjects were stratified into two groups based on thin or thick corneas, the rate of conversion to glaucoma was 46% in eyes with thin corneas compared to 11% in eyes with thicker corneas.[1]

Dr. Brandt: The question that arose then was this: is CCT truly a risk factor or is it merely a source of error in intraocular pressure (IOP) assessment? One possibility is that eyes with thin CCT have higher IOP than we measure using Goldmann tonometry, and that is why these eyes fare less well. An alternate possibility is that CCT is an indicator of more global ocular biomechanics. Several lines of research suggest that CCT is an important risk factor indepen-

dent of any effect on IOP measurement. In OHTS, CCT was an independent risk factor even in models that included IOP—in other words, CCT added information about risk that was not included in IOP.[2] Also in OHTS, correcting IOP on the basis of CCT using any of several formulas failed to fully explain the effect of IOP on risk.[3] In support of CCT as a biomarker for ocular biomechanics was a small, early study in which differential compliance of the lamina cribrosa was observed in eyes with thin vs. thick corneas.[4]

Dr. Radcliffe: CCT as a biomechanical indicator has limitations, and chief among them is that most models relating CCT and IOP assumed that the cornea is a purely elastic structure. In fact, the cornea is viscoelastic. To understand this difference, consider the shock absorbers in your car. On a bumpy road, they dampen the bumps and smooth out the ride. If you had only springs, which are elastic—and not shock absorbers, which are viscoelastic—you would feel every bump much more significantly. The shock absorbers dissipate energy. In terms of the eye, the cornea's response to deformation (for instance, applanation) is rate dependent: when moved rapidly like a car wheel hitting a bump, it dissipates some of the energy absorbed during the deformation. This differential tissue response to the load/unload of stress is called hysteresis, a term that was coined in the 19th century. CH is not a measure of the stiffness of the cornea, but rather a measure of how corneal tissue absorbs and dissipates energy during deformation and return. It can be considered a measure of tissue function rather than a geometrical attribute. There are correlates to CH in other bodily systems. The ascending aorta exhibits viscoelastic behavior with every heartbeat, expanding to accept blood from the heart and absorbing energy in the process, then rebounding and dissipating that energy as that blood flows more distally.

Dr. Brandt: Because both CCT and CH are biomechanical parameters of the cornea, they tend to be weakly correlated. Data suggest, however, that CH may be a better predictor of glaucoma than CCT. These data will be reviewed in the next section. So in summary, CH has followed a similar path as CCT in becoming recognized as a risk factor for glaucoma. CH may be more closely related to glaucoma risk than CCT. This likely relates to its functional nature (how the eye responds to dynamic changes in IOP compared to CCT's more structural nature (how thick it is).

›››››› Corneal Hysteresis as an Indicator for Glaucoma Progression Risk

Dr. Radcliffe: It is useful to review the key studies supporting the clinical utility of CH as a risk factor for glaucoma and its progression.

Among the first studies to demonstrate this was a retrospective report of 230 glaucoma patients and suspects with the goal of identifying associations with progression.[5] The study utilized the OHTS criteria for the determination of both the presence of glaucoma and the progression of glaucoma. Among the associations for progression were patient age, lack of treatment, and CH. Of note, neither IOP nor CCT were found to be significant associations of progression. This study concluded that CH was the only ocular parameter associated with progression.

CH has also been associated with the risk of progression in normal-tension glaucoma (NTG). A retrospective study of 82 eyes being treated for NTG included an assessment of CH.[6] The average value of CH in the group was 10.1 mmHg. The study sample was then divided into two groups: those with CH higher than the mean and those with CH lower than the mean. The risk of progression of NTG was 67% in the 39 eyes with low CH, and only 35% in the 43 eyes with high CH. In a multivariate model of visual field progression, CH was highly predictive while CCT was not significantly predictive at all. This study demonstrated that CH can be utilized independently of IOP and CCT as a prognostic factor for glaucoma progression.

Asymmetry of primary open-angle glaucoma (POAG) may also be explained, at least in part, by CH. One hundred seventeen POAG patients with asymmetric glaucoma (with

asymmetry defined as an inter-eye difference in AGIS visual field score of >5 points) were observed in a prospective cross-over study to evaluate factors associated with asymmetry of glaucoma severity.[7] Among the potential factors evaluated were Goldmann IOP, CCT, the number of IOP-lowering medications used, and CH. Of these, only CH was significantly different between the fellow eyes, being lower in worse eyes (mean 8.2 versus 8.9 mmHg, p<0.001). This study demonstrated that CH offered the best discriminative power for discerning the worse eye in asymmetric POAG.

The rate of visual field progression may also be related to CH. A recent retrospective study of 152 glaucomatous eyes evaluated the correlation between CH and CCT and their relationship with the rate of visual field change over time.[8] This study found that rapidly progressing eyes had lower mean CCT and CH than stable or slowly progressing eyes, and that CH and CCT were modestly correlated (r=0.33). In a multivariate model of visual field progression, only age, peak IOP, and CH were predictive; CCT was not. This study demonstrates that glaucomatous eyes with low CH are at higher risk for progression and progress faster.

The Diagnostic Innovations in Glaucoma Study has been ongoing since the mid-1980s. Its goals are to develop better methods for detecting glaucoma progression, to characterize the rate of progression, and to identify risk factors for progression of glaucoma. Enrolled subjects were either healthy glaucoma suspects or established glaucoma patients. They underwent full eye examinations every six months. This database of patients provides a wealth of

Case 1. Progression Despite Low IOP

Dr. Radcliffe: One of my patients is a 54-year-old Hispanic lady with recently-diagnosed POAG. Her IOP on treatment is 10 mmHg. Her CCTs are in the 540s, her vertical cup-disc ratio is 0.8, and her optic nerves and visual fields are shown in the figure.

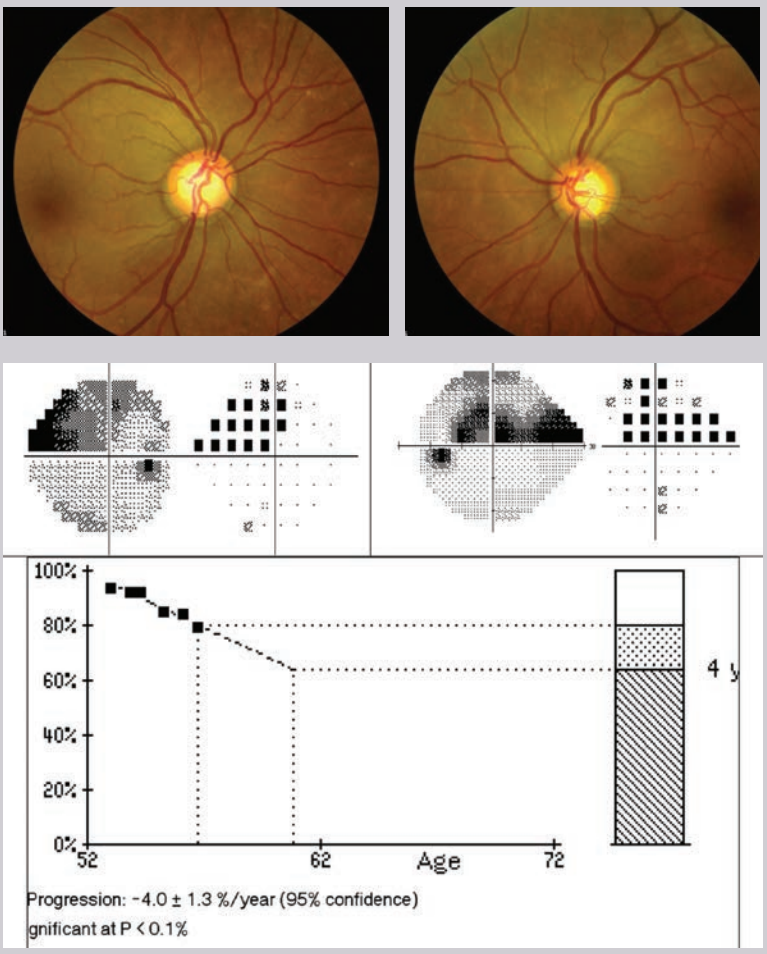


Figure. Optic nerve photographs (top) and visual fields (middle) from the patient described in Case 1. Visual field progression was noted over time (bottom).

Most of us would be satisfied that, with an IOP lowered to 10 mmHg, we have this patient's glaucoma adequately controlled. However, over the next several years, her visual field continues to progress despite maintaining an IOP in the 9-11 mmHg range. We measured her CH before we initiated treatment at the time of her diagnosis, and it was 6.1 mmHg. That's well below the normal range. That CH value indicates that she is at high risk for progression, and that is exactly what happened.

information about glaucoma, progression, and risk factors. A recent analysis of a subset of 68 glaucoma patients followed for four years revealed that Goldmann IOP was significantly influenced by CCT but not by CH, and that CH and CCT were modestly correlated (r=0.48).[9] In a multivariate model of glaucoma progression, CH was three times more strongly associated with the rate of progression than CCT. This study was among the first prospective studies to confirm the relationship between CH and the risk of glaucoma progression.

››› Is Corneal Hysteresis a Biomarker for Susceptibility to Glaucoma Damage?

Dr. Brandt: There are convincing data that CH is related to glaucoma risk and to progression risk. CH is lower in patients with glaucoma than in healthy subjects, and it is lower in glaucoma patients who progress than in glaucoma patients who remain stable. Is this association due solely to CH's effect on IOP measurement, or does CH also tell us something about the biomechanics of the eye? Can CH be a biomarker for optic nerve damage in glaucoma?

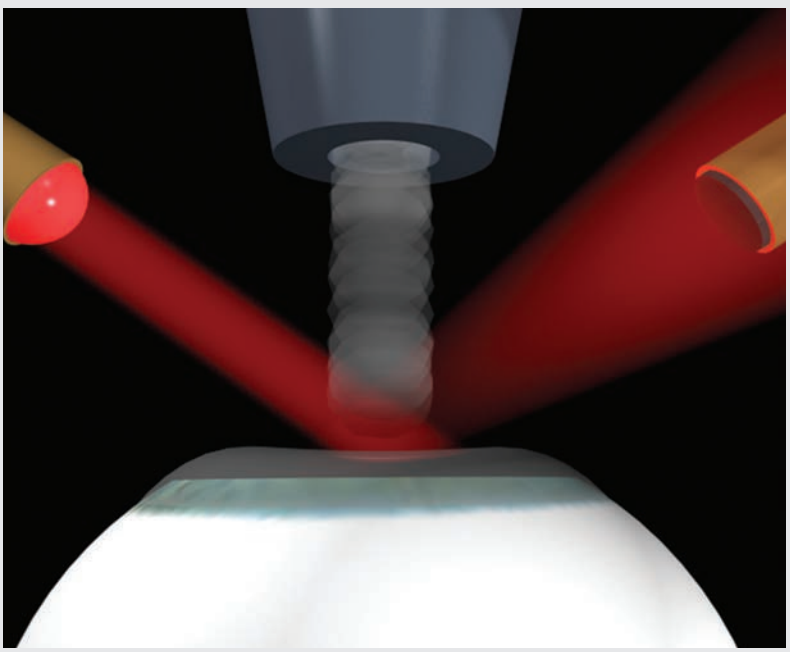
Dr. Radcliffe: There are some interesting studies that give in-

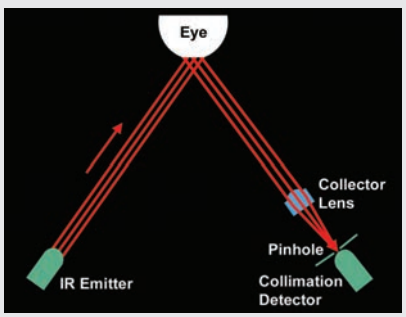
The Reichert Ocular Response Analyzer

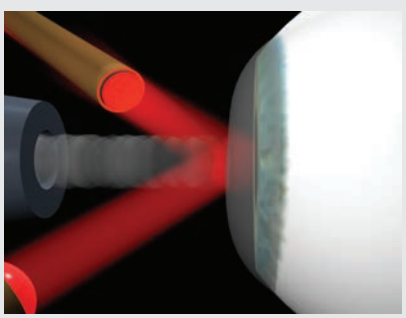
How It Works

Dr. Radcliffe: Corneal hysteresis is easily and noninvasively measured in the office. CH can only be measured using the Reichert Ocular Response Analyzer (ORA). This device functions very much like a noncontact tonometer. A metered puff of air is delivered to the cornea, flattening it into an applanation configuration, much like Goldmann tonometry (see figure). The air puff deforms the cornea past the applanation point, making it briefly concave. As the pressure of the air puff diminishes, the cornea returns to its normal configuration, passing through the applanation position a second time on its rebound. Interestingly, the pressure of the air puff at the point of the first and second applanations is different (being lower on rebound than it was upon initial applanation), as the cornea's viscoelastic nature dissipates some of the energy. The difference in IOP at each of these two applanation points is defined as the corneal hysteresis. If the cornea were perfectly elastic and did not dampen some of the energy, the two applanation points would occur at the same IOP level.

BELOW: Schematic diagrams of ORA measurement procedure.







Corneal Compensated IOP (IOPcc) Accuracy and Safety Advantages

Dr. Radcliffe: In addition to CH, the ORA provides two other parameters, both estimates of IOP. One is a Goldman equivalent IOP (IOPg), which is designed to match Goldmann values. The second is a cornea compensated IOP (IOPcc), which is an estimate of true IOP taking the biomechanics of the cornea into account.

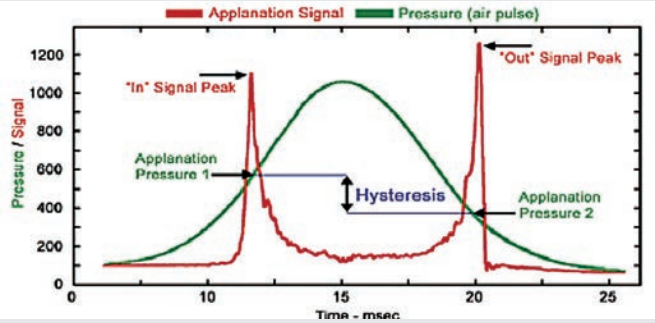
Dr. Medeiros: Goldmann tonometry remains the clinical standard for IOP measurement in most parts of the world. There are some limitations to Goldmann tonometry. Goldmann IOP is not objective—deciding when the mires are aligned is subjective. An objective tonometer that returns a value digitally without a subjective interpretation could make IOP measurement more objective. Likewise, a device that is fast and technician-friendly would be of value. If IOPg reasonably estimated Goldmann tonometry, the ORA could have value in clinical glaucoma management.

Dr. Radcliffe: There is definitely less random variability with IOP measured by the ORA compared to Goldmann tonometry. My experience has been that IOPg provides cleaner data than Goldmann IOP in mathematical models that incorporate IOP. Frankly, if I had an ORA in every one of my exam rooms, I would be comfortable using IOPg in place of Goldmann IOP in clinical practice.

Dr. Realini: The IOPcc measurement is interesting to me. Certainly the biomechanical properties of the cornea affect our IOP measurements regardless of the tonometer we use. None measures true intraocular pressure, and the difference between our measurement and true IOP is likely highly dependent upon corneal biomechanics. Before we had CH, we used CCT as a surrogate measure of corneal biomechanics. But CCT is not a measure of the functional biomechanical properties of the cornea—it is a structural measurement of corneal thickness. Not all thick corneas are stiff, and not all thin corneas are floppy. This is why I have never been a believer in the practice of correcting IOP based on CCT. They are—as Dr. Brandt pointed out—independent risk factors. Correcting Goldmann IOP based on CCT is akin to adding 5 mmHg to IOP for a positive family history—why would you combine two independent risk factors into one? However, CH is different from CCT in that in is a measure of the functional biomechanical structure of the cornea—it tells us how that individual cornea responds to being applanated. It makes far more sense to correct an IOP measurement based on CH than on CCT.

Dr. Fingeret: But the data also show that CH and IOP are independent risk factors, so doesn't the same logic apply? Wouldn't this approach also be combining two independent risk factors into one?

Dr. Realini: That's a valid point. Once we correct IOP based on CH, we have incorporated the component of CH's risk associated with IOP measurement error. Is there also a structural component to CH as a risk factor? Does it both affect our IOP measurement and tell us something about the susceptibility of the optic nerve head and lamina cribrosa to glaucoma damage? It would be interesting to know if CH remains significant in a model of glaucoma progression that includes IOPcc. This would tell us whether CH still brings relevant information to the table after a CH-based IOP correction.



Dr. Brandt: The IOPcc measurement may also be useful in eyes that have previously undergone refractive procedures. LASIK both flattens the cornea and dramatically changes its biomechanical properties. An IOP measurement that takes the altered biomechanics into consideration would be of value. In coming years more and more patients will have had corneal refractive procedures decades earlier and will forget to tell you or your technician about them.

Safety Advantages

Dr. Flanagan: The ability to obtain a Goldmann equivalent IOP using noncontact tonometry offers a variety of important safety issues. Obviously, the risk of corneal abrasion—although very small to begin with—is eliminated. Also, there is no need for anesthesia or fluorescein dye, so we also eliminate possible adverse reactions to these products as well. But perhaps the biggest advantage is the elimination of the risk of infection.

