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June 15, 2015

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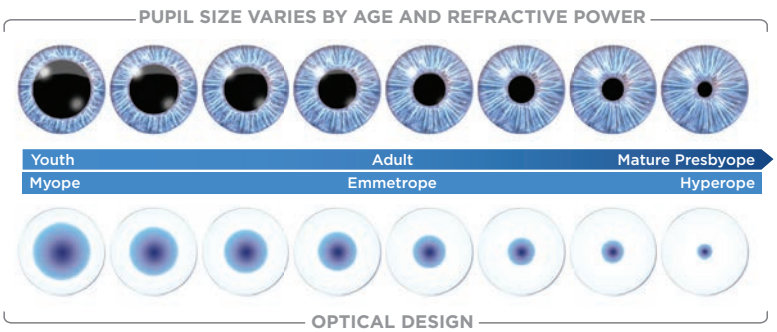

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VOL. 152 NO. 6 ■ JUNE 15, 2015

IN THE NEWS

A **new bill**, which recently passed in California's Senate with a nearly unanimous vote, could allow California optometrists to perform **post-cataract surgery laser procedures** for glaucoma patients, **eyelid lesion removal**, and some **vaccinations** for adults. SB 622, authored by Sen. Ed Hernandez (D-West Covina), who is also a practicing optometrist, is now headed to the Assembly for a vote.

Researchers from the University of Nevada School of Medicine in Reno have discovered that nearly half of the **genes dysregulated** in the **strabismic medial rectus muscle** are also **schizophrenia biomarkers**. The study, presented at the annual meeting of the Association for Research in Vision and Ophthalmology in Denver, suggests that imbalances in key signaling molecules are related in the development of both diseases. Researchers hope their findings will help with the development of a tool to assess the risk of a strabismic child developing schizophrenia.

A new study published online by *JAMA Ophthalmology* found that taking **metformin** was associated with reduced risk of developing **open-angle glaucoma in diabetics**. Researchers from the University of Michigan, Ann Arbor, studied data from 150,016 patients with diabetes and found that patients prescribed greater than 1,110g of metformin in two years had a 25% reduced risk of open-angle glaucoma compared with those who took no metformin. For diabetics, taking 2g of metformin per day for two years would result in a **20.8% reduction in risk** of open-angle glaucoma, they concluded.

New Insights into The Treatment of Diabetic Retinopathy

Blocking a newly identified protein could help boost the effectiveness of anti-VEGF injections for these patients.

By Rebecca Hepp, Senior Associate Editor

Researchers at The Johns Hopkins University and the University of Maryland have discovered that targeting a protein called angiopoietin-like 4 (ANGPTL4) along with VEGF may be the key to improving treatment effectiveness for patients with diabetic retinopathy (DR). New studies document the success of anti-VEGF treatment in delaying the development of proliferative diabetic retinopathy (PDR), but it doesn't work for all patients—which prompted continued research.

To better understand the varying success of anti-VEGF treatment, investigators tested levels of VEGF in eye fluid samples from healthy people, diabetics without diabetic retinopathy and those with varying degrees of DR. They found that some of the fluid collected from patients with PDR had less VEGF than the fluid from healthy participants; however, ANGPTL4 was increased in the fluid of PDR patients, independent of VEGF levels.

Guided by these findings, lead author Savalan Babapoor-Farrokhran, MD, and colleagues took a closer look at ANGPTL4's involvement in PDR pathogenesis. They found that blocking the action of both ANGPTL4 and VEGF in the fluid from the eyes of PDR patients significantly reduced blood vessel growth.

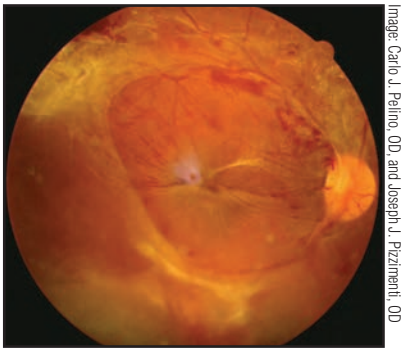

Image: Carlo J. Pelino, OD, and Joseph J. Pizzimenti, OD

This patient exhibited proliferative diabetic retinopathy.