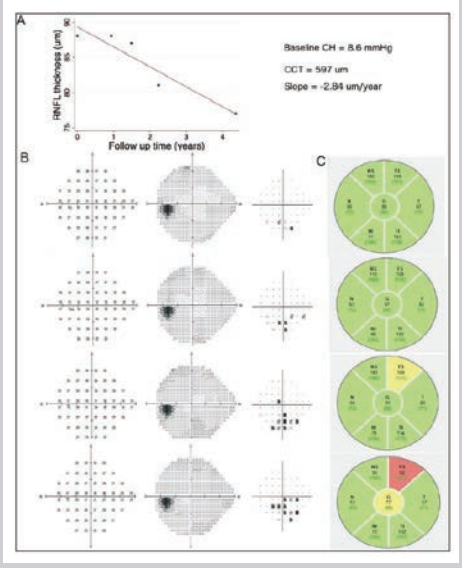
Dr. Coleman: There have been a number of significant outbreaks of epidemic keratoconjunctivitis in eye clinics. Among the methods by which microbes are transmitted from one patient to the next is the incomplete sterilization of the tonometer tip between patients. There are a variety of ways to clean the Goldmann tonometer tip. They can be soaked in bleach or alcohol for five to 20 minutes. Also, it requires that every room have multiple tips, which is costly. A more extreme approach is to sterilize them the same way we do our surgical instruments, but this can lead to cracking of the tip over time. These methods are most likely to be effective, but they come with inconveniences. There are more pragmatic approaches. They can be wiped off with an alcohol pad, or washed by hand with soap and water, or soaked in hydrogen peroxide, although the infection control experts are uncertain that these are adequate.

Dr. Liebmann: There are disposable Goldmann tonometer tips, but they cost approximately $1.25 apiece, which adds considerable expense to every eye examination.

Dr. Flanagan: In the United Kingdom, they have the added concern about prion-based diseases such as Creutzfeldt-Jakob disease. The use of reusable Goldmann tonometer tips ended in the UK more than 10 years ago—everything is now disposable.

Case 2: Progression with Thick Cornea

Dr. Medeiros: This patient from my practice also illustrates how CH might be useful in identifying the patients at risk for progression. This 70-year-old man with POAG has a CCT of nearly 600, which would suggest a relatively low risk of progression. Yet he is clearly progressing by both visual field and OCT criteria (figure). His CH, however, is 8.6, which is not as low as the CH in Dr. Radcliffe's patient, but is still moderately low. This is an eye in which CH revealed a propensity for progression that was in contrast to the CCT.



direct support for this idea. One demonstrated that CH was lower in glaucomatous eyes with acquired pits of the optic nerve than in glaucomatous eyes without such pits.[10] Interestingly, in this study the patients in both groups were matched for peak IOP, so it is less likely that acquired pits form as an IOP-dependent process. Perhaps CH is related to an IOP-independent mechanism of glaucomatous optic nerve damage.

Dr. Brandt: We conducted a study in which we measured axial length in glaucomatous eyes before and after trabeculectomy.[11] We also measured CCT and CH preoperatively. We found that CH was significantly associated with the shortening of axial length, while CCT was not. This study was confirmed by other investigators. These data suggest that the biomechanical responses of an eye to significant IOP reduction can be predicted in part by the CH measurement.

Dr. Radcliffe: There was also a study in which patients with and without glaucoma underwent optic nerve imaging before and after an induced IOP rise. In this study, the higher the CH, the more the lamina cribrosa was deformed backward in response to the pressure. In other words, eyes with higher CH were able to adapt to the IOP change and absorb it, while eyes with lower CH had less of a tissue adaptation to deal with the elevated IOP.

Dr. Brandt: Think of it as optic nerve head compliance. Eyes with higher CH were better able to buffer the IOP rise. As more indirect evidence of this, a study demonstrated that CH but not CCT was correlated with the structural parameters of glaucoma damage measured by confocal scanning laser ophthal-

Future Studies and Challenges

Dr. Fingeret: The data to date suggest that CH can play a role in glaucoma risk assessment. What additional studies would help us to fine tune our understanding of CH?

Dr. Myers: We should consider longitudinal studies. These will tell us several things. First, is CH stable over a patient's lifetime? We cannot answer this question with cross-sectional studies. Second, does CH change as glaucoma progresses? In other words, is low CH an indicator of progression risk or a consequence of it? And third, long-term studies will provide us with the data we need to better incorporate baseline CH values into a risk calculator for glaucoma progression.

Dr. Radcliffe: There are data that suggest CH changes in response to IOP reduction. Specifically, CH goes up as IOP is lowered. This may be a purely mechanical effect, as CH is known to be slightly correlated to IOP. But it may also be a sign that the low CH associated with glaucoma is recovering when glaucoma is treated. So it would be interesting to better characterize the effect of treatment on CH, and to see if the change in CH with treatment is predictive of progression vs. stability.

Dr. Gross: I would like to see a study of CH measured before and after trabeculectomy. Once there is an expansile reservoir incorporated into the eye—the bleb—I would expect the biomechanical compliance of the eye to increase. Also, I wonder if CH would help us to understand why some patients with very low postoperative IOP—say, 5 mmHg or less—develop hypotony maculopathy and others do not.

moscopy, with lower CH being associated with worse nerve damage.[12]

Dr. Realini: The question is: are these eyes progressing because they have low CH, or do they have low CH because they have glaucoma? Does glaucoma lead to a reduction in CH? In cross-sectional studies, this cannot be determined. A longitudinal study will be necessary to see if CH diminishes as patients progress from early glaucoma to moderate or advanced glaucoma.

Dr. Myers: This is an important point. In response to stress, bone creates more bone. In response to chronic hypertension, arteries produce more collagen and become stiffer and less compliant. Does a glaucomatous eye undergo connective tissue responses that would change its compliance and thus its hysteresis?

Dr. Realini: Either way, low CH is a sign of high risk for progression.

Billing for CH and Incorporating Into Clinical Practice

Dr. Radcliffe: The evidence supports a role for measuring CH in our patients with POAG. There is now a CPT code for the measurement and interpretation of CH. It is 92145. This is for one or both eyes. The frequency—once per year? once per lifetime?—has not been established, nor have the diagnostic codes that will support the test been established.

Dr. Gross: I suspect that even though Medicare has assigned the code and seems to be paying for it, that, like with many new codes, most private insurance providers will initially not pay for the service. But we need to bill for it anyway in order to demonstrate a volume of use that will play a role in making it payable eventually by private payers.

Dr. Brandt: As we move toward greater and greater office efficiency, we spend more time thinking about workflow. How can we best incorporate CH assessment into our clinical workflow? There are several issues. Who are the optimal patients for CH assessment—and who are not? How often does it need to be done? At what point in the workflow should it be done?

A new CPT code, 92145, has been published specifically for the Corneal Hysteresis measurement provided by the Reichert® Ocular Response Analyzer®. In the 2015 CPT handbook, a new, permanent, Category I CPT code, 92145 (Corneal hysteresis determination, by air impulse stimulation, unilateral or bilateral, with interpretation and report), replaces the prior temporary, Category III CPT code, 0181T. The new code took effect January 1, 2015.

According to "An Insider's View" published by the American Medical Association, this test achieved Category I status because the clinical utility has been established and usage has grown since 2007 when the Category III code was implemented. The code descriptor was changed slightly; it now describes a test performed on a single eye or both eyes (e.g., unilateral or bilateral).

"This change relieves a significant administrative burden for ophthalmologists and optometrists who perform corneal hysteresis and seek reimbursement for this diagnostic test. For most payers, including Medicare, Category III CPT codes are not covered while Category I codes are usually covered and reimbursed,"

—Kevin J. Corcoran, COE, CPC, CPMA, FNAO, president of Corcoran Consulting Group, during his presentation at the 2015 Hawaiian Eye meeting.

Dr. Myers: I think patient selection for measuring CH will be similar to that for CCT when it first emerged. We would want to know CH in patients who are glaucoma suspects to assess their risk of developing glaucoma. We would want it in treated patients who are progressing despite what appears to be adequate IOP control. We might want it in those odd patients who have markedly high IOP but no evidence of damage in order to better understand how the eye is tolerating the IOP.

Dr. Coleman: We should consider getting it in all of our established glaucoma patients if helps us decide which of them is at high risk for progression.

Dr. Brandt: I would add that patients who have undergone refractive surgery such as LASIK might be good candidates. The CH value may not be useful—it would be a measure of their altered cornea and not of their native eye. But the ORA IOP measurement (IOPcc) might be useful. We know that these

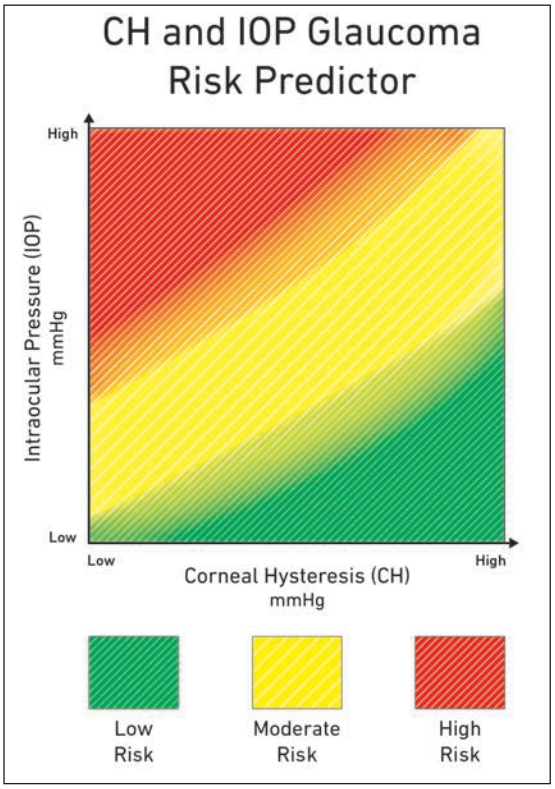
procedures alter the accuracy of Goldmann tonometry by altering corneal biomechanics in a way that the Goldmann tonometer cannot compensate for. IOPcc, may be a better measure of IOP than Goldmann IOP in these eyes.[13]

Dr. Radcliffe: Eyes with IOP over 30 mmHg may not be good candidates for CH measurement. In these eyes, the ORA will underestimate CH in order to avoid hitting the eye with an air puff strong enough to measure it accurately. This is not a significant limitation, however, because once the IOP is above 30, the need for IOP reduction is usually evident.

Dr. Realini: In those ocular hypertensives with high IOP and normal nerves, it might still be useful. If CH is underestimated in high-IOP eyes, an elevated CH in such an eye would be particularly compelling given that the true CH might be even higher.

Dr. Weinreb: In our 24-hour study, short-term variability of CH was not seen.[14] In studies by our group and others, CH does tend to diminish with age, by approximately 0.2 mmHg per decade.[15] But other studies suggest that CH may be a more dynamic measurement than CCT, especially when pathology is present.

Dr. Brandt: As for when to do it, I would ideally like to have it available when I see the patient. It should be available when I see the IOP, so I can use these two pieces of data together. It is not feasible to put an ORA into every exam room. One way to incorporate CH assessment into practice is to have a work-up area that all patients come through for their vision, pressures, and CH, then come to the exam room to be seen by the doctor.



Dr. Realini: That's a good point. Pachymeters are easily portable. But the ORA cannot come to the patient—the patient has to come to the ORA.

Dr. Brandt: Many of our devices are moving toward DICOM compatibility, so that they interface with our electronic health record. Therefore EHR software should have fields for corneal hysteresis. It is not clear yet whether EHR companies are incorporating this parameter.

Dr. Liebmann: One potential solution is to integrate this test in combination with our other standard glaucoma tests. Get CH after your visual fields, or after your OCT. Make it an automatic part of the process until you've gotten CH on all your patients.

Dr. Coleman: Once we have

Normal Values for CH

Dr. Radcliffe: In studies including healthy subjects from the United States, United Kingdom, South America, Europe and Asia, normal values for CH fall in the range of 10.1 to 10.9 mmHg.

Dr. Weinreb: We conducted a study several years ago in which 15 healthy subjects underwent 24-hour assessment of IOP, CCT and CH in our sleep laboratory at the Hamilton Glaucoma Center, University California San Diego. Both IOP and CCT demonstrated significant 24-hour variability, with highest values recorded during the nocturnal period. In contrast, CH was quite stable throughout the 24-hour period, with no significant variation at all.[14] Children tend to have high hysteresis (around 12 mmHg),[16,17] and our group and others have also demonstrated that CH does decrease slightly with age.[18,19] The significance of age-dependent decreases in CH is unknown.

Points of Consensus on Corneal Hysteresis

- CH is associated with the risk of glaucoma progression
- CH measurement would be valuable in assessing the risk of glaucoma suspects progressing to glaucoma, and in assessing the risk of progression of established glaucoma.
- At present, CH should be considered a semi-quantitative risk factor: low (CH <8 mmHg), medium (CH 8-12 mmHg) or high (CH >12 mmHg).
- Future research will enhance our understanding of how to best utilize CH in glaucoma risk assessment.

incorporated the testing process into our workflow, how do we incorporate the data into our patient management? What is the normal value for CH? What is the normal range? At what CH level should I consider my patient to be at increased risk of progression?

Dr. Radcliffe: The mean value in most normal populations is between 10 and 11 mmHg. The normal range is typically between 8 and 14 mmHg.

Dr. Brandt: I don't think we have adequate data yet to establish the risk of progression associated with specific values of CH. My approach will be to utilize the same approach I do with CCT. I think of the values as low, medium or high, and I think of the associated risk in the same way. A low CH with no other risk factors is no more compelling than a low CCT with no other risk factors. But in a patient you are already worried about—say, they have already gone blind in one eye or they have a strong family history of glaucoma blindness—in these patients, a low CH might be the straw that breaks the camel's back and prompts you to be more aggressive to prevent progression, while a normal or high CH might make you decide to maintain your current therapy and watch closely.

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Corneal Hysteresis is more associated with visual field progression than CCT or IOP.[1-3]

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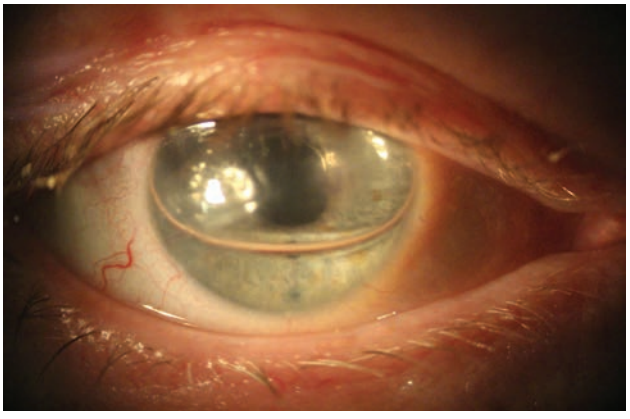
22nd Annual Surgery Report

Go Thin for the Win: A Review of Endothelial Keratoplasty

New techniques may be on the verge of becoming common practice, but sometimes the preference depends on the patient. **By Mitch Ibach, OD**

Fuchs' endothelial dystrophy headlines a group of posterior corneal diseases that cause blurred and hazy vision, glare and halos, progressive corneal edema and, eventually, pain due to corneal bullae. Fuchs' dystrophy is a bilateral autosomal dominant corneal dystrophy characterized by continuous depletion of endothelial pump cells and abnormal outgrowths on the posterior cornea called guttae (commonly referred to as guttata).[1] As endothelial pump cells become fewer and weaker, the cornea swells, leading to a decrease in visual acuity. Corneal transplantation with healthy endothelial cells is the only long-term treatment to restore corneal transparency and reverse corneal edema.[2]



This slit lamp photo shows a patient one day after cataract surgery with DMEK. A gas bubble is covering the pupil with patent inferior PI.

Recently, lamellar endothelial keratoplasty (EK) has become the standard surgical option for these patients.[2,4] According to the Eye Bank Association of America, EK represented more than 40% of corneal grafts in 2010, and this number continues to rise.[5] With rapid and predictable visual recovery and lower risk of serious postoperative complications, it's no wonder surgeons are choosing lamellar EK procedures for endothelial disease in place of penetrating keratoplasty (PKP).[6]

Identification

Diagnosis of Fuchs' dystrophy starts with a detailed case history. Depending on disease severity, patients may note blurred or hazy vision, glare, starbursts and difficulty driving at night. A hallmark symptom of Fuchs' dystrophy is blur upon waking—due to corneal edema—that improves throughout the day.

On slit lamp exam, the endothelium has guttae, the accumulation of which, especially as they become confluent, can render an "orange peel" appearance when the posterior cornea is retroilluminated. Auxiliary testing centers on viewing the corneal edema and imaging the endothelial cells. Corneal edema can be analyzed with both ultrasound pachymetry and tomography. Devices that can perform corneal tomography (not to be confused with optical coherence tomography), such as the Pentacam (Oculus), are helpful for these patients because they use "slit imaging" to analyze both anterior as well as posterior corneal surfaces in near three-dimensions. Contrast this with reflective Placido ring-based topography systems, which precisely measure anterior corneal elevation and surface.[7]

Specular microscopy is also a big driver in diagnosis and surgical decision making in these patients. The

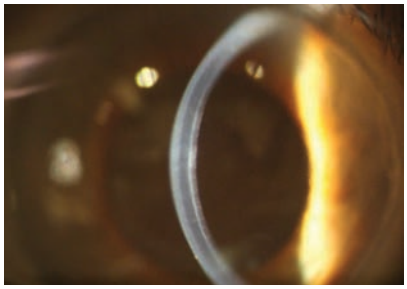
technology provides a quantitative endothelial cell count, as well as qualitative analysis of cell morphology. CellChek (Konan Medical) also measures corneal thickness. A young healthy eye has an endothelial cell density (ECD) of 2,900 cells/mm^2 to 3,500 cells/mm^2, but as patients age into their 40s and 50s, they experience a natural slow decline in ECD.[8] Fuchs' dystrophy and pseudophakic bullous keratopathy (PBK) accelerate ECD decline. Research shows that ECDs above 500 cells/mm^2 are necessary to maintain corneal transparency and ward off cornea edema.[8]

A critical decision for all eye care providers is when to refer these patients for surgical intervention. Relying only on preoperative refraction, pachymetry, tomography or endothelial cell count can be misleading for a struggling patient. Clinicians must listen to patients regarding their symptoms to develop a better sense of when to refer, as morning blur, or corneal edema, is a big clue for visually significant endothelial disease.

Due to significant advancements in safety and visual outcomes with EK procedures, this option can be confidentially recommended earlier in the disease process.

Two of a Kind

The major endothelial keratoplasty techniques now commonly performed include: Descemet's stripping endothelial keratoplasty (DSEK/DSAEK) and Descemet's membrane endothelial keratoplasty (DMEK). The 'A' in DSAEK denotes an automated stripping of the donor tissue, which is by far the most common graft preparation method. DSEK involves removing Descemet's membrane (DM) and endothelium and inserts a graft tissue of posterior stroma, Descemet's, and endothelium. The corneal graft tissue has extra stroma. In this case (DSEK) stroma sticks to stroma and helps with adhesion. In contrast, DMEK involves removing DM and endothelium and inserts a graft tissue of DM and endothelium. The same layers go in as what the surgeon takes out, so native corneal anatomy is kept the same.



This slit lamp photo shows classic endothelial changes in a patient with Fuchs' dystrophy. Guttata are evident in this photo.

Both procedures start similarly with an inferior Nd:YAG peripheral iridotomy (PI). Because DSEK involves contact between host stromal tissue and posterior stromal tissue from the graft, the sticky stromal tissue in the cornea helps with attachment. In DMEK, the lamellar graft does not have stroma, so host stroma is pressed up against donor DM. The donor graft is inserted into the anterior chamber and slowly unfolded. After the endothelial graft is placed, a gas or air bubble is injected posteriorly to press the tissue into place.

Postoperatively, the patient is instructed to position themselves with their nose pointed to the ceiling to allow gravity to aid in massaging the graft into place. Over the next four to seven days, the bubble will dissipate; surgeon preference dictates the duration of positioning, which often depends on graft appearance.

Management

Following endothelial keratoplasties, management starts immediately after the bubble is placed. Patients should expect hand-motion to count-fingers vision for the first few days as the gas bubble dissipates superiorly above the pupil. This is a critical time to closely monitor the intraocular pressure (IOP), as the bubble can block aqueous movement, leading to an iatrogenic pupillary block angle closure.

Patient education is crucial to ensure they understand the proper positioning with a gas bubble, in addition to inferior PI, and the relief that may come by simply changing from a supine to an upright position. If the PI isn't functioning or a portion of the bubble moves posteriorly behind the iris, the patient's IOP can become elevated, which causes an aching periocular pain, nausea and even vomiting. In this case, the patient needs to return to the surgical team so they can, most commonly, create an incision to manipulate the bubble position, known as "burping."

Since an EK involves a tissue graft, postoperative medication, including a steroid, will help prevent graft rejection episodes. Steroid regimens differ depending on the surgeon, but most commonly patients are started more aggressively on a steroid and tapered over a year to indefinite minimum dosing. Most patients with DMEK can stop steroid medication one year postoperatively, while the treatment paradigm for DSEK is still evolving. In our practice, we stop steroids at one year post-op in both DMEK and DSEK patients, but any patient who develops a graft rejection episode will be restarted on steroid and moved to a tapering schedule with steroids once a day indefinitely.

The post-op day one check revolves around IOP and graft position; most importantly, the graft

should be anterior to the bubble and starting to adhere. Over the next seven to 10 days, the graft will adhere, and comanaging optometrists should be on the lookout for early graft scrolling or detachments. If the graft is detaching, the surgical team will need to decide on treatment options, such as: monitoring, adding a second air bubble at the slit lamp or returning to the operating room for graft repositioning. Once the graft is attached and functioning properly, the patient is ready for new glasses, which commonly occurs around three months post-op.

DMEK and DSEK have been game-changers for patients with endothelial disease and resulting corneal edema. With DSEK, the cornea is routinely 600µm or greater due to extra stromal tissue inserted on the graft, while DMEK commonly maintains pachymetry postoperatively at around 540µm to 550µm. The varying amount of graft tissue inserted back into the eye differentiates these procedures, and both DMEK and DSEK have advantages worth reviewing.

Comparing DSEK and DMEK

First, and most importantly, patients care about visual acuity. With DSEK, patients are left with their own corneal stroma and posterior stromal tissue on the donor graft. Research suggests this causes abnormal posterior astigmatism, a hyperopic shift and increased higher order aberrations.[3] Recently, researchers published comparative results of 100 DSAEK eyes vs. 100 DMEK eyes. The DSAEK group had a preoperative best-corrected visual acuity (BCVA) of 0.41 logMAR (20/50- Snellen) compared with a BCVA of 0.27 logMAR (roughly 20/40+ Snellen) in the DMEK group.[9] At the six-month postoperative visit, the DSAEK group's mean BCVA improved to 0.20 logMAR (20/32- Snellen) compared with a mean BCVA in the DMEK group of 0.11 (20/25- Snellen).[9] In a similar study directly comparing DMEK and DSEK, researchers found both groups had a BCVA of approximately 20/100 at three and six months postop.[3] In the DSEK group, BCVA improved to 20/60 and 20/40 at three and six months, respectively; in the DMEK group, BCVA was 20/32 at three months and improved to 20/25 at six months.[3] These studies suggest that with DMEK, patients recover best visual acuity sooner, and best potential acuity is greater.

Table 1. Endothelial Cell Density by Age[8]

Age	Average Endothelial Cell Density (cells/mm^2)
10-19	2,900-3,500
20-29	2,600-3,400
30-39	2,400-3,200
40-49	2,300-3,100
50-59	2,100-2,900
60-69	2,000-2,800
70-79	1,800-2,600
80-89	1,500-2,300

Success rates. Long-term graft success is directly correlated to minimizing endothelial cell loss during tissue implantation and reducing the risk of transplant rejection. Decreased ECD is the principal reason for shortened graft longevity. When researchers compared ECD at the six month follow up between DMEK and DSEK, the results were almost identical: 1,520 cells/mm^2 and 1,532 cells/mm^2, respectively.[3] Other researchers also found no statistical difference in ECD between DMEK and DSEK at six months, and ECD percent loss matched comparative published data.[9,10] Recently, researchers analyzed rejection episodes at one year post-op and the prospective risk of rejection at two years in DMEK vs. DSEK. At one year, there was a 0.7% rejection rate in the DMEK group compared with 9% in the DSEK group; DSEK had a two-year prospective risk of 12% compared with 1% in the DMEK group.[6] This data parallels other research, which found no rejection episodes in over 100 DMEK eyes at the nine-month follow up.[11]

Decreasing detachment rates. Graft adhesion is an evolving process for DMEK and DSEK, but detachment rates for both procedures have decreased as the techniques are perfected. In DSEK, the sticky stromal tissue in the cornea helps with attachment, while in DMEK, host stroma is pressed up against donor DM. Although researchers found a much higher partial detachment rate in DMEK at 82% compared with DSEK's 20%, the study grossly overestimates detachment rates in both surgeries, as the goal was to catch graft dehiscence at the absolute earliest point.[3] Another key to interpreting this research: they used air bubble injections to tamponade the new graft into place vs. a gas bubble with sulfur hexafluoride (SF_6). SF_6 gas has a higher surface tension and a longer half-life inside the eye compared with air and serves a nice compliment to both DMEK and DSEK.[4] Investigators measured DSEK outcomes with air bubbles vs. DSEK with SF_6 gas bubbles and found that, on graft adhesion, the air group had a 27.2% detachment rate vs. zero detachments in the gas group.[4] Interestingly, the air group had a statistically significant higher ECD loss than the gas group.[4]

As surgeons conquer the learning curve with lamellar transplants, graft detachment rates continue to fall, in part due to the switch to gas bubbles. While DMEK lamellar transplants pose greater risk for graft detachment, many surgeons are reporting rates below 10%.[9,10]

A Patient-dependent Decision

The added adhesiveness of DSEK benefits patients with a previous glaucoma shunt procedure, as they already have a crowded anterior chamber. The same is true for patients with sutured intraocular lenses (IOLs), anterior chamber IOLs or patients who have undergone vitrectomy. Lamellar transplants, usually DSEK grafts, can safely be used under a previous PKP as well. Finally, in a patient with greatly reduced visual potential, lessening their risk of multiple air/gas additions due to graft detachments may favor DSEK.

A growing population of patients with endothelial diseases, mainly Fuchs' dystrophy, suffers from reduced vision and corneal edema secondary to unhealthy pump cells. Fortunately, innovation in lamellar transplants offers these patients a long-term and reliable surgical treatment option. As ODs on the front lines, we must educate patients on corneal transplant advancements and maintain our role as pivotal comanagement teammates.

With positive research results and standardized techniques, the excitement grows as surgeons slowly transfer to DMEK as their go-to procedure. It replaces diseased endothelial cells with a near-perfect anatomic replacement to restore corneal transparency and pump functions. When discussing surgical options with patients, you can confidently state that DMEK offers a lamellar transplant with less risk of graft rejection and a faster visual recovery with great endpoint visual acuity. ■

Dr. Ibach specializes in advanced anterior segment surgery care and pathology at Vance Thompson Vision in Sioux Falls, SD. He is a fellow of the American Academy of Optometry and a member of the American Optometric Association.

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Better Target Dry Eye with Three Easy Tests

These simple tests can help you catch a significant amount of DED cases.

By Chris Lievens, OD

The prevalence of dry eye disease (DED) has focused the attention of many in eye care on the plethora of new methods of analysis and treatment. Sure, any clinician would love to successfully identify and manage dry eye 100% of the time and would even settle for success most of the time, especially if it doesn't mean spending a king's ransom. We may never reach 100% success, but we can maximize our efforts by establishing a specific protocol when confronted with these patients.

Proper dry eye diagnosis can fall into three categories:

1. Patients who present with symptoms and signs of dry eye.
2. Patients who have symptoms, but lack signs.
3. Patients free of symptoms, but show an anomalous sign indicative of dry eye and similar diagnoses.

The trick to correctly diagnosing dry eye is to identify the presence of lid wiper epitheliopathy (LWE), measure tear osmolarity and determine the presence or absence of ocular surface inflammation.

This article provides an in-depth look at how to approach these three steps and get closer to hit-

Dry eye tests need not be exorbitantly costly. The InflammaDry (Rapid Pathogen Screening), is a single-use, noninvasive, disposable test used to detect matrix metalloproteinase-9, a proteolytic enzyme elevated in the tears of dry eye patients.

ting the bull's eye with your DED patients.

Check Under the Lid

The lid wiper is a band of tissue that courses the parallel underside of the lid margin, beneath the eyelashes. Though the lid wiper can be viewed in bright white light, disturbances in the tissue and lid wiper epitheliopathy can best be observed with the aid of vital dyes.[1]

Surprisingly, until 1965 researchers presumed that much of the internal eyelid was in contact with the globe upon the blink.[2] We now know the lid wiper comes into contact with the anterior ocular surface some 3,000 to 15,000 times daily.[3] If lubrication is inadequate at the boundary of the tissue interaction, one or both tissues could eventually become compromised. If only the ocular surface is compromised, DED diagnosis is a simple matter of applying diagnostic dyes and using a slit lamp to view the cornea and conjunctiva.

However, when the lid wiper is affected, an OD could easily overlook it, as this part of the anatomy has been routinely ignored. In any state of dry eye disease, the lid wiper suffers greater trauma and epithelial compromise than the ocular surface.[3] After all, the lid wiper is in apposition to the globe at all times and, with poor lubrication, is constantly susceptible to mechanical trauma. The ocular surface, in contrast, comes in contact with the lid wiper for only a fraction of a second. Interestingly, a damaged lid wiper could result in symptomatology and be the sole clinical sign. Since it is so easy to overlook, researchers speculate that LWE could explain the clinical phenomenon of patients who experience symptoms but lack other more commonly recognized clinical signs.[4]

Grading Lid Wiper Epitheliopathy

1. Grading of horizontal length of fluorescein or lissamine green staining, or both, of the epithelium of the lid wiper.[3]

Horizontal Length of Staining	Grade
<2mm	0
2mm to 4mm	1
5mm to 9mm	2
>10mm	3

2. Grading of sagittal height (width) of fluorescein or lissamine green staining of the epithelium of the lid wiper. [3]

Sagittal Height (Width) of Staining	Grade
<25%	0
25% to 50%	1
50% to 75%	2
>75%	3

3. For fluorescein or lissamine green grading, or both, the above two tables are averaged to determine an overall score.

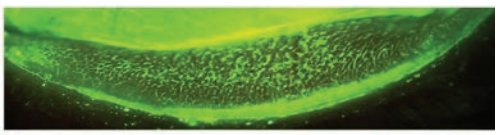
4. Grading of lid wiper epitheliopathy is calculated by taking the average score from step 3. The higher of the final fluorescein and the final lissamine green staining is used as the LWE severity grade.[3]

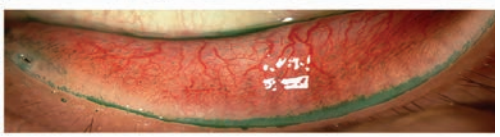
Grading Average	LWE Severity Grade
0	No LWE
0.25 to 1.0	Grade 1 LWE
1.25 to 2.0	Grade 2 LWE
2.25 to 3.0	Grade 3 LWE

LWE is becoming more noteworthy as its association with dry eye and contact lens discomfort is better understood. LWE can occur with or without contact lens wear and correlates with symptoms for both dry eye and contact lens discomfort.[3-5] Researchers believe LWE stems from increased friction between the lid wiper and the ocular surface (or the anterior lens surface in a contact lens wearer) due to inadequate lubrication.[1]

The term epitheliopathy is used to note the uptake of vital dye(s), thereby implying that the tissue is found to have devitalized cells or is in the presence of neighboring cellular apoptosis.[7,8,9] Since 2002, researchers have used a combination of fluorescein, rose bengal and lissamine green. Investigators explain that subjects experience different staining patterns with the various dyes and show optimal presentation when applied in pairs

Photo: Blair Lonsberry, OD





Positive lissamine green conjunctival staining as seen in the above photo can indicate dry eye, whereas the lid margin staining is indicative of lid wiper epitheliopathy

Photos: Jalaiah Varikooty, Centre for Contact Lens Research, University of Waterloo, Ontario.

(e.g., fluorescein paired with rose bengal or lissamine green).[1,5]

A recent study proposed an optimal technique for LWE staining and identification by using fluorescein and lissamine green paper strips moistened with saline and subsequently applied to the eye. Specifically, optimal dye effect is observed when two strips of fluorescein (same procedure for lissamine green) are moistened simultaneously with saline and applied to the eye, two times, one minute apart. With this procedure, there is enough dye and enough time for the tissue effect to be observed. The lid margins can be very well inspected three minutes after dye application. This procedure is repeated one minute later and the lid is visualized three minutes later with the benefit of a Kodak Wratten 12 barrier filter (for use with fluorescein dye; transmitting above 495nm).[1,10,11] If LWE is present, the lid wiper will stain with either dye. The lid wiper staining is assessed and graded from zero to three in two characteristics: the linear area and width of involvement. The higher of the two characteristics is the resultant grade of LWE.[1,3,5]

Tear Osmolarity

Dry eye complaints can bewilder an OD given the variability of their presentations. The discovery of tear osmolarity as a biomarker is extremely valuable.

When the tear fluid's volume is reduced, as is seen with dry eye, the osmolarity increases. A prospective, observational case series of 314 consecutive subjects rated tear osmolarity the single best metric to diagnose and classify DED.[12] One study shows tear osmolarity—when compared with traditional tests for dry eye, such as tear break-up time, Schirmer, corneal staining, conjunctival staining and grading of the meibomian glands—is the only test with a greater than 62% sensitivity (72.8%) and specificity (92%).[12]

Normal tear osmolarity ranges between 275mOsms/L and 307mOsms/L, whereas a reading of 308mOsms/L in one or both eyes or a difference greater than 8mOsm/L between the eyes is noted as abnormal.[12,13]

An unstable tear film is usually more concentrated and is more variable in dry eye patients when compared with healthy patients.[14]

There are a few key points in performing this assessment to obtain ideal results. The patient should direct their gaze upwards and away from the tear collection point (generally from the lacrimal lake upon the lower lid at the lateral canthus). The tip of the instrument pen should not touch the sclera. When there is an obvious lacrimal lake, the tip does not need to touch the lid margin. Instead, the fluid resting on the lower lid will be wicked away towards the pen as the device is placed in the tears.

When an inadequate tear sample is suspected, the tester can slide the pen tip along one third of the lower lid margin to collect a greater sample. Once an adequate sample is collected, the unit can quantify the osmolarity.

Eye care professionals should look towards trends and variability when using osmolarity testing. A single test isn't as valuable as monitoring changes over time. Sporadic changes and variations between the two eyes are far more indicative of a problem than one assessment.

Identifying MMP-9

Dry eye is often accompanied by increased osmolarity of the tear film and inflammation of the ocular surface.[15] Hyperosmolarity contributes to the inflammatory cascade, causing distressed epithelial cells and increased levels of cytokines and matrix metalloproteinase-9 (MMP-9), a proteolytic enzyme.[16] Research shows MMP-9 is elevated in the tears of dry eye patients.[13] These levels correlate well with clinical examination findings. Increased MMP-9 activity increases proportionately with ocular surface dryness and can contribute to damaged corneal epithelial barrier function, increased corneal epithelial desquamation and corneal surface irregularity.[17,18]

InflammaDry (Rapid Pathogen Screening) is a single-use, noninvasive, disposable test to detect MMP-9. It can detect the presence of inflammation in a simple way in-office and within 10 minutes.