Researchers hope a new drug can be found that inhibits ANGPTL4 action; it, combined with the anti-VEGF drugs, could prevent many cases of PDR. Encouraged by their findings, the investigators are now exploring whether ANGPTL4 might also play a role in other eye diseases such as macular degeneration.

Babapoor-Farrokhran S, Jee K, Puchner B, et al. Angiopoietin-like 4 is a potent angiogenic factor and a novel therapeutic target for patients with proliferative diabetic retinopathy. Proceedings of the National Academy of Sciences. 2015 May 26. [Epub ahead of print].

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Calcium Supplements Associated with Increased Prevalence of AMD

A new study suggests taking too much supplementary calcium, especially for older patients, could increase the odds of a diagnosis of age-related macular degeneration (AMD).

Researchers from the University of California, San Francisco, (UCSF) evaluated 3,191 study participants older than 40 years for the presence of AMD by fundus photography and interviewed them about dietary supplement intake. The results revealed those who reported consuming more than 800mg of supplementary calcium a day were 85% more likely to be diagnosed with AMD than those who did not report taking a calcium supplement. The researchers note in the study that 800mg is below the recommended total daily intake of calcium for men and women in the United States.

The mean age of those diagnosed with AMD was 67.2, while the mean age for those without AMD was 55.8—leading investigators to speculate that the stronger association in older participants may be due to the longer duration of calcium supplementation or the greater tendency for calcium to cause harm in terms of AMD risk in patients more advanced in age.

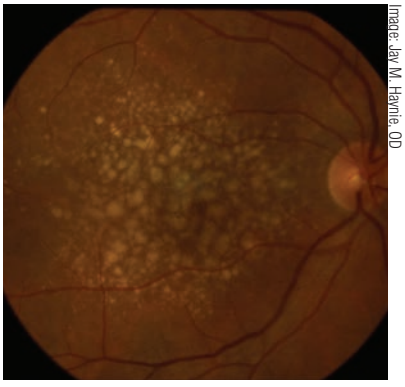

Image: Jay M. Haynie, OD

Soft drusen observed in a patient with early age-related macular degeneration.

While the cross-sectional study, published in *JAMA Ophthalmology*, did not allow researchers to identify a dose-response association between calcium intake and AMD, "the findings suggest that there is a threshold of calcium supplementation above which there are increased odds of AMD," lead author Caitlin Kakigi, a UCSF medical student, says.

"Further longitudinal analyses are needed to understand the relationship between the incidence of AMD and varying levels of calcium intake," Ms. Kakigi says. "However, this study supports the general idea that physicians should refrain from recommending high levels of calcium to patients who are at high risk for AMD and otherwise have no medical indication for calcium supplementation."

Kakigi CL, Singh K, Wang SY, et al. Self-reported calcium supplementation and age-related macular degeneration. JAMA Ophthalmol. 2015 Apr 9. [Epub ahead of print].

Dyslexia Study Raises Ire of Eye Docs

Is dyslexia simply a result of a visual disorder? The answer, once again, is 'no,' according to a recent study published in the journal *Pediatrics*. For most of the optometric community, the study simply reiterates widely accepted science. But for some, the study got their hackles up.

The study tested more than 5,800 children and found 3% with dyslexia, 80% of which had normal eye function.

Cathy Wittman, OD, an optometrist in Lubbock, Texas, worries this article may steer parents away from seeking eye care for their children. "I do not believe vision problems cause dyslexia, but it makes sense that if dyslexia is a neurological condition (which it is), that vision would also be affected because every lobe in the brain is associated with vision, and there are at least 35 areas of the brain that are involved in vision with 305 connections between them" she says.