Not all patients respond to anti-inflammatory agents, and it is now understood that not all patients with dry eye have significant ocular surface inflammation.[18] Differentiating between patients with and without inflammation using InflammaDry can help direct management in an appropriate direction and can assist in the OD's expectations. The results of InflammaDry can be used to guide care when symptoms and signs are confounding to the eye care provider. For example, identifying patients with underlying inflammation can guide therapeutic recommendations, including artificial tear replacement, punctal occlusion or anti-inflammatory therapeutics such as a short course of corticosteroids, oral doxycycline, or long-term maintenance treatment with cyclosporine or lifitegrast.[13]

To perform the InflammaDry test, a technician collects a small tear sample from the palpebral conjunctiva by dabbing the collector's sampling fleece against the tissue. Adequate saturation is indicated by glistening fleece, turning a pink hue, or both. The device then gets assembled and activated by a buffer solution. After 10 minutes, the device is read in much the same way as a home pregnancy test. A positive test result is noted by a red line on the screen, which is indicative of MMP-9 ≥40ng/mL.[13]

DRY EYE PATIENTS CHALLENGE OPTOMETRISTS BY DELIVERING AMBIGUOUS SYMPTOMS. THESE THREE IN-OFFICE ASSESSMENTS CAN BE EASILY ADDED TO OUR TESTING SEQUENCE.

Dry eye patients challenge optometrists by delivering ambiguous symptoms—sometimes with and sometimes without obvious clinical signs. These three in-office assessments can easily be added to our testing sequence for this patient base. They can help us:

1. Examine a region of the eye that previously went undescribed;
2. Measure a value that previously would have required extensive and expensive laboratory equipment; and
3. Provide direction as to when to tackle ocular inflammation. With a few more steps we can simply and affordably detect and manage most of our dry eye cases. ■

Dr. Lievens is a professor and chief of staff of The Eye Center at Southern College of Optometry.

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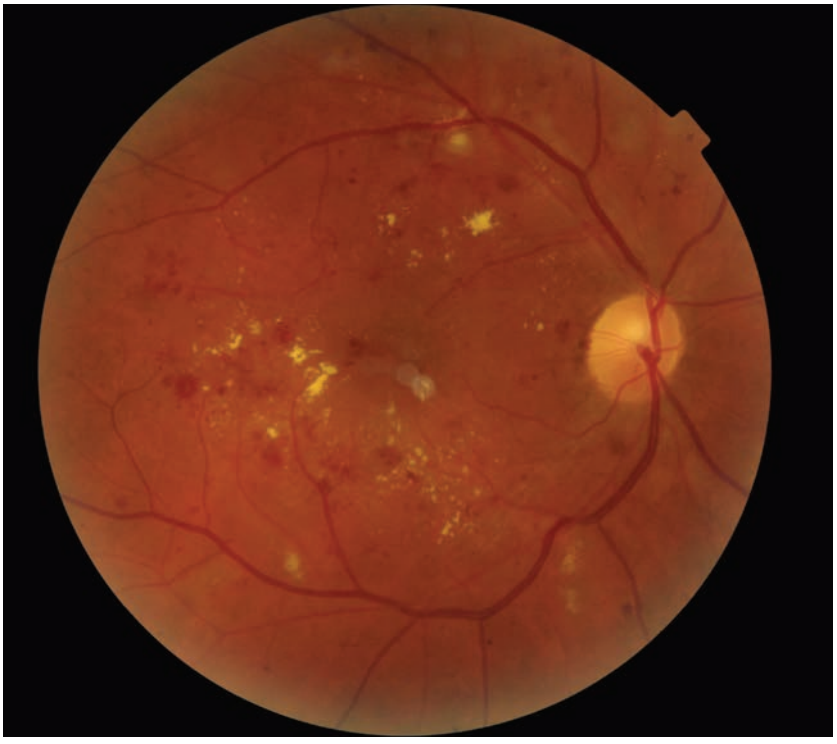
PEER • REVIEWED • RRA • ARTICLE

Teleretinal Imaging for Diabetic Retinopathy

Think remote screening doesn't apply to your practice? Think again.

By Richard J. Zimbalist, OD, and Amber R. Scharnweber, OD

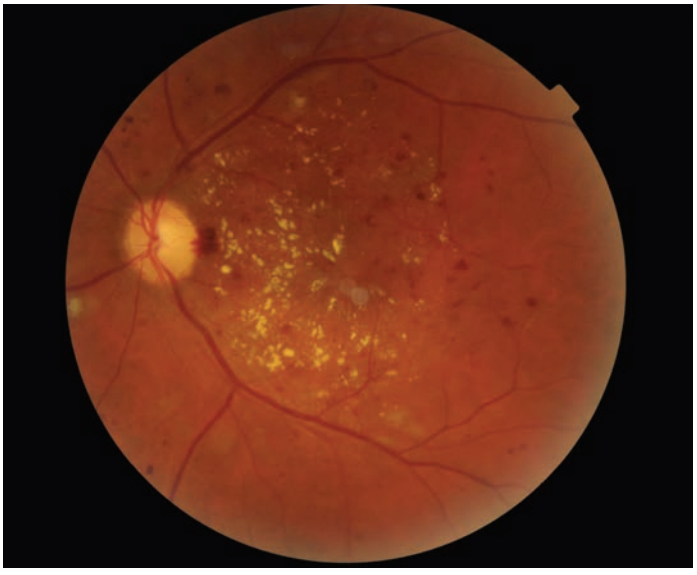
It's safe to assume the majority of you are thinking about skipping this article, believing it has absolutely nothing to do with the way you practice. Well, think again. Let's take a step back for a moment and reflect on some of the technological changes to eye care over the past several years. Optical coherence tomography (OCT) came to the market in the early 2000s and quickly changed the way we diagnose and manage glaucoma and retinal conditions.[1,2] The mid-2000s brought the ever-growing popularity of 1-800-CONTACTS. In 2007, Zenni Optical drastically changed the optical world when it started marketing online glasses at a fraction of the price. Skip ahead to 2013 and Opternative offers online refractive exams that it markets as being "as accurate as a traditional refractive exam performed by an optometrist or ophthalmologist using a phoropter."[3]



This patient's last reported eye exam was in July 2014 at an unknown location. Teleretinal imaging was performed in November 2014 and revealed severe nonproliferative diabetic retinopathy with probable clinically significant macular edema (CSME) in both eyes.

As the landscape of medicine continues to evolve, telehealth is at the forefront. It is not inconceivable to envision a time when telemedicine becomes the norm. In 2015, Kaiser Permanente performed 14 million virtual visits.[4] They predict that virtual visits will actually outnumber traditional in-person visits by 2018.[4] As technology advances and the infrastructure for such examination modalities expands, one can only expect patients to continue to embrace this concept.

Telehealth appointments can improve access, offer cost savings, and provide convenience to patients. Optometry can embrace this by implementing teleretinal screening for diabetic patients and forming strong relationships with local primary care providers. Teleretinal screening programs are already being applied throughout the Veterans Affairs (VA) system, the Joslin Vision Network (JVN), and via web-based applications such as the Eye Picutre Archive Communication System (EyePACS).[5,6] It's time to start thinking of telemedicine like Darwinism: if you don't adapt to our evolving profession, you may not survive.



This image shows the left eye of the patient on page 64. The patient was referred to a retinal specialist where OCT confirmed the presence of CSME OU and widefield angiography demonstrated retinal capillary nonperfusion in the periphery. Panretinal photocoagulation and intravitreal bevacizumab were performed in both eyes at follow-up.

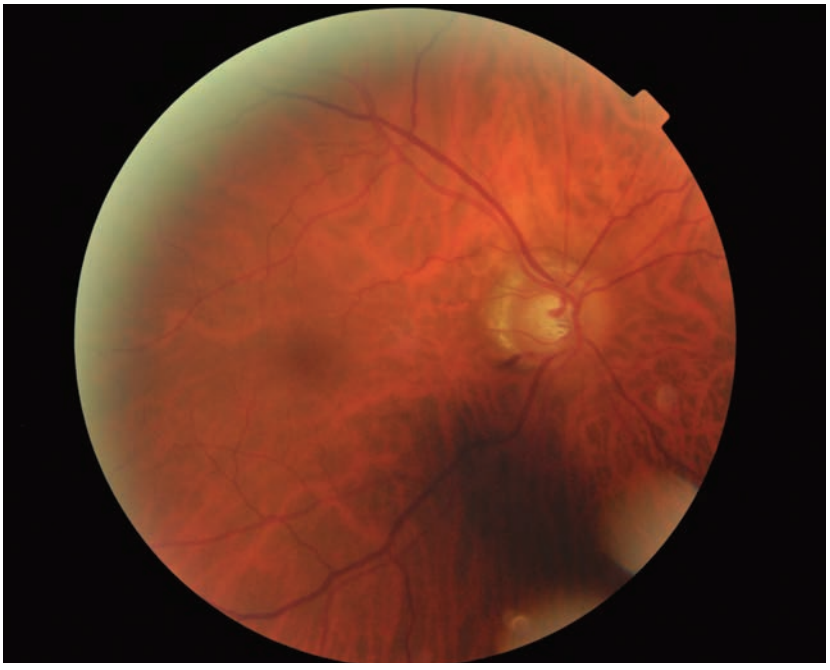
Quick Diagnoses for Prompt Treatments

Diabetes is the leading cause of new blindness in adults in the United States.[5,6] A 2014 CDC report shows more than 29 million Americans have diabetes but 27.8% of patients are undiagnosed.[7] The American Diabetes Association recommends retinal examinations annually. However, despite this recommendation, only 60% of patients with diabetes have their eyes examined regularly.[6,8] But while those studies show worrisome numbers, others suggest hope. For instance, effective blood glucose control and the early evaluation and treatment of diabetic retinopathy can lead to favorable visual outcomes.[9,10]

The VA and Indian Health Service have a disproportionate rate of diabetes patients (25% and 16% to 33.5%, respectively) compared with the general population (9%).[7,11,12] This large cohort of patients requiring timely evaluation of diabetic retinopathy creates a major health care burden. In 2001, Congress recommended the VA collaborate with JVN, a leader in diabetic teleretinal imaging (TRI), to provide teleretinal services as a screening tool for patients with diabetes in the VA.[13] Teleretinal imaging has since become commonplace at VA hospitals and community based outpatient clinics throughout the country. It's an accurate and effective method of assessing the retina for the presence of sight-threatening diabetic retinopathy.[14,15]

The primary goal of TRI is to screen for vision-threatening diabetic retinopathy and allow for prompt treatment. Moreover, TRI can reduce the risk of vision loss from diabetic macular edema and proliferative neovascularization.[14,16] TRI does not take the place of a dilated eye examination. The VA continues to recommend dilated exams every one to two years in coordination with TRI. Teleretinal imaging is particularly valuable in the VA and other hospital systems in which patients must travel long distances for an ocular examination due to the large catchment area with remote and rural locations. Telehealth allows diabetes patients to have their annual retinal screening performed closer to home minimizing travel time—time away from work for the patient and their family, as well as improving convenience for those with mobility issues.

The TRI procedure consists of retinal images of the posterior pole, superotemporal arcade, nasal retina and anterior segment taken by a trained technician.[13,14,17] Images are remotely reviewed by a credentialed reading specialist (optometrist or ophthalmologist) who grades the level of retinopathy based on established guidelines from the Early Treatment Diabetic Retinopathy Study (ETDRS).[10] Patients with levels of mild diabetic retinopathy or greater are referred to the eye clinic for a dilated exam. Incidental ocular findings such as hypertensive retinopathy, retinal emboli, macular degeneration, choroidal nevi and suspicious optic disc cupping also prompt referrals. Those without diabetic retinopathy, or other ocular findings, are typically evaluated by an eye care provider within one to two years.



This teleretinal photograph demonstrates a highly suspicious case of glaucoma in the patient's right eye. Despite the shadow, there is obvious rim thinning inferiorly with an adjacent Drance hemorrhage.

TRI: A Screening Companion

Teleretinal imaging has limitations and is not suitable for all patients. It can be difficult to obtain high quality images on individuals with small pupils, media opacities, and physical and/or mental impairments. Image quality is also highly dependent on the skill level of the imaging technician. Telehealth technicians undergo specific camera training and learn to manipulate the camera with difficult images. Overall, however, the image quality with teleretinal screening is highly successful and several studies boast rates of readable images (i.e., free of artifacts and aberrations) greater than 85%.[14,15,18] Additionally, traditional in-office examinations in conjunction with TRI has helped the VA evaluate an astounding 90% of its diabetes patients on a yearly or biannual basis as medically indicated.[19]

Teleretinal imaging has the capability to overcome many of the obstacles impeding traditional diabetic retinopathy surveillance, including: socioeconomic factors, lack of patient and physician awareness to the importance of annual retinal exams, and geographic challenges.[16] However, barriers exist to implementing TRI in many primary care clinics. One of the largest impediments to widespread adoption of TRI is equipment cost. On average, a nonmydriatic retinal camera costs $15,000 to $25,000.[20] Moreover, there are additional costs of training, IT support, infrastructure and software. Reimbursement for TRI can also be a challenge.

Teleretinal imaging does not take the place of a comprehensive eye examination. There is a common misconception that the use of telemedicine will result in fewer referrals for eye care services. The goal of TRI is to capture the 40% of diabetes sufferers who are noncompliant with annual retinal examinations.[6,8] It has been found that establishing a TRI program with primary care actually generates more workload for the affiliated eye clinic.[19] Several cohort studies have found that approximately 25% to 43% of patients screened through TRI are referred for additional ophthalmic care.[6,14,19] The most common reasons for referral are diabetic eye disease, optic nerve-related disease, lens opacities and macular degeneration.[23-25] Studies have also shown that TRI has excellent sensitivity and specificity when screening for these common ocular conditions.[23-25]

This premise is also closely held by Gerald Selvin, OD, Chief of Optometry, Boston Healthcare System—a national leader in the VA TRI program—who notes, "Teleretinal imaging for diabetes risk management is the tip of the iceberg. In short order we should be screening for many other commonly occurring conditions, including AMD and glaucoma and other conditions as well. Our job is to make sure it is safe, but I think telehealth will occupy a much bigger part of VA and non-VA health care in the future."

Partnering With PCPs

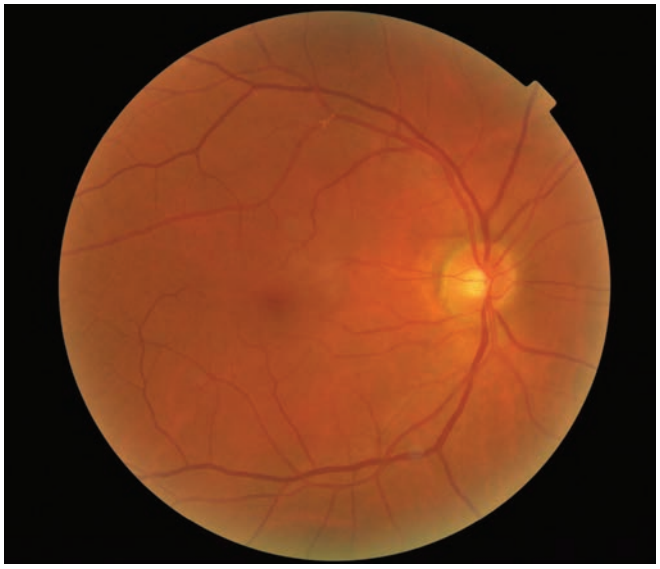
Introducing teleretinal screening at the point of care of the primary care provider improves the detection rate of diabetic retinopathy, and optometry needs to be closely involved.[16,19] Welch Allyn is now marketing a small, portable retinal camera to primary care providers (PCPs) to improve the management of their diabetes patients at a fraction of the cost of a full nonmydriatic camera. The RetinaVue100 (Welch Allyn) imager transfers nonmydriatic images via a secure

network to a board-certified retinal specialist; reports and referral plans are even generated the same day for the PCP.[21] Other companies have developed software to analyze the retinal photographs without the use of a certified reader. It is able to detect diabetes-related retinal anomalies (i.e., microaneurysms, intraretinal hemorrhages, neovascularization, and cotton wool spots) and also perform a serial analysis to identify retinal changes from year to year.[26,27]

Optometry should embrace telehealth services, not as a substitution to face-to-face patient care but as an additional avenue to reach patients and achieve the common goal of minimizing preventable visual impairment. Creating a partnership with our primary care colleagues will generate new referrals and provide diabetic retinal screenings to those unlikely to follow-up independently. Now is the time to strengthen bonds with other physicians. Now is the time to exert yourself in the field. Now is the time to step into 21st century eye care. Now is the time to act. Will you adapt to our ever-changing profession or will you be left behind? ■

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

Dr. Scharnweber practices at the St. Cloud VA Health Care System in St. Cloud, Minn.



This teleretinal image shows a retinal plaque in the superotemporal arcade at the second bifurcation. The patient was examined in clinic and an additional plaque was noted along the inferotemporal artery. A carotid duplex revealed 80% to 99% stenosis in the right internal carotid artery. The patient underwent a successful carotid angioplasty and stenting to relieve the stenosis.