Steven J. Gallop, OD, an optometrist specializing in behavioral optometry in Broomall, Pa., believes optometry is the victim of

(Continued on page 8)

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Dyslexia, Vision Disoder Not Linked

(Continued from page 6)
a strawman attack launched by researchers dismissive of behavioral vision care professionals, according to a recent blog post. "People in the medical community find it necessary to attack behavioral optometry and vision therapy. They start off with a made-up premise, such as 'eye problems cause dyslexia,' and imply that behavioral optometry says that eye problems cause dyslexia or that vision therapy can cure dyslexia. I have never heard anyone in my profession even come close to such statements," he wrote.

Dr. Gallop also says many of his pediatric patients were misdiagnosed with dyslexia or ADD when they have simple tracking or eye teaming problems.

According to a recent study published in the *Journal of Vision*, while dyslexia "is considered a phonological processing impairment that might be linked to a cross-modal, letter-to-speech sound integration deficit," new theories suggest that "mild deficits in low-level visual and auditory processing can lead to developmental dyslexia."

Research into dyslexia is ongoing. Behavioral optometry addresses visual processing issues, and vision therapy helps many read and learn more easily, according to Dr. Gallop.

Never Lose Your Reading Glasses

To solve his patients' frustrations with forgetting their reading glasses, Eric Radzwill, OD, who practices in Fort Lauderdale, Fla., invented reading glasses that magnetically attach to an iPhone 6 case.

They have scratch resistant lenses and come in +1.00, +1.50, +2.00 and +2.50 powers.

Lens Wearers Have More *Demodex*

Contact lens wearers have greater numbers of *Demodex* than nonwearers, according to a study from optometric researchers at the University of New South Wales in Australia.

The study findings, published in June's *Optometry & Vision Science*, did not uncover why contact lens wearers have more *Demodex* in their lashes. But the investigators speculate that contact lens wear—which is associated with colonization of lid margins by *Staphylococcus epidermidis*, *Propionibacterium acnes*, *Corynebacteria* and *Staphylococcus aureus*—may provide a more favorable environment for *Demodex* mites to proliferate.

"It is possible that the contact lens may act as a vector for microorganisms that offer an environment more favorable to accumulate excessive bacteria, which may further lead toward *Demodex* infestation," the authors wrote.

Image: Elyse L. Chaglasian, OD

Collarettes are a predictable sign of *Demodex*.

The study enrolled 40 participants, half of whom were contact lens wearers and half were not. Using confocal microscopy, investigators found *Demodex* in 90% of lens wearers and in 65% of nonwearers. Conventional light microscopy detected lower numbers in both groups, although contact lens wearers still had more *Demodex* (70%) than did nonwearers (60%).

As expected, the number of *Demodex* tended to increase with participants' ages. But, surprisingly, *Demodex* did not appear to affect ocular comfort or cause any clinical signs. The investigators say further research is needed to confirm these findings.

Yet, they add, "manufacturers of contact lens care products may want to formulate future products with these results in mind." ■

Jalbert I, Rejab S. Increased numbers of *Demodex* in contact lens wearers. Optom Vis Sci. 2015 Jun;92(6):671-8.

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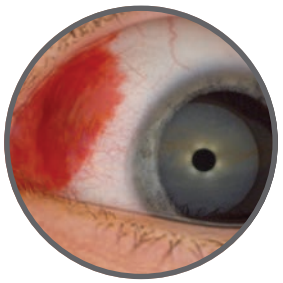