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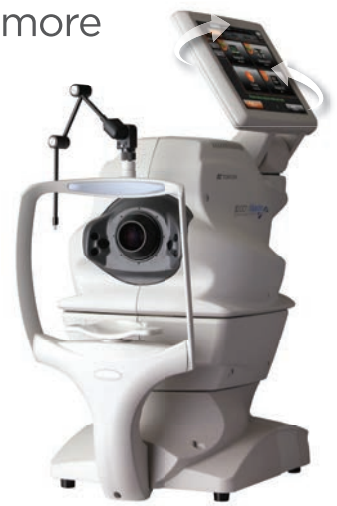


TOPCON

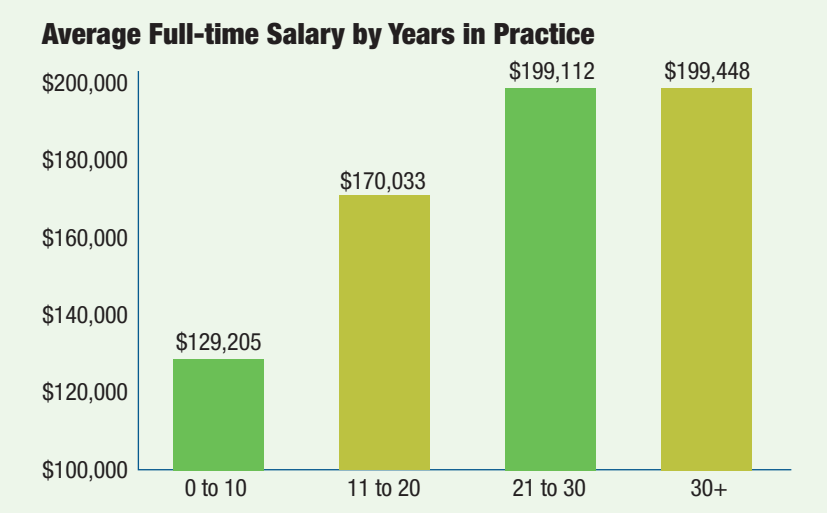
2016 Salary Survey:
THINGS ARE LOOKING UP

They say if you do what you love, you will never work a day in your life. According to our annual survey, that rings true for many optometrists.

By Rebecca Hepp, Managing Editor

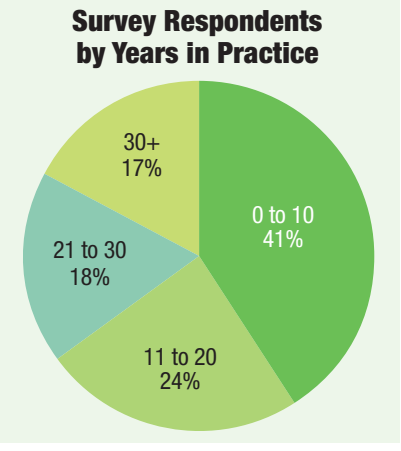
With the New Year practically upon us, it's a good time to look back at 2016 to see how everyone fared, and according to our annual salary survey, optometrists are better off than ever. Nearly 1,000 optometrists responded to this year's survey, and the average income for all participants was $157,650, 9% higher than we reported in our 2015 survey results.

But we asked respondents to share more than just their income, giving us a chance to crunch the numbers and get a closer look at compensation in the field from many angles. Salaries varied significantly depending on practice region, time in practice, gender, practice setting and more. Overall, the highlights from this year's survey indicate a growing field—and one that is well compensated.



Increases Across the Board

Nearly all (90%) respondents work full-time, and they averaged a salary of $163,267, also a 9% increase from 2015. But 2016 was the year to work part-time, as the 10% of survey takers who work part-time had average earnings of $109,851, a whopping 34% higher than was reported by part-timers last year. The higher average seems to be a combination of factors instead of some mass exodus to part-time-hood by all the high-earners. Compared with last year, roughly 3% more have been in the field more than 20 years, and 6% more are practicing in the South—two factors that consistently lead to higher average salaries.



To Employ or Be Employed

Of all the demographics that can affect your pay, whether you are self-employed or not is the most important factor, as it could mean a swing of nearly 60%, according to this year's survey. While the field of full-time respondents was split 50/50 between employed and self-employed, they were compensated quite differently. Self-employed ODs averaged $197,533, while employed ODs reported an average of $125,099—a 58% difference. Many self-employed survey takers also said they enjoyed the flexibility that comes with setting their own schedule.

"I love the fact that I have the flexibility to run my own practice and have a beautiful place to work every day," one survey taker says. "I am able to purchase the most up-to-date equipment and can practice to my highest potential."

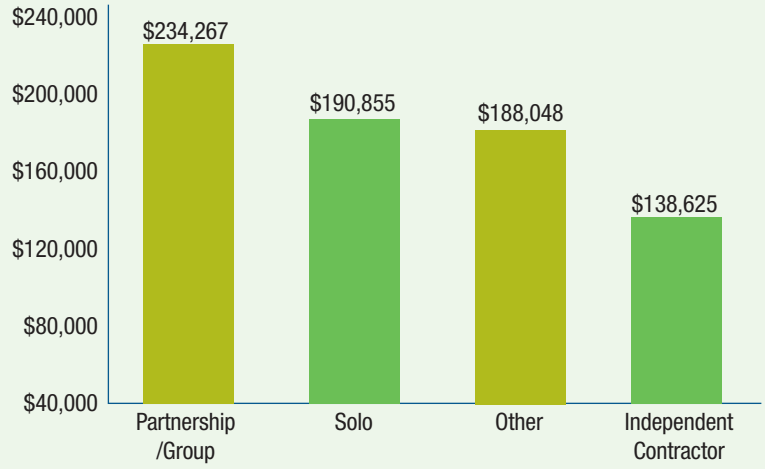
Given the huge pay gap and the lifestyle benefits, it's surprising more ODs aren't opening up shop for themselves. Yet, the challenges that come with being your own boss may make up for the difference in take-home. One survey taker said she made more before she owned her own business, while another said the cost of starting and growing a private practice from scratch took a giant chunk of profit out of his hands. Even after a practice is up and running, the headaches of ownership rarely ease up.

"Reimbursements from insurances are lower, cost of business operation is increased, so therefore income is lower despite efforts to cut costs and pursue marketing options," one respondent said. "Fast service discount stores and online ordering by patients are eating into profits and income."

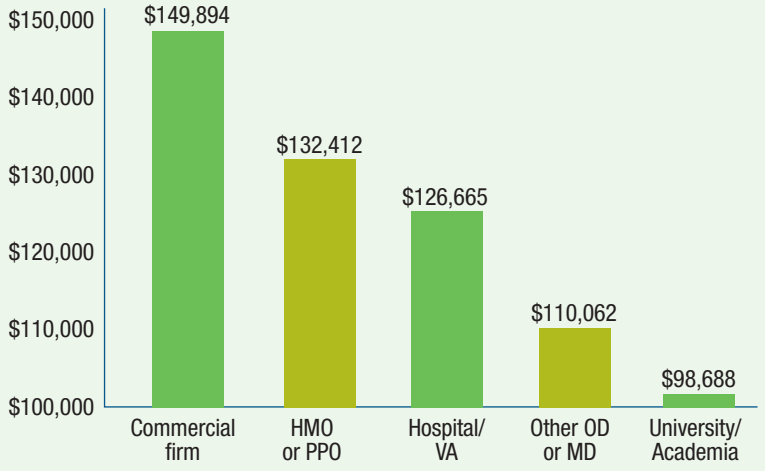
This respondent isn't alone, which is why the ratio of employed to self-employed seems to remain steady from year to year. Yet, some go-getters feel the headaches are worth it and are ready for the challenge next year.

"I'm buying the practice, creating more incentives for staff and techs, and increasing online marketing," one ambitious survey taker says. "I want to get reps more involved in displays and staff training and get more exposure to the community by involvement with health fairs, schools and an open house." At least he knows the hard work will pay off in the end.

Average Full-time Self-employed Salary by Practice Setting



Average Full-time Employed Salary by Practice Setting



The Perks of Your Practice Setting

Perhaps the happy medium between juggling solo practice woes and skating by on an employee salary is joining a partnership or group. At least, the price is right. The 34% of self-employed ODs who work in a partnership or group setting reported the

highest average salary of all, $234,267. A partnership also allows for specialization, which comes with its own perks.

"I own and operate an eye care practice that is partnered with a separate optical business," one respondent said. "Thus, I only do eye care and no product sales, which makes my business a lot more manageable. Since the practice stays very busy and my overhead is low, my income is above average."

No wonder nearly 30% of self-employed respondents said they are very satisfied with their income, compared with only 10% of employed survey takers.

But, for those who want to skip the hassle of self-employment altogether and still want to bring in the big bucks, working for a commercial firm is going to get you the closest with an average salary of $149,894.

While incredibly rewarding, working in academia provides the lowest reported full-time salary of $98,688. Only 2% of respondents work in a university setting, but more than half of them, 63%, are satisfied with their pay, citing better benefits, stability and the lack of weekend and on-call hours.

Time is Money

Another income-booster is time and the clinical experience that comes with it. Like last year, this year's survey showed a continued trend of younger ODs in practice, with 41% of survey takers noting they have been in the field for 10 years or less. Those respondents have some financial rewards to look forward to, as experience pays. Respondents practicing for 20+ years averaged $198,636—54% more than those with at least 10 fewer years under their belt. Still, practitioners in the field for 10 years or less seem to be nearly as satisfied with their income as those with 20+ years of experience, 72% vs. 79%.

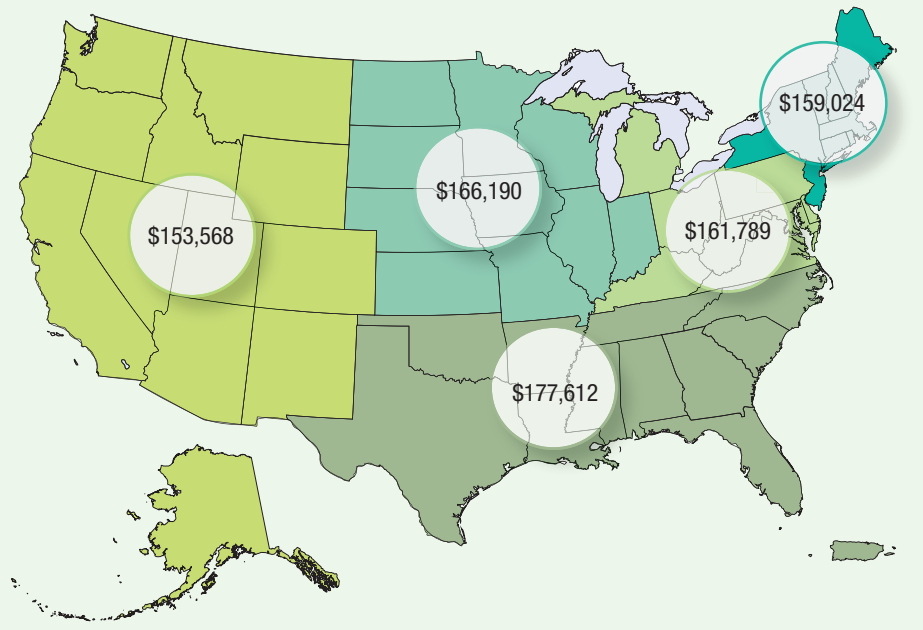
Of course, numbers can be deceiving. More than 70% of practitioners in the field for more than 20 years are self-employed. If you break down the stats, employed ODs practicing for 20+ years only average $144,317, while self-employed ODs in the field for the same time raked in an average of $219,671 this year.

The good news for those striving for that $200k mark? They might get there sooner, as the 41% of respondents practicing for less than a decade reported an average salary 10% higher than last year. And no matter the age, the key to salary satisfaction seems to be the same across all age groups: the ability to save for retirement.

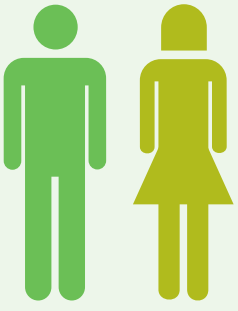
"It is enough for me to pay my expenses and to save for retirement," one respondent with more than 30 years of experience said.

"It provides a nice lifestyle, pays loans, allows for retirement savings and college savings," another survey taker with less than 10 years of experience echoed.

Average Full-time Salary by Region



Average Full-time Salary by Gender



Location, Location, Location

Where you have chosen to live and work could impact your income by as much as 16%, according to this year's numbers. Like last year, respondents in the South reported the highest annual income of $177,612, a nice 7% increase from last year's report.

This year, those in the West reported the lowest average, even in California ($153,684), where cost of living can be remarkably high. Not surprisingly, 34% of California ODs are unsatisfied or very unsatisfied with their pay.

"The current costs of business is too high living in California," according to one respondent. "My salary does not match cost of living in California," another said.

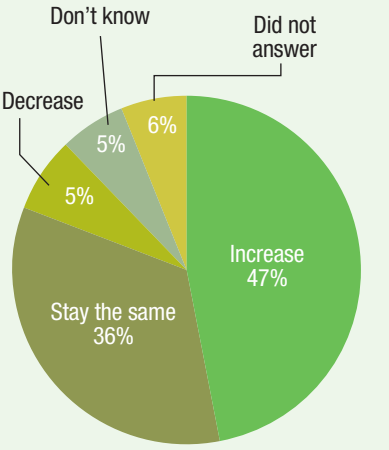
But salaries were on the rise in other parts of the country. Last year's lowest earners, practitioners in the Mid-Atlantic and Lower Great Lakes region boosted their pay this year by about 14%, reporting an average salary hovering in the middle of the pack this year at $161,789: 5% more than those in the West, but 9% less that southern practitioners. ODs in the Midwest also reported a pay raise of about 11% compared with last year.

The Gap is Closing

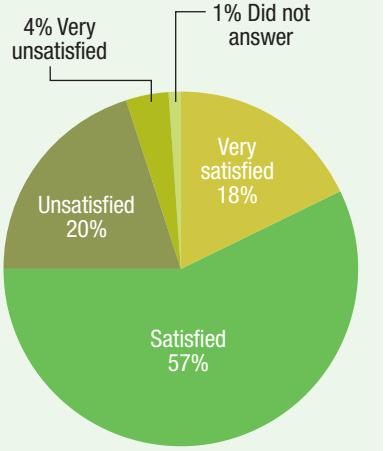
Unfortunately, the gender gap continues to be an issue that impacts compensation. The good news is that it's narrower than ever. Last year, men out-earned women by a shocking 68%. This year, the disparity is down to about 37%.

The gap isn't consistent, however, and varies considerably based on years in practice. Men account for 93% of respondents in the field for more than 30 years, and they still report making, on average, 33% more than their female colleagues with the same experience (the same difference reported for this group in 2015). But it's a different story for ODs in the field for less than 10 years. For one, men are no longer the majority—57% are women—and the pay gap narrows to just 3%. For once, our numbers suggest equal pay is just a few percentage points away for women optometrists entering the field.

Next year, you expect your net income or salary to…



How satisfied are you with your current income?



Gearing up for 2017

Perhaps the narrowing gender gap is just one reason most respondents are hopeful for the year to come. About half of survey takers expect their salary to increase next year, and another 36% don't think it will change at all.

Those gunning for more money have some tricks up their sleeves to make sure it happens, including specialization and improved efficiency.

"Our practice has improved in our EHR ability, allowing me to increase my patient per day volume and my per-patient revenue," one respondent says. "New sources of revenue are improving our dry eye care and our diabetic care with continued MD referral through our clinical reports sent back to the primary care doctor, internist or endocrinologist. Maximizing both medical eye care revenue and profitability with contact lenses, frames and lenses allows our profession to thrive in a managed care environment."

Many, however, will be looking to the tried-and-true revenue boosters, such as seeing more patients, raising fees, hiring more help or taking less time off. For some, 2017 will be the year of the job hunt to find a higher-paying position.

But for those who already have a good thing going, all they need to do is "continue providing memorable customer service," one respondent says. After all, patient experience is everything.

The Bottom Line: A Comfortable Profession

Despite many industry woes such as reduced reimbursements and the threat of online sales, a surprising number of this year's survey takers used the word *comfortable* to describe how they feel about their career and its associated compensation. At the end of the day, 75% of this year's survey takers would have to agree with one respondent's comment, "Optometry is very rewarding financially and professionally." ■

Seeing Stars: How to Diagnose and Manage Migraine

Be prepared to help patients with non-emergent, yet painful and potentially debilitating, migraines. By Bernard H. Blaustein, OD

Headaches frequently localize in and around the eyes, and patients who experience them on a recurring basis often present to you, their primary eye care provider, for help. There are myriad reasons why a patient is in your office discussing their headache, and arriving at the proper diagnosis isn't as straightforward as you might think.

For example, although headaches are commonly associated with eyestrain, or asthenopia, these patients often describe vague feelings of visual discomfort rather than true head pain. Certain ocular pathologies result in chronic recurrent head pain as well, but these diagnoses are usually self-evident by gross observation, or are revealed through biomicroscopy or ophthalmoscopy. While knowing which ocular pathologies present with symptoms of head pain is important, headaches of ocular origin do not represent the vast majority of chronic recurring headaches.

Photo: Welcome Library

This drawing by Joseph Babinksi vividly portrays the visual aura seen by individuals who suffer from migraine.

Recurring headaches are broadly classified as primary or secondary. Secondary headaches are caused by an underlying pathology or structural abnormality and may be indicative of a potentially serious systemic or neurologic entity *(Table 1)*.[2-4] Fortunately, most patients have primary headaches, which are benign and are not caused by an underlying disease or structural problem.[1,2] Although they may cause significant pain and disability, they are not dangerous. The two most common primary recurring headaches are tension-type headache and migraine.[2]

Because migraines are so common, it is important that you be ready to diagnose and manage patients who present with them. This review will help you better understand the etiology and pathophysiology of migraine and will walk you through clinical evaluation and management approaches that are integral to helping this patient population.

Signs and Symptoms

Researchers estimate approximately 10% of the population suffers from some form of migraine disorder.[5,6] Most patients note that their migraines began in the early teens or, less commonly, in childhood.[2] The onset of migraines is not common after age 40 and is rare after the age of 65.[6] In addition, migraines have a strong family tendency, are more common in females, are influenced by hormonal factors and may be induced by certain triggers *(Table 2)*.[7,8]

Table 1. Secondary Headaches Requiring Additional Investigation

Secondary Headache	Possible Etiology
Recurrent headaches in patients younger than age five.	Arteriovenous (AV) malformation
Recurrent headaches in patients older than 50.	Cranial arteritis, mass lesion.
Abrupt-onset, acutely painful headache ("worst headache of my life").	Subarachnoid hemorrhage.
Headaches of recent origin that are becoming increasingly more painful.	Mass lesion; subdural hematoma.
Headaches with concomitant fever, stiff neck, vomiting, cutaneous rash.	Meningitis, encephalitis, Lyme disease, collagen vascular disease.
Headaches associated with non-remitting neurological signs or symptoms such as papilledema, vertigo, seizures, personality changes.	Mass lesion, AV malformation, increased intracranial pressure, encephalitis, meningitis.
Headaches abruptly after bending, coughing, exertion or Valsalva.	Mass lesion, subarachnoid hemorrhage.
Headaches abruptly after head trauma.	Epidural or subdural hematoma.
Headaches associated with systemic cancer or HIV.	Metastasis, opportunistic neurologic infection.
Headaches during pregnancy or postpartum.	Venous sinus thrombosis.