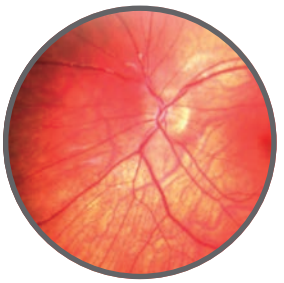

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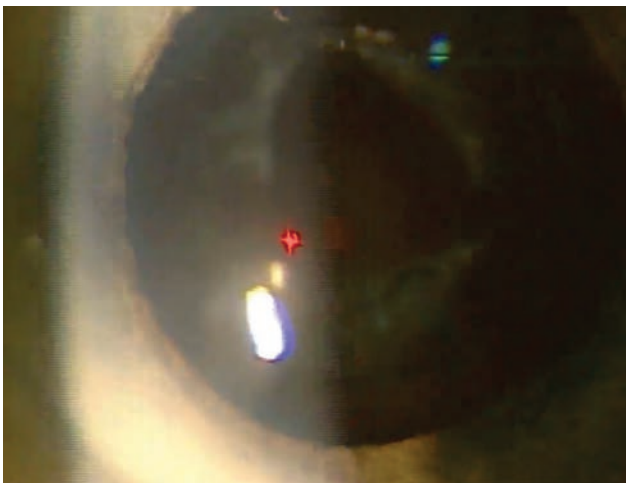

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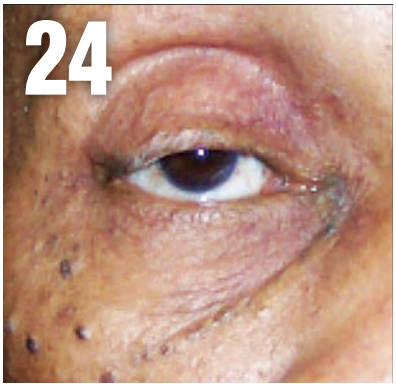

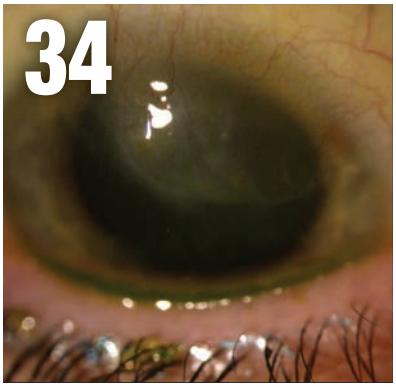

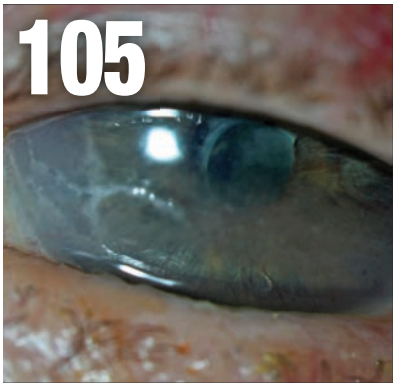

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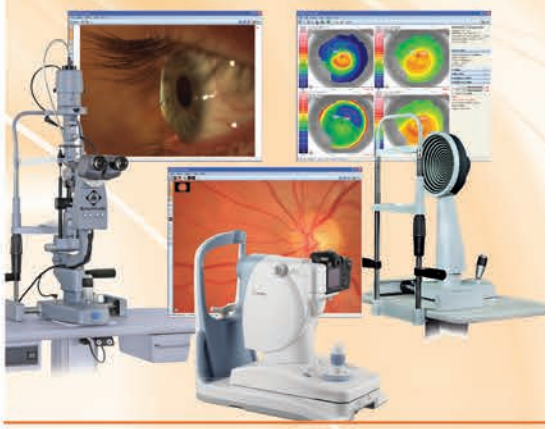

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Letters to the **Editor**

Humbled by a 'Good Catch'

Recently, I saw a new patient to our practice, a 61-year-old white female who worked as an accountant for the local school district. This was her first ever eye exam, and she'd been using cheaters since her mid-40s. She came in because her night distance vision seemed blurry for the past year, and thought it was time to take advantage of her vision plan.

Upon examination, her IOPs were normal and astigmatic refraction improved acuity from 20/50 to 20/20 OD and OS. Motilities and pupils were normal. Biomicroscopy was unremarkable. I instilled 1% tropicamide and had my staff perform our screening field while the patient dilated.