Diagnosis

Migraine is a diagnosis of exclusion; all other potential pathologies related to secondary headache must be eliminated through examination before diagnosing migraine. Patients with migraines usually present with a stereotypical symptom complex. The head pain is:

- Described as throbbing, pulsating and pounding.
- Usually unilateral upon onset but may spread to the other side of the head.

Accompanied by a variety of physical symptoms that often include lack of appetite, nausea, vomiting, diarrhea, cramping, photophobia, hyperacusis leading to sonophobia, excessive sweating (diaphoresis), vertigo and extreme fatigue.[7]

Patients can be quite sick during these episodes and often refer to them as "sick headaches." Following the intense head pain, there may be sustained muscle contractions in the neck and shoulders.

The intense pounding headache, with the attendant constitutional signs and symptoms, usually lasts from three to seven hours, but it may persist for up to 72 hours. A migraine without aura lasting more than 72 hours is known as status migrainosus. Chronic migraine fulfills the diagnostic criteria for typical migraine, but occurs for 15 days or more per month for more than three months.[7,8]

Pathophysiology

Although the underlying mechanisms of migraine are not completely understood, researchers theorize that, subsequent to stress or non-specific stimuli, platelets aggregate and release the neurotransmitter serotonin. In turn, the neurotransmitter causes vasoconstriction of certain blood vessels that supply the base of the brain and other posterior brain vasculature. The regional reduction in blood flow causes a cortical spreading depression (CSD) that proceeds anteriorly. The CSD results in the prodromal signs and symptoms, as well as the aura. Additionally, the reduced blood supply results in local tissue abnormalities such as hypoxia, acidosis and carbon dioxide buildup. The parenchymal arteries dilate in response to increased local tissue demands, activating pain-modulating afferents within the trigeminal nerve and trigeminal vascular system at the base of the brain.

Activation of the trigeminal vascular system by CSD stimulates neurons in dural blood vessels to release plasma proteins and pain-generating substances such as calcitonin gene-related peptide (CGRP), substance P, vasoactive intestinal peptide and neurokinin A. These sterile inflammatory substances produce edema, sensitize cranial pain receptors and lower the pain threshold, leading to the characteristic pounding headache.[9-12]

Before, During and After

About 30% to 40% of migraine patients report a prodromal phase that occurs from 24 to 48 hours prior to the onset of the migraine.[6] This should not be confused with the migraine aura, which is more proximal to the headache. Prodromal signs and symptoms may include aphasia, mood changes, diarrhea, excessive urination fatigue, repetitive yawning, food cravings, increased thirst and problems sleeping and concentrating.

Additionally, many migraine

patients experience a postdromal phase, sometimes called the migraine hangover, which can last several hours and includes malaise, fatigue, poor concentration and altered mood.

Several variations of migraine exist, each with their own characteristic symptoms:

Migraine with aura (classic migraine). Approximately 10% to 15% of migraine sufferers experience auras that precede the onset of the headache.[13] The visual phenomena develop gradually and last less than 60 minutes. They appear as flickering, flashing or scintillating positive scotomas that may surround an area of reduced vision. They may also appear as sparkles or heat waves that have a zigzag appearance. The scintillations often begin near the center of the visual field and expand slowly as they move outward toward the periphery. The light flashes are usually hemianopic and manifest contralateral to the headache. Occasionally, patients experience visual hallucinations such as macropsia or micropsia.

Some patients experience gradually developing, non-visual, sensory and motor auras, including numbness or tingling in the arms and hands, paresthesias of the tongue resulting in slurred speech and, rarely, vertigo and ataxia. Visual and non-visual auras precede the headache by 10 to 20 minutes. It is important to emphasize that both visual and non-visual auras build up gradually and are reversible in most cases. In contradistinction, the neurologic signs and symptoms of stroke occur suddenly and are usually non-reversible.

Migraine without aura (common migraine). Migraine without aura affects approximately 85% of migraine sufferers and is characterized primarily by headache and attendant gastrointestinal signs and symptoms.[13] Patients do not manifest focal, gradually-developing symptoms of aura.

Retinal migraine. Also called ophthalmic or ocular migraine, this is a fairly common cause of transient monocular blindness in young adults.[14,15] This disorder is manifested by recurrent attacks of unilateral visual disturbance or blindness lasting from minutes to one hour. The visual phenomena may be associated with the typical migraine headache or with minimal or no headache (acephalgia). Patients often describe a developing central scotoma that gradually enlarges to produce total unilateral visual loss. Postural changes, exercise and the use of oral contraceptive agents may precipitate attacks.[15] The visual symptoms are totally reversible.[8,15]

Retinal migraine is thought to result from transient vasospasm of the choroidal or retinal arteries. Rarely, when patients with retinal migraine are examined during an attack with visual loss, optic pallor or narrowing of the retinal vessels can be seen.[14] A history of recurrent attacks of transient monocular visual disturbance or blindness, with or without a headache and without other neurologic symptoms, is suggestive of retinal migraine. A personal or family history of migraine confirms the diagnosis along with exclusionary examination and testing. Retinal migraine must be differentiated from ocular or vascular causes of transient monocular blindness such as carotid artery disease and coagulation disorders.

Table 2. Characteristics of the Migraine Patient[7,8]

Childhood or early teens
Strong family history
More common in females
Increase in frequency during menstruation
Increase in frequency with birth control pills
May decrease in frequency during pregnancy
Usually decrease in frequency after menopause
May be triggered by foods containing tyramine, nitrites or monosodium glutamate
May be triggered by mental stress, exertion, dazzling light or high altitude

Aura without headache (acephalgic migraine). During an acephalgic migraine, the patient experiences visual, sensory and motor auras without headache. This phenomenon is characterized by repeated episodes of visual, sensory and motor neurologic symptoms that develop gradually, last approximately one hour or less, are completely reversible. This presentation can occur in patients of any age but often occurs in older patients who have had a history of migraine with aura at an earlier age.[8] Aura without headache must be differentiated from transient ischemic attacks, occipital lobe seizures and temporal lobe seizures.

Migraine variants. This term is not used in the classification of the International Headache Society, but includes those forms of migraine that are not typical of migraine with or without aura.[8] Migraine variants often have significant neurologic manifestations associated with the headache. The neurologic signs and symptoms may precede or occur coincident with the headache and may persist for hours or days after the headache subsides. The headache is often not the most important feature of these rare migraines, and it is usually shorter in duration and less severe than in the typical migraine.

• *Hemiplegic migraine* is an especially rare form of familial migraine that often starts in childhood and disappears in adulthood. These recurrent headaches are associated with unilateral hemiparesis or hemiplegia. Although the neurologic deficit usually resolves before the headache, occasionally the problems persist for days to weeks. Most people with hemiplegic migraine have inherited a dominant gene mutation from a parent who also suffered from the condition.[16-18]

• *Basilar-type migraine*, formerly known as Bickerstaff's syndrome, is, in essence, a migraine with aura, with the symptoms originating from the brainstem and affecting both hemispheres of the brain at the same time. Patients present with fully reversible symptoms of vertebral basilar vascular insufficiency, which usually precede the headache by an hour. However, there is no motor weakness. The most common symptoms are vertigo and dizziness, but symptoms may also include diplopia, hemianopsias, ataxia, tinnitus, decreased hearing, nausea, bilateral paresthesias, syncope and loss of consciousness.[19]

• *Ophthalmoplegic migraine* is a rare disorder that typically starts in childhood. It is characterized by repeated typical migraines associated with paresis of one or more extraocular muscles. Most commonly the oculomotor nerve is affected, but the abducens or trochlear nerves may also be involved. Brain scans do not reveal any intracranial masses; however, several studies show reversible thickening or contrast enhancement of the cisternal portion of the oculomotor nerve on MRI.[20-22] This finding suggests that the ocular muscle palsies caused by oculomotor nerve involvement may be due to recurrent inflammation.

Table 3. Rare Primary Headaches[2,7]

Headache	Clinical Characteristics
Cluster headache	Sudden recurrent headache localized around one eye; headaches occur in clusters of one to five per day for a period of four to six weeks; each headache lasts from 20 to 90 minutes and is accompanied by ipsilateral facial sweating, lacrimation, nasal and conjunctival congestion, miosis and ptosis.
Hemicrania continua	Continuous unilateral headache of moderate intensity lasting up to three months; occasional exacerbations of severe pain accompanied by ipsilateral nasal and conjunctival congestion, ptosis and miosis.
Primary stabbing headache ("ice pick headache")	Recurrent episodes of stabbing pain in or around one eye, lasting several seconds.
Primary cough headache	Sudden headache that lasts several minutes after coughing, sneezing or straining.
Primary exertional headache	Throbbing, pulsatile pain starting during or after exercise.
Primary sex headache	Dull, bilateral headache that starts during sexual activity and becomes worse during orgasm.
Hypnic headache	Short-lasting headache that starts a few hours after falling asleep; may recur several times during the night; often awakens the patient from sleep.

Other rare primary migraines are listed in *Table 3*.

Treatment

Intervention should have a three-pronged approach: general measures with analgesia, abortive measures to prevent an acute attack and prophylactic therapy to prevent recurrence.

General measures include minimizing or avoiding the conditions and agents that trigger migraine attacks. In particular, the patient should attempt to avoid dietary items with vasoactive properties. Refraining from using birth control pills and minimizing mental stress and physical fatigue has also been found to be helpful. Clinicians should also investigate possible sleep dysregulation, such as obstructive sleep apnea, periodic limb movement disorder, insomnia and hypersomnia, since studies suggest migraines may be consequent to, or aggravated by, these disorders.[23-25]

Abortive measures are pharmacological and include both simple analgesics, such as acetaminophen, and nonsteroidal anti-inflammatory drugs (NSAIDs). Simple analgesics, such as acetaminophen, can help by raising the pain threshold. Nonsteroidals such as aspirin, naproxyn, ibuprofen and indomethacin inhibit prostaglandin synthesis and reduce pain induced by the trigeminal-vascular system.[26]

The mainstay abortive pharmaceutical treatment for more severe migraines historically has been ergotamine tartrate, which is a derivative of ergot alkaloids and is a potent vasoconstrictor. It specifically counteracts the dilation of various branches of the trigeminal-vascular system affected in migraine. To be effective, ergotamine tartrate must be administered during the painless, pre-headache phase or soon after onset.

Triptans, first introduced in 1992, are currently considered the first-line treatment for moderate to severe migraines.[28] These serotonin receptor agonists inactivate receptors located on the peripheral trigeminal nerve terminals that supply pain-sensitive vascular meningeal structures. Additionally, these drugs block the neuropeptide-mediated inflammatory response after trigeminal stimulation and may also block transmission in trigeminal neurons.[30,31]

Triptans are contraindicated if the patient is also taking selective serotonin reuptake inhibitors (SSRIs). These drugs cause serotonin to remain in an elevated concentration, and the addition of triptan could result in a serotonin syndrome consisting of anxiety, flushing, paleness, tremors, increased heart rate, fever, diarrhea and vomiting. Additionally, vasoconstrictive agents such as triptans and ergotamine tartrate should be avoided for retinal and basilar-type migraines.[32]

Prophylactic treatment is warranted when migraine attacks are frequent and the patient's lifestyle is disrupted. Among the most effective prophylactic drugs are the beta-adrenergic blockers (e.g., propranolol, atenolol, metoprolol) and the calcium channel blockers (e.g., verapamil, diltiazem, amlodipine, nefedipine).[33-35] The beta-adrenergic blockers inhibit platelet aggregation and reduce the liberation of prostaglandins and other sterile inflammatory substances that induce pain. The calcium channel blockers are thought to prevent intracranial vasoconstriction and the spreading of migrainous cortical depression.

Other useful prophylactic drugs include tricyclic antidepressants (TCAs) such as amitriptyline and certain antiepileptic drugs such as Depakote (valproic acid, Abbott Pharmaceuticals), Topamax (topiramate, Janssen Pharmaceuticals) and Neurontin (gabapentin, Pfizer). TCAs act primarily as serotonin-norepinephrine reuptake inhibitors. The resultant elevated concentration of serotonin and norepinephrine inhibit pain-inducing nerve impulses from the trigeminal-vascular system.[35] Depakote, Topamax and Neurontin are believed to enhance gamma-aminobutryic acid neurotransmission, which may suppress events related to migraine.[36-39]

Injections of Botox (onabotulinum toxin A, Allergan) into the glabellar, frontalis and temporalis muscles have proven to be an effective prophylactic for migraine patients who do not respond to other therapies.[40,41] Botox directly decreases the release of pain mediators, including substance P and CGRP from trigeminal sensory afferent terminals.[42] Additionally, Botox inhibits the release of glutamate, which helps stimulate the release of pain mediators. Finally, Botox inhibits sensitization of central trigeminal vascular neurons. Research shows central sensitization is an integral factor in the development and progression of migraines.[40-42]

The FDA approved the transcutaneous electrical nerve stimulation (TENS) device, the first prophylactic medical device for migraines in adults, in 2014. It fits across the forehead and over the ears and stimulates the trigeminal nerve with a self-adhesive electrode in the center of the forehead. In one study, the device reduced the number of migraine days per month and reduced the amount of abortive medication use.[43]

Conclusion

Properly diagnosing your patients with typical or atypical migraine syndrome calls for a systematic approach. A detailed and relevant history is the most important factor, and careful questioning often reveals a particular headache profile that allows you to make the diagnosis. Ask about onset, time of day, location, frequency, duration, quality and severity, prodromes, precipitating factors, associated symptoms, family history, medical history and response to therapy. A thorough ocular health examination will help you rule out anterior segment, retinal or neuro-ophthalmologic pathologies.

You should be particularly concerned if the history, examination or both suggest the headaches are secondary to an underlying pathology or structural abnormality, in which case an appropriate referral is mandatory. ■

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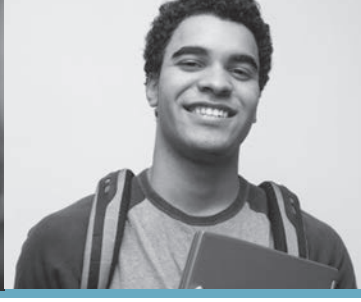

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She Who Snoozes, Loses

A noncompliant glaucoma patient suffers progressive damage, yet is 'surprised' by my concern. **By James L. Fanelli, OD**

A 69-year-old white female presented to the office for an evaluation in June with complaints of visual blur at distance and near. She reported that her vision had changed gradually in the previous six months and relatively symmetrically between the eyes. Prior to entering the room to evaluate the patient, I glanced at her chart and noticed a routing sheet from a visit three years earlier, at which point she was scheduled for a glaucoma progression evaluation, to which she never showed.

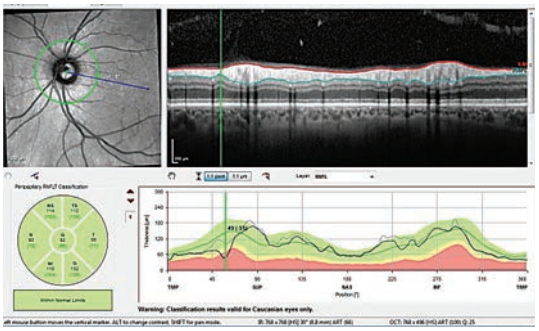

Fig. 1. Close examination of the TSNIT graph in this OCT scan shows a loss of RNFL in the superior temporal sector, in the location marked, of 51µm. Close examination of the optic nerve image shows a subtle but distinctly present wedge defect.

She confirmed the history obtained by my technician that her distance and near vision had gradually changed and that she felt she needed an update to her eyeglass prescription. I prodded her a bit about our last visit and the discussion we had about her being a very strong glaucoma suspect. She implied that she had no real recollection of that conversation, though she admitted that I had said she needed to return shortly after having been seen, but since her eyes "weren't bothering" her, she didn't feel it was necessary.

Diagnostic Data

Her systemic medications included Prilosec (omeprazole, Procter & Gamble) and Vytorin (ezetimibe/simvastatin, Merck) and she reported no allergies to medications. She mentioned that she was prediabetic and, on further probing, I got the impression that her PCP had noted a gradual increase in her A1c and discussed with her the possibility that she may be headed toward medication.

Her entering visual acuities were 20/50 OD and 20/40 OS through hyperopic astigmatic correction. Best-corrected acuities were 20/20 OD and OS and 20/20 OU through an increased hyperopic and astigmatic correction. Pupils were equal, round and responsive to light and accommodation with no afferent pupillary defect. Extraocular movements were full in all positions of gaze.

A slit lamp examination of her anterior segments demonstrated clear corneas, slightly narrowed angles by Van Herick estimation and a quiet anterior chamber in both eyes. Applanation tensions were 29mm Hg OD and 31mm Hg OS at 10:55am. IOPs at her last visit were 26mm Hg OD and OS at 3:00pm. She was dilated in the usual fashion with phenylephrine and tropicamide. Pachymetry readings obtained at the previous visit were 513µm OD and 509µm OS.

Examination of her crystalline lenses was characterized by early nuclear cataracts with cortical spokes in both eyes. Previously, she was noted to have incipient lens changes consistent with her age. The vitreous examination was essentially unremarkable, except for vitreous syneresis. Her cup-to-disc ratios were estimated to be 0.55 x 0.65 OD and 0.55 x 0.75 OS. There was a subtle RNFL wedge defect noted superior temporally in her left eye, which had not been documented previously. Her retinal vascular evaluation was consistent with mild arteriolarsclerotic retinopathy in both eyes. Her macular evaluations were remarkable only for fine RPE granulation, consistent with her age. Her peripheral retinal evaluations were unremarkable.

Given that she was already dilated, we took optic nerve photos and obtained both HRT 3 optic nerve scans, as well as OCT imaging of the RNFL and the macular region in both eyes. Close examination of the disc photos demonstrated minimal change, as the original images were not of the best quality. HRT 3 imaging showed a subtle difference in the neuroretinal rims in the right and left eyes and was consistent with her estimated

cup-to-disc ratio. OCT evaluations demonstrated several interesting findings worthy of further discussion, including a focal loss of the perioptic RNFL in the superotemporal (ST) sector of the left eye, consistent with the fundus findings.

Findings

Compliance (or lack thereof) aside, this case demonstrates the classic findings associated with glaucomatous optic neuropathy as it progresses. Certainly, compliance will need to be addressed, and a treatment plan established that will be conducive to patient compliance. But let's take a look at several important findings in the OCT imaging of her left eye.

We can see the standard RNFL circle scan common to all OCT instruments in the context of glaucoma, along with the TSNIT graphs (*Figure 1*). The images also show the current RNFL scan overlying the baseline RNFL scan obtained three years earlier. Close examination of the TSNIT graph shows a clear and focal loss of RNFL in the ST sector, in the location marked, of 51µm. Close examination of the optic nerve image in the same figure, at the area under observation, shows the subtle but distinctly present wedge defect extending outward. However, many clinicians tend to gravitate their attention to the statistical database comparisons, looking for a quick reference of whether the patient falls within (green), borderline (yellow) or outside (red) normal limits.

Looking at this same scan, in particular at the Garway-Heath sectors, one sees that the parameters of the sector analysis (all green) do fall within normal limits. If not closely examining the details of this scan, one might assume that everything is, in fact, fine, when it truly isn't.

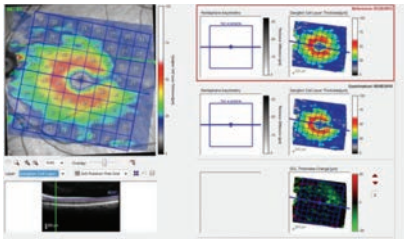

Fig. 2. The image in the lower right shows a decline in the ganglion cell layer in the superior arcuate region.

And herein lies one of the shortfalls of normative databases: they are simply statistical measures of where your patient fits into the normal bell curve distribution of patient parameters. Why are the sectors green if there is a wedge defect and there is a noted TSNIT change? The answer is simple: the TSNIT is looking at each point along the scan reference line, whereas the Garway-Heath sectors are looking at the overall (global) sections of the optic nerve. While there is a decrease in the RNFL in the ST focal area, that change is not enough to statistically alter the Garway-Heath sector analysis; thus each sector, including the ST sector, remains green. I've mentioned on numerous occasions previously the presence of "red disease." Well, this is just the opposite: green wellness.

OCT readings show the ganglion cell layer scans of the macular region in her left eye, with the baseline image from 2013 at the top, and the current scan in the middle (*Figure 2*). The image in the lower right clearly shows a decline in the ganglion cell layer (not ganglion cell complex) in the superior arcuate region—consistent with the RNFL wedge defect seen clinically and on the circle OCT scan—and extending to the temporal horizontal raphe. Not surprisingly, this structural defect produces an arcuate visual field defect inferiorly, with a nasal step, obtained with field testing at a date subsequent to this image.

Finally, *Figure 3* shows a similar printout but of the overall macular retinal thickness, rather than just the ganglion cell layer, as in *Figure 2*. Note here that the retinal thickness change demonstrates negligible difference between the two visits, whereas in *Figure 2*, the ganglion cell layer change was readily identifiable. Again, the explanation is simple: detecting a few microns difference in tissue (total retina) that is 300µm thick vs. a few microns difference in tissue (ganglion cell layer) that is 50µm thick, is proportionally less noticeable. So when we look at the retinal thickness change analysis and see no difference, the tendency might be to assume that there is no substantive difference.

The important point here is that we as clinicians need to be intimately aware of what exactly our instruments of choice are showing us, and consequently, what is the clinical significance, if any, of that information. While the hemisphere asymmetry reports in *Figure 3* show change, the retinal thickness change color map does not. Interestingly, the hemisphere asymmetry report in *Figure 3* correlates well with the ganglion cell thickness change report in *Figure 2*, owing to the loss of ganglion cells in this area.

Encouraging Compliance

So, what does all this mean, other than she got worse? As mentioned above, it means we all need to step back from our instruments for a few minutes and look carefully at the information those instruments are giving us. What exactly does this information mean, in the context of the patient in front of us? As we begin to talk more and more about personalized glaucoma care, we need to realize that the most significant

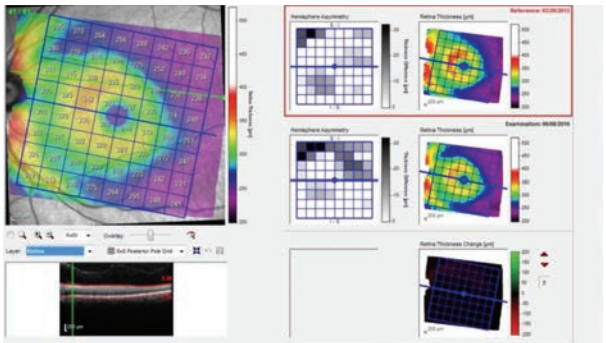

Fig. 3. This OCT scan shows the patient's overall macular retinal thickness.

player in allowing us to provide the personalized care is not this instrument or that instrument; rather, it is our clinical decision making process that can drive us to provide that personalized care every patient deserves.

Now, getting back to the patient at hand and her management: clearly she has glaucoma that has worsened, and she has been noncompliant in the past. How should you proceed? There are several viable options.

The advantage of SLT therapy is evident on two counts. First, SLT tends to be more effective on eyes that are not chronically medicated, and can result in lower IOP than in eyes already medicated. And second, if she continues to be noncompliant, perhaps the SLT will offer her some protection from herself.

Alternatively, medical therapy is also an option, and can possibly result in adequate IOP reduction with a fixed combination drug. But that option presupposes her being compliant. Compliance can be difficult to obtain. The simple truth is that some patients are more compliant than others. The recalcitrant noncomplier is difficult to deal with, and probably should be dismissed from your practice. But in cases like this, where the patient claims to not have known the significance of her condition, maybe we should start with modifying what we say and do to help foster that desired compliance by getting the patient to buy in to their own care. I would certainly rather have a patient who is aware of the need for compliance than simply just being indifferent to me and their condition.

Either way one proceeds (SLT or medical therapy), the patient will still need to be seen for progress evaluations to determine stability. If she complies with those evaluations, there's a good chance she can be stabilized; if not, then she probably will get worse. A patient always has to assume a certain amount of responsibility for their own care. ■

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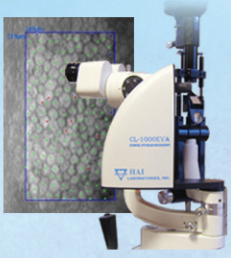

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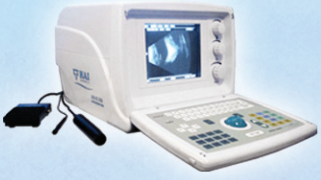

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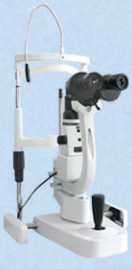

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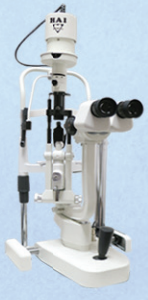

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A New Way to Treat GCA?

The first advancement in 50 years for a blinding disease.

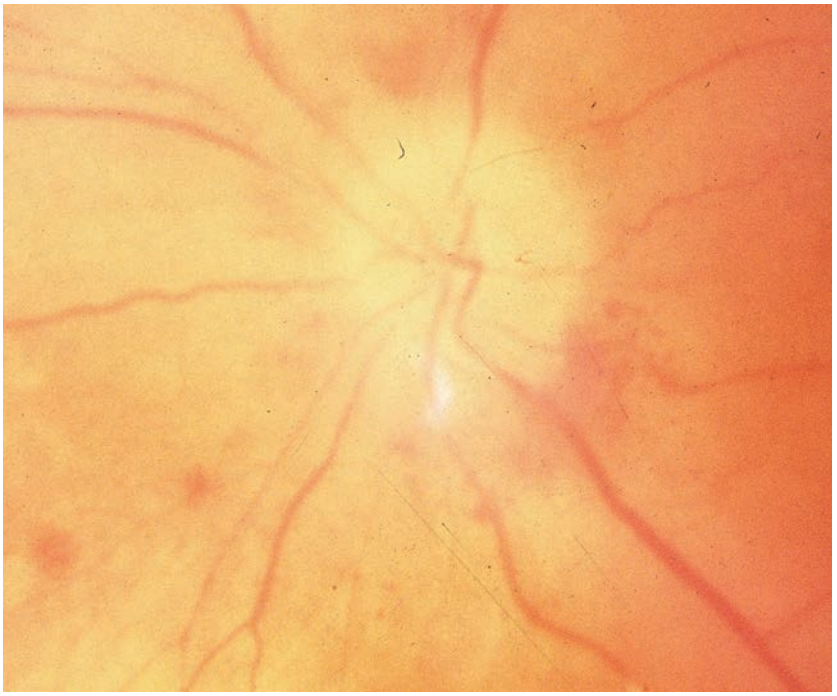
By Joseph W. Sowka, OD, and Alan G. Kabat, OD

One of the true emergencies in all of eye care is a patient suffering vision loss from giant cell arteritis (GCA). Vision loss comes in the form of ischemic optic neuropathy or retinal artery occlusion. In many cases, what begins as unilateral devastating vision reduction quickly progresses to bilaterality and total visual disability for the patient.[1,2] Treatment is high-dose systemic steroids, either oral or inpatient intravenous infusion.

Prevalence

Patients suffering from GCA have a mean age of 71 years at presentation.[3] The prevalence increases with increasing age.[3] This condition is generally considered only after the age of 50. Women are somewhat more likely to develop GCA, and it is much more common in Caucasians.[4]

A multitude of systemic manifestations can signal the presence of GCA, including malaise, weight loss and anorexia, headache (typically in the temporal or occipital region), pulseless and indurated temporal arteries, night sweats, tongue necrosis and oral ulceration, dental abscess, scalp pain and scalp necrosis, jaw claudication when eating, head and neck swelling, anemia, depression, mental disturbance, neck pain, low-grade fever, transient ischemic attack and stroke, proximal myalgia, breast masses, gynecological disorders, malignant disease, persistent flu-like illness, chronic pharyngitis, vertigo, muscle aches, cardiac arrhythmia, congestive heart failure and myocardial infarction.[5-13]



A pale, swollen disc with parapapillary hemorrhages in arteritic anterior ischemic optic neuropathy.

Pathophysiology

GCA is a granulomatous inflammation of medium- and large-sized arteries that have a defined internal and external elastic lamina.[5] The interleukin-6 (IL-6) pathway is upregulated in GCA. There is cellular infiltration of the muscular wall of these vessels by T-lymphocytes, macrophages, histiocytes, plasma cells and multinucleate giant cells.[14,15] The resultant inflammation fragments the vascular walls and leads to collapse of the vessel lumen with resultant ischemia.

Unquestionably, systemic steroids are needed to preserve vision and reduce morbidity and mortality.[16-18] One report recommends that patients with vision loss or other ocular complications receive three to four daily infusions of 250mg of methylprednisolone for three days.[19] For oral administration, the initial prednisolone dose is 60mg/d to 80mg/d. It should be reduced in weekly steps of 5mg

to 10mg until 20mg/d, and by 2.5mg until 10mg/d.[16-18] Dose reduction is 1mg/month below 10mg/d, depending on symptoms and erythrocyte sedimentation rate or C-reactive protein. Suppression of the disease usually takes months to years, leaving patients and physicians to cope with complications of long-term steroid use such as ulcers and gastrointestinal bleeding, osteoporosis, increased risk of heart disease, diabetes, decrease in bone density, increased risk of infections, thin skin, easier bruising and slower wound healing. A steroid-sparing therapy would help patients and physicians greatly.

The Search For New Therapies

One of the greatest difficulties in finding new therapies for GCA involves the direct consequences of inadequately treating the disease in clinical trials. It is well known that steroids are an effective treatment for GCA; hence, it would be medically unethical for a study to directly compare steroids to any medication whose efficacy is unknown or merely theoretical due to the devastating consequences that subjects may experience.[16-19] Thus, all study treatments have to be performed in conjunction with steroids to see if a combination with steroids would be superior to steroids alone. This is the only way the efficacy and role of a newly tested medication can be assessed in a safe and appropriate fashion.

Recently, the FDA granted a breakthrough therapy designation to Actemra (tocilizumab, Genetech) for the treatment of GCA. This is the first innovative therapy for GCA in more than 50 years. The breakthrough therapy designation is designed to speed the development for treatments of serious diseases such as GCA and certain cancers.

THE BREAKTHROUGH THERAPY DESIGNATION IS DESIGNED TO SPEED THE DEVELOPMENT FOR TREATMENTS OF SERIOUS DISEASES, SUCH AS GCA AND CERTAIN CANCERS.

The FDA designation was based upon unpublished results of the GiACTA trial, a multicenter, randomized, double-blind, placebo-controlled study designed to test the ability of tocilizumab, an IL-6 receptor antagonist, to maintain disease remission in patients with GCA.[20] GiACTA data will be submitted for presentation at an upcoming medical conference and to the FDA for approval consideration.

Patients were randomized to receive tocilizumab 162mg weekly injections plus a six-month and 12-month prednisone-taper compared with controls receiving placebo plus similar steroid taper. The preliminary results indicate that patients receiving high dose tocilizumab had superior disease remission at one year compared with the steroid-only taper. Further investigation from this study will attempt to identify the lowest therapeutic dose of prednisone that can be used in patients also using tocilizumab, the amount of tocilizumab needed to induce remission and how long patients stay in remission on this therapy.

GCA is a devastating disease that afflicts approximately 200,000 Americans annually. Steroid treatment, while effective, has its own negative impact on patients. We now stand on the cusp of a new medication which promises to at least reduce the amount of steroids used in treatment with an expected reduction in side effects. Perhaps tocilizumab will be proven through studies to not only be an adjunct to steroids, but even perhaps a replacement. ■

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†Rooms limited. **Additional CE fees if attending both meetings. See website for details: www.reviewofoptometry.com/Events

Corneas in the Crosshairs

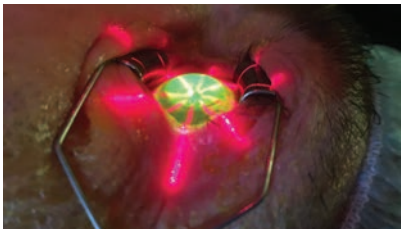
Patients with keratoconus and corneal ectasia have a new treatment option in the United States. **By Derek N. Cunningham, OD, and Walter O. Whitley, OD**

As of April 2016, we finally have an FDA approval for a specific method of corneal collagen crosslinking (Avedro), a procedure indicated for the treatment of progressive keratoconus and corneal ectasia following refractive surgery. Investigators believe the mechanism of action involves the creation of new corneal collagen crosslinks, shortening and thickening the collagen fibrils, and strengthening corneal collagen by activating absorbed riboflavin with an ultraviolet light. This makes the collagen resistant to thinning and deformation.

Who Is Eligible?

Although the FDA study was confined to patients with progressive keratoconus and corneal ectasia following refractive surgery, research shows crosslinking is effective in most types of corneal ectasia.[1] Candidates are identified using visual acuity, retinoscopy and manual keratometry. Ultimate patient candidacy should be confirmed by topography to assess posterior corneal curvature. Patients in the FDA clinical study were at least 14 years old, and no subjects were 65 or older.

Setting the expectations for the procedures is important. Patients need to understand that contact lenses or spectacles will still be required. Healing time may vary per patient, but on average, steepening of the Kmax is observed at one month, followed by flattening through 18 months.



Avedro's KXL system uses laser crosshairs to align the optical head.

How it's Done

A central 9mm corneal epithelial defect is debrided on the cornea followed by one drop of Photrexa Viscous (riboflavin 5'-phosphate in 20% dextran ophthalmic solution, Avedro) applied to the cornea every two minutes for 30 minutes. Full absorption of the riboflavin is confirmed by the presence of yellow flare in the anterior chamber. The eye is then irradiated for 30 continuous minutes at 3mW/cm^2 at 365nm while riboflavin is instilled every two minutes. After the procedure, a bandage contact lens (CL) is placed on the cornea and antibiotic, steroid and, possibly, nonsteroidal anti-inflammatory drops are prescribed.

Postoperative

Due to the removal of the central corneal epithelium, comanaging clinicians can expect a healing course similar to photorefractive keratectomy. One significant difference is increased discomfort the first night after the procedure, which may be due to the light energy absorbed by the corneal nerves. Corneal re-epithelialization will typically take place in three to five days after the procedure, but occasionally takes longer due to the steep nature of the cornea and the apical bearing of the cornea on the bandage CL. Patients should be advised to not rub their eyes ever again, as it can contribute to breaking collagen bonds and the progression of keratoconus. Once the cornea re-epithelializes, the comanaging OD can remove the bandage CL and discontinue the antibiotics and steroids. Normal CL wear can then be resumed.

In 1% to 2% of patients, adverse effects including corneal epithelial defect, corneal edema, corneal opacity and scar continued to be observed for 12 months.[2] The only unique complication seems to be deep corneal haze, but in most cases this was temporary and rarely visually debilitating.