The fundus exam was normal, but the field results were a mess. My tech reported the patient's fixation was all over the place, and since she was a hyperopic presbyope and it was her first field, my inclination was to set those results aside as spurious. While scrawling the glasses script, I commented on how special I felt getting to be her first eye doctor, especially at this point in her visual career. That's when she admitted it was her husband who made her come in after she'd pulled out into oncoming traffic, and on another instance almost hit a pedestrian, both within the past month.

Those comments got my attention. I asked if both near misses were to the same side of her vision and, sure enough, both were in her right field. A quick confrontation field revealed a distinct bilateral homonymous hemianopsia respecting the midline.

Of course, it was 2 p.m. on a Friday. I immediately sent the patient to the ER with a handwritten note requesting intracranial imaging with my suspicion of a left side temporal or parietal lesion. I also described the nature of her field defect. I faxed a more detailed note to her PCP, which I understand he read the following Tuesday.

The patient's oncologist called today and asked to speak with me personally. Unfortunately, he reported my patient's grave diagnosis: multiform glioblastoma, the most aggressive of all brain cancers. Average mortality rate is three months after initial diagnosis, and often there is no treatment because the tumor has become so invasive. However, if caught early (we did), and with surgery (already done), followed by radiation and chemo (she's doing both), recent studies at UCLA include cases with 10- and even 20-year life spans. Due to our early diagnosis and relatively smallish tumor size, the doctor has hopes our patient will be one of the latter cases.

The best part of this story? The oncologist finished our conversation with these two words: "Good catch."

What does an OD say after that? Well, I would have said "thank you" if I hadn't dropped the phone already.

—Jerome L. Brendel, OD
Yuba City, Calif.

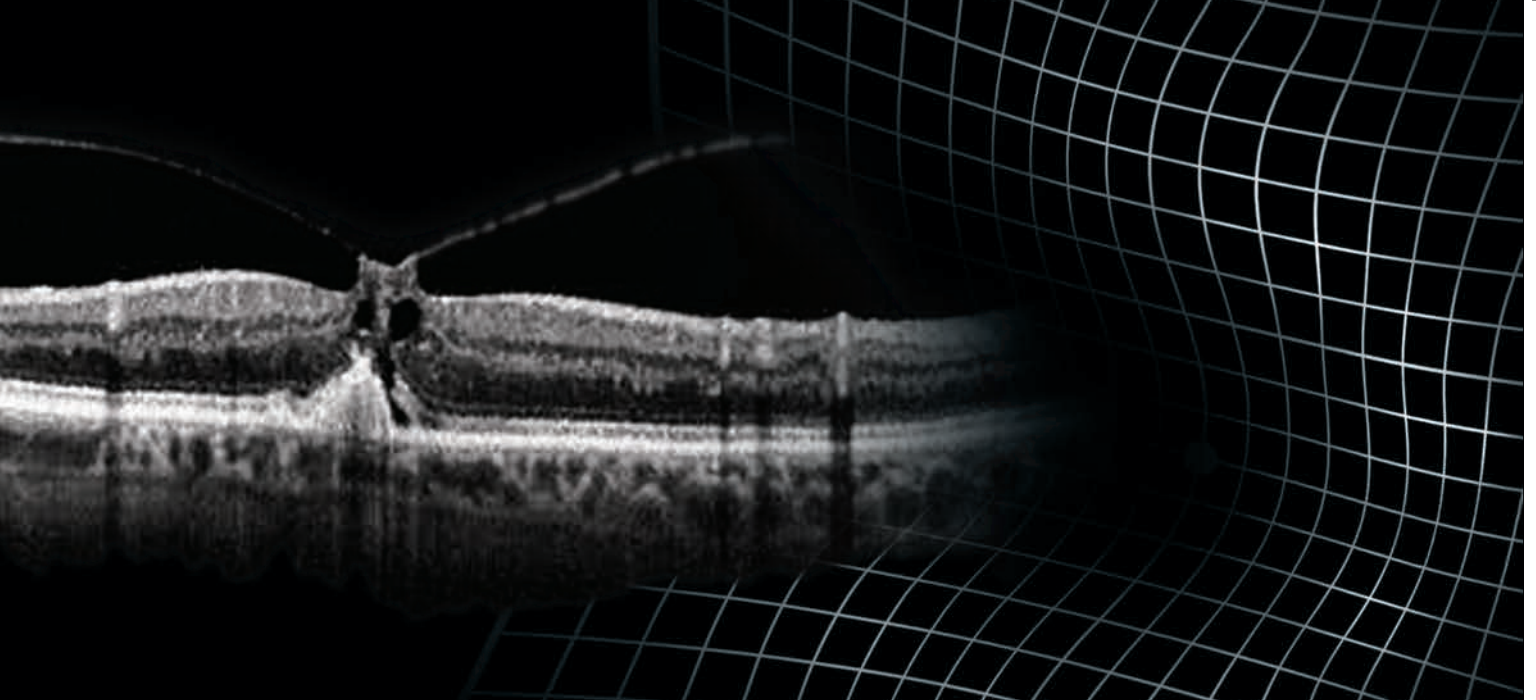
Update from Dr. Brendel:

I called my patient to see how she was doing, and unfortunately she reported that her brain surgeon discovered during her craniotomy that her tumor was much larger than initial imaging projected. Therefore, he performed a partial resection and advised against chemo and radiation due to quality of life considerations.

Nevertheless, we had a very enjoyable conversation. She expressed gratitude for the opportunity to

Sight **Gags**

By Scott Lee, OD



SYMPTOMATIC VITREOMACULAR ADHESION (VMA)

SYMPTOMATIC VMA MAY LEAD TO VISUAL IMPAIRMENT FOR YOUR PATIENTS[1-3]

IDENTIFY | Recognize metamorphopsia as a key sign of symptomatic VMA and utilize OCT scans to confirm vitreomacular traction.

REFER | Because symptomatic VMA is a progressive condition that may lead to a loss of vision, your partnering retina specialist can determine if treatment is necessary.[1-3]

THE STEPS YOU TAKE TODAY MAY MAKE A DIFFERENCE FOR YOUR PATIENTS TOMORROW

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References: 1. Sonmez K, Capone A, Trese M, et al. Vitreomacular traction syndrome: impact of anatomical configuration on anatomical and visual outcomes. *Retina*. 2008;28:1207-1214. **2.** Hikichi T, Yoshida A, Trempe CL. Course of vitreomacular traction syndrome. *Am J Ophthalmol*. 1995;119(1):55-56. **3.** Stalmans P, Lescrauwaet B, Blot K. A retrospective cohort study in patients with diseases of the vitreomacular interface (ReCoVit). Poster presented at: The Association for Research in Vision and Ophthalmology (ARVO) 2014 Annual Meeting; May 4-8, 2014; Orlando, Florida.

reconcile some fractured family relationships, and has been devoutly telling everyone she comes in contact with to "live for the day, because tomorrow is not a guarantee."

As we said our goodbyes, she said, "Thank you, Dr. Brendel. You may not have saved my life, but you did help me experience more of the days I have left."

For my part, I am humbled. And also thankful for the opportunity to practice optometry.

The patient's tumor was much larger than initial imaging projected ... She expressed gratitude for the opportunity to reconcile some fractured family relationships, and has been devoutly telling everyone she comes in contact with to "live for the day, because tomorrow is not a guarantee."

'Myopia' is Not 'Nearsightedness'

Many of us use the terms *myopia* and *nearsightedness* interchangeably. We should stop.

Nearsightedness describes the subjective experience of blurred distance vision relative to near vision. *Myopia* is a condition that results in light being focused anterior to the macula often due to excessive axial length.