Our experience has shown that, although there is typically a slight thinning of the cornea immediately post-op, ectasia progression is halted in roughly 98% of cases. The FDA study for this procedure also shows significant improvement in corneal flattening as well as BCVA and UVCA when compared with sham treatment groups at one year.[3] ■

To see a narrated video of this procedure, visit www.reviewofoptometry.com, or scan the QR code.

1. Bayraktar S, Cebeci Z, Oray M, Alparslan N. Corneal collagen cross-linking in pellucid marginal degeneration: 2 patients, 4 eyes. Case Reports in Ophthalmological Medicine. 2015;2015:840687.
2. Avedro. Package Insert.
3. Hersh PS, Greenstein SA, Fry KL. Corneal collagen crosslinking for keratoconus and corneal ectasia: One-year results. J Cataract Refract Surgery. 2011;37(1):149-60.

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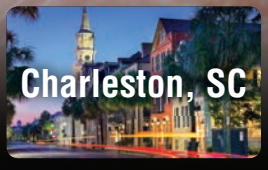

March 24-26, 2017
Charleston Marriott
Program Chair:
Paul Karpecki, OD

Hands-on Workshops†

April 20-23, 2017
Joint Meeting: NT&T/OCCRS
San Diego Marriott Del Mar
Program Chair:
Paul Karpecki, OD

June 8-11, 2017
Disney's Yacht & Beach Club
Program Chair:
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November 3-5, 2017
Loews Philadelphia Hotel
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†Workshops not available for "Aspen, CO" meeting. See Review website for any meeting schedule changes or updates.

Product **Review**

Contact Lenses

Air Optix plus HydraGlyde

Alcon has a new monthly replacement silicone hydrogel lens option to offer your patients, called Air Optix plus HydraGlyde. Two unique attributes—a wetting agent and a protective outer layer—help to keep it wet and to resist lipid deposition, which should enhance comfort, according to the company.

The company also recently shared two additional updates to its contact lens portfolio:

- The AquaComfort Plus daily contact lens will now be sold with a fresh, new design and come with illustrated preparation, insertion and removal instructions, a helpline phone number and email address and the Alcon Dailies website address.
- Finally, the Dailies Total1 line of multifocals offers expanded parameters. It now ranges from +6.00D to -10.00D and comes in three add powers, Alcon says.

Visit www.alcon.com.

Contact Lens Recycling Program

Bausch + Lomb now gives your patients the ability to help the Earth with a new contact lens recycling campaign called #OneByOne, in partnership with the recycling company TerraCycle. With the #OneByOne campaign, patients can recycle all of their lenses and blister packs. Typically, these materials are excluded by recycling facilities when mixed with other items, B+L says.

Visit www.bausch.com/our-company/one-by-one-recycling.

Diagnostic Technology

D-Eye New iPhone App Version and Bumper Design

D-Eye now offers a new version of their iPhone app and quick release bumper design, compatible with iPhone 5 and all newer iPhone models.

New features in the D-Eye App 2.0 include additional image editing capabilities and an image mask that allows users to highlight the posterior pole in real time. The new app update also includes auto focus calibration for myopic, hyperopic and emmetropic eyes, to save exam time, according to the company.

Visit www.d-eyecare.com.

ImageNet Connect

Topcon, in partnership with newly aqcuired company ifa, now participates in the vendor-neutral Picture Archive Communication System for data sharing. ImageNet Connect includes the following, according to Topcon:

- Vendor-neutral archive compatible with more than 450 devices.
- All clinical information available on one program.
- Efficient workflow management.
- Intelligent patient flow to track progress from check-in to check-out.
- Statistics to optimize practice workflow and minimize patient waiting time.
- Integrtion of test interpretations, clinical remarks, diagnosis and treatment plans.

For more information, visit www.topcon.co.jp/en.

Pharmaceuticals

Multidose Restasis Bottle

Practitioners can now offer patients the choice of a multidose bottle in place of single-use vials. A unidirectional valve and air filter eliminates the need for a preservative, according to Allergan.

Visit www.allergan.com for more.

New NSAID Available

BromSite (bromfenac ophthalmic solution) is now available in the United States. It is the first NSAID approved to prevent ocular pain and treat ocular inflammation following cataract surgery, according to Sun Pharma.

More info available at www.bromsite.com.

Surgical Comanagement

Xen Gel Stent

Allergan will launch the newly approved Xen glaucoma treatment system in early 2017, a stent 6mm long and about the width of a human hair. It is injected through a small self-sealing corneal incision using a preloaded injector similar to that of an IOL. Xen is indicated for the management of refractory glaucomas, cases of previous surgical failure and glacuoma patients unresponsive to maximum tolerated medical therapy, Allergan says.

Find out more at www.allergan.com. ■

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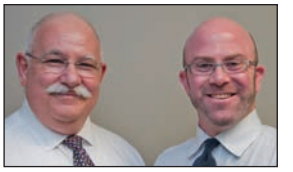
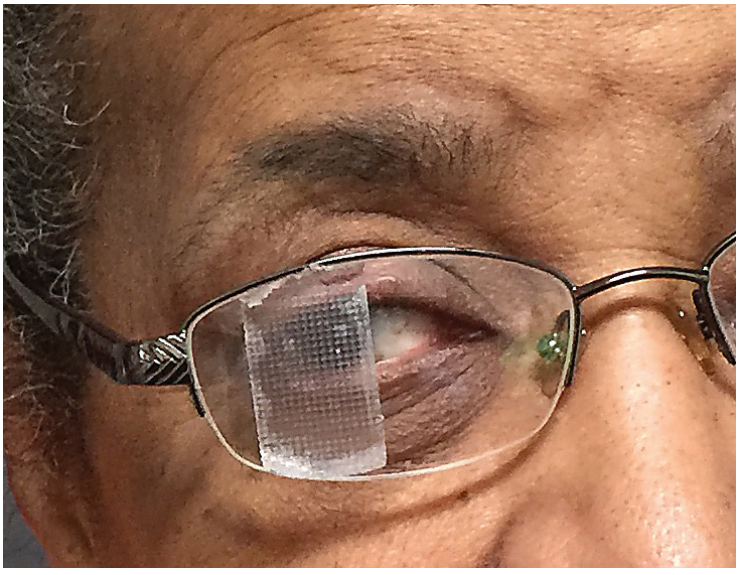


Little Occlusion Goes a Long Way

Often, the simplest method of improving diplopia immediately following stroke is the most effective. **By Marc B. Taub, OD, MS, and Paul Harris, OD**

One of the most common ocular findings following an acquired brain injury, such as stroke, is diplopia. Not only is it disorienting for the patient, it also interferes with the rehabilitation process. Considering improvement immediately following a stroke is essential to the patient's long-term overall life outcome, eliminating the diplopia is a vital first step. Before we made diplopia a priority at our rehabilitation facilities, it was common to find patients doing rehab therapies while still wearing the dreaded black patch. Although patching treatment does the trick to eliminate double vision, it inhibits the brain's ability restore the visual system to fusion. Luckily, far superior options are available.



Shown here is the patient with tape placed in a variation of the initial proper position, with the tape placed on the right half of the right lens.

Case Example

A 71-year-old African American female presented to a local emergency room with complaints of double vision. Radiologic studies confirmed a stroke. At the time of our examination, she was undergoing rehabilitation to improve activities of daily living. She reported double vision that was horizontal, vertical or both, and variable. She had been self-patching to eliminate the double vision. She was wearing a progressive bifocal.

Distance visual acuities were taken at six feet—standard for a visit at a rehabilitation center due to the patient's prone positioning—and measured 20/20 OD and 20/50 OS. Because the patient was tilting her head while lying in bed during the exam, we surmised she was looking through the incorrect portion of the PAL. Pupils were normal with no APD. Confrontation fields were full-to-finger count. Cover test showed a constant right exo/hypotropia in the patient's right eye. Eye movements showed the patient's left eye was restricted to the right and her right eye restricted in upgaze; movement was of poor quality with numerous fixation losses. The anterior and posterior segment evaluation revealed no issues.

Achieving Fusion: Keep it Simple

Although we typically start with prism to attempt fusion, this patient's variable double vision made finding the right power and direction of prism impossible. With our next option, occlusion, we sought to implement an important overarching concept: use the least amount of occlusion possible to reduce or eliminate the double vision. For example, we have used binasal occlusion, spot occlusion and monocular full lens occlusion in the past. For this patient, simply putting a strip of tape down the center of the right lens eliminated her double vision as long as she turned her face toward objects she was trying to view.

In addition to educating the patient's therapists on this simple therapy, we also instructed them on eye stretches to keep all of the muscles from suffering contracture. We want to get the eyes moving in all directions as far as possible. When an eye shifts to a new

location in the head for too long, the muscle that is dominating can actually shorten in length. This makes recovery harder should the affected muscle begin to come back to life. So, eye stretches help to keep the system as mobile and adaptable as possible. The patient was instructed to follow up with us upon release from the rehabilitation center.

A Bump in the Road to Recovery

A few days later, the patient showed up again on our patient list at the rehabilitation hospital. Upon entering the room, we observed the patient without her glasses, squinting her right eye shut. When asked where the glasses were, she pointed to the table. In spite of Dr. Taub putting the tape on the right lens, it was now clearly down the center of the left lens. A number of family members were present, and one said that our patient's daughter, who was not present, had moved the tape.

Although the family was hoping for prism, extensive testing showed that 25D of base-in prism only gave her single vision in primary gaze in one specific direction of gaze and at one specific distance in space. Her non-concomitants (variable degrees of turn in different positions of gaze) made it impossible for her to see within a large volume of space. She also complained about the distortions caused by the Fresnel prism of that magnitude.

We decided to return to a variation of Dr. Taub's treatment plan and put tape on the right half of the right lens. The variation from the initial placement directly in front of her right eye most likely was due to some changes in her eye posture and restrictions due to minor recovery between visits. This made it much easier for her to find an object and rotate her head to just the right place to tuck the second image behind the tape.

This time, the entire family, except for the enterprising daughter, was present to learn about the patient's treatment plan. (Dr. Harris spoke to the daughter over the phone the next day.)

Having members of the rehab team and the patient's family all on the same page was crucial for this patient's long-term success. Since her discharge from the rehab hospital, the patient has been coming regularly for vision therapy.

This case shows a little bit of tape can go a long way—but it needs to be in the right place, and patients, staff and even family members need to be educated on this simple but effective modality. ■

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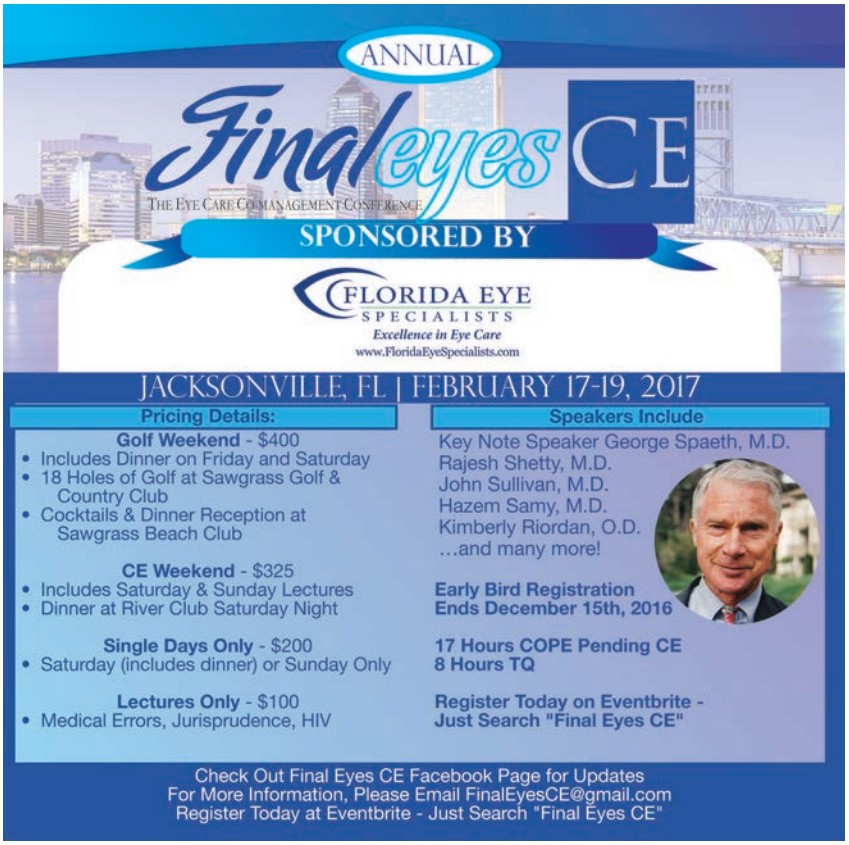

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Diagnostic **Quiz**

Getting Off Track

By Andrew S. Gurwood, OD

History

An 11-year-old Caucasian male reported to the office for a routine eye examination. He explained that his doctor wanted him to get his first eye exam and that recently—within the last month—he noticed some blur in his left eye.

His systemic and ocular histories were unremarkable and he denied allergies of any kind.

Diagnostic Data

His best-corrected entering visual acuities were 20/20 OD and 20/25 OS at distance and near. His external examination was normal with no evidence of afferent pupillary defect.

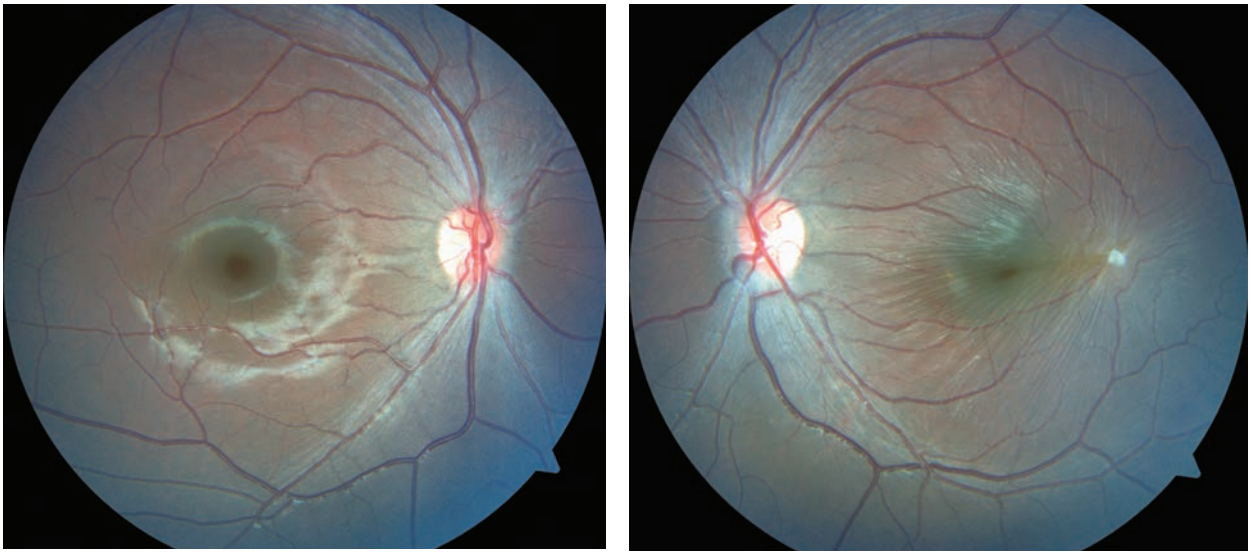
The biomicroscopic examination of the anterior segment was normal in every way. IOP by Goldmann applanation tonometry measured 15mm Hg OU.

The pertinent fundus finding are demonstrated in the photographs.

Your Diagnosis

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

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



Do these fundus photographs provide any explanation for our patient's blurred vision?

Next Month in the Mag

In January, *Review of Optometry* will present its annual corneal disease report.

Topics include:

- *Develop the Skills for Managing Corneal Erosion*
- *A Step-By-Step Guide to Performing EDTA Scrape for Patients with Band Keratopathy*
- *Amniotic Membranes: Pearls for the OD*
- *Foreign Body Removal: Principles for Success*

Also in this issue:

- *Gear Up For Glaucoma Screening—It's More Prevalent Than You May Realize*
- *Arm Yourself for Dry AMD* (earn 2 CE credits).

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*When the ECP followed the fitting guide for the 3-Zone Progressive™ Design of PureVision®2 for Presbyopia lens.

REFERENCES: 1. Data on file. Bausch & Lomb Incorporated. Rochester, NY; 2013. **2.** Data on file. Bausch & Lomb Incorporated. Rochester, NY; 2015. **3.** Thirty-nine ECPs (from 10 countries) refitted 422 existing soft contact lens wearing presbyopes into PureVision®2 Presbyopia lenses. Patients returned for follow-up visits after 1-2 weeks. ECP assessment of lens performance including ease of fit, and patient satisfaction with lenses in real-world conditions, were measured using a 6-point agreement survey.

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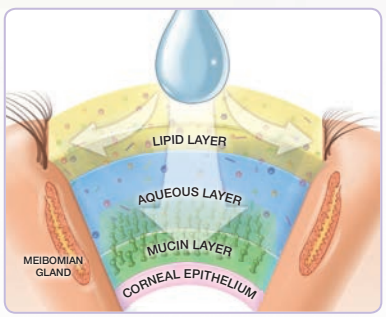

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References: 1. Akpek EK, Smith RA. Overview of age-related ocular conditions. *Am J Manag Care.* 2013;19 (5 suppl):S67-S75. **2.** Korb DR, Blackie CA, Meadows DL, Christensen M, Tudor M. Evaluation of extended tear stability by two emulsion based artificial tears. Poster presented at: 6th International Conference on the Tear Film and Ocular Surface: Basic Science and Clinical Relevance; September 22-25, 2010; Florence, Italy.

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