Semantics are an important aspect of language. If we choose language that more appropriately describes this condition and use the term myopia in the examination room when educating patients, it may lead to greater compliance with treatments that aim to limit myopia progression.

The fact that our society's lexicon has equated nearsightedness with myopia has led to a ho-hum attitude towards this condition: "My child has a little blurred vision at distance and it has gotten worse over the past year, no big deal."

But it is a big deal. The prevalence of myopia is increasing worldwide and the implications of myopia are well known and include increased risk of maculopathy, retinal detachment, glaucoma and earlier cataracts. How the increased rates and severity of myopia in today's youth will impact ocular health data in 40 years remains to be seen, but many believe we should attempt to limit myopia progression because of the compelling evidence mentioned.

The difference between myopia and nearsightedness not only affects patient education and treatment decisions but also impacts research. Over the years, there have been many attempts at limiting myopia progression—including corneal reshaping/orthokeratology, atropine ophthalmic drops, soft bifocal contact lenses, bifocal glasses, progressive addition lenses, vision therapy and more. Vision therapy—while extremely effective for many binocular vision, accommodative, oculomotor and visual perception deficits—has no research supporting its effectiveness in myopia control. Any publication supporting vision therapy has described improving nearsightedness through improved blur interpretation with less myopic lens compensation. Vision therapy may be able to limit the progression of myopia but it hasn't been proven yet. Any research that is purported to improve myopia should include axial length measurements over time and cycloplegic refractive status.

There has been quite a bit of buzz over myopia these last few years. I hope you join me in cleaning up the language we use to help increase awareness of this important public health issue.

—Daniel J. Press, OD
Clinical Director of Pediatrics, Binocular Vision and Vision Therapy, North Suburban Vision Consultants, Park Ridge, Ill.

Signs of Traumatic Cataract

I am writing in regards to "Caution! Traumatic Cataracts Ahead" in the April 2015 issue. The authors did a very nice job of describing the challenges of surgery on traumatic cataracts. It would have been even better had they described the clinical signs that might suggest a traumatic cataract is present. It is the role of the OD to detect and inform the surgeon of these clinical signs.

Patient history is the first line of defense in detecting traumatic cataract and potential negative sequelae. Even if the patient has denied trauma earlier in the exam, it is a good idea to ask again.

Sometimes patients forget trauma that was years or decades in the past and then later remember the event, but they may not bring it up unless you ask about it another time.

Clinical clues to significant ocular trauma include any full-thickness corneal scar, a dyscoric pupil, iridodialysis, iridodonesis (a fluttering motion of the iris on ocular movement) and irido-corneal adhesion in conjunction with a full-thickness corneal scar. Gonioscopy should be performed looking for angle recession.

On dilated exam, the lens should be evaluated for phaco-donesis (a fluttering of the lens on eye movement), subluxation, peripheral blunting of the lens margins (indicating zonular dehis-cence) and for the integrity of the capsule. Any presence of vitreous in the posterior or anterior cham-ber is also evidence of zonular compromise.

Fundus exam, if possible, should include a careful check for peripheral retinal tears or dialysis and lacquer cracks in the macula or around the disc.

If significant zonular dehiscence and subluxation is present, it may be best to have the cataract removed via a pars plana ap-proach by a retinal subspecialist (so that vitreous can be controlled and any retinal issues addressed) with a subsequent surgery for IOL implantation, either with an ante-rior chamber IOL or a posterior chamber IOL with iris sutures. This should be discussed with the selected surgeon(s).

Thanks for your great work in educating our profession. ■

—Howell M. Findley, OD
Lexington, Ky.

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Optometry's Power Trio

With our annual disease guide, Drs. Sowka, Kabat and Gurwood take center stage. **By Jack Persico, Editor-in-Chief**

The rock and roll "power trio" lineup relies on just a core group of players (all virtuosos, typically) who approach their craft with creative bravado, producing epics that push the boundaries. Groups like Cream and the Jimi Hendrix Experience epitomized the power trio during the classic rock era. The form may have fallen out of favor in recent years, but it's a dynamic I've always enjoyed witnessing. (Here at *Review*, production manager Scott Tobin and I are unabashed fans of Rush, touring this summer for their 40th anniversary.) A tight trio that really clicks can produce amazing results.

Meet the Band

As we wrap this issue of *Review*, I feel like we have a power trio of our own: Drs. Joe Sowka, Alan Kabat and Andy Gurwood, all expert clinicians who can wield a slit lamp like Clapton playing a Stratocaster. (Andy, in fact, is a guitarist for real.) These three play so many roles here it's sometimes hard to keep track.

Dr. Kabat, as one of our associate clinical editors, helps to shape the content of the publication, vet manuscripts and suggest authors. He also consistently raises the bar for editorial quality—he's as tenacious about grammar as he is about scientific rigor and clinical relevance.

Dr. Sowka coauthors the monthly Therapeutic Review column with Dr. Kabat, and reviews manuscripts as a vital part of our editorial screening process. He also authors standalone features, notably on glaucoma (look for one next month on glaucomatocyclitic crisis) and contributes to the overall planning process for the publication.

Dr. Gurwood authors the highly popular Diagnostic Quiz on the back page of the magazine, where he challenges readers to test their clinical acumen. In the same vein, Andy reviews and coordinates our case reports (look for a great one this month on page 82). And he even found time this month to coauthor a thorough overview of enhanced depth imaging OCT (page 70). Whenever there's something new to help improve diagnosis, we're confident that Andy can give our readers the lowdown.

Summer Festival

But this month's showpiece for these esteemed clinicians is, of course, *The Handbook of Ocular Disease Management*. It's here that the trio really rocks, as their unique strengths converge to create something truly outstanding. Begun nearly two decades ago, this ambitious project bridges academia and practice—distilling the hands-on clinical expertise the triumvirate has cultivated in treating ocular diseases from the mundane to the exotic, all supported with copious references. This edition weighs in at 40,000 words and nearly 1,000 references (952 for you sticklers), with 30 entries in the guide.

We have the privilege of working with many outstanding optometrists to produce RO and its offshoots, and thank them all for their generous commitment of time and talent—and, this month especially, Joe, Al and Andy. ■

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All Boxed In

I'm drowning in half-empty contact lens boxes. It's time I take control, consequences be damned. **By Montgomery Vickers, OD**

My desk has been invaded by vast quantities of soft contact lenses in half-empty boxes and cases. I can only guess where they came from, when they arrived and what I should do with them. Therefore, I have decided to throw them all—every single dried out blue cornflake wannabe—into the trash.

Please don't tell my wife.

Why can't I stay ahead of this junk? It's like roaches and ants; they replicate faster than I can stomp 'em. One day, my desk has neat little stacks of paper (which are, admittedly, largely also a mystery to me, but at least they are all the same size so my desk looks organized). The next day, I am swamped with lenses that tore after two and a half weeks ("Aren't these monthly? Must be defective!") and half-empty boxes from patients who "only wore three pair" before they decided they hated bifocal contact lenses after all.

What should I do with them? I guess I should fuss at my sales rep until he trades them in or gives me credit on our account.

Do you think the fact that I have no clue whose they were, when they were ordered or why they expire next week should have any impact? Unless they cost me a couple hundred bucks, I think it's more cost effective to toss them than to send them back, which takes time away from what is important in my office—reading the obituaries.

I often think I should get this whole system more organized. I used to have designated employees whose responsibility included dealing with wayward contact lenses. They became experts in spending 30 minutes looking and then announcing, "I can't find this invoice." It's probably our complicated filing system's fault. It's a system none of my staff members has ever had any real experience with: the alphabet.

So, I finally gave up and told my assistants I would deal with them. This has changed my perspective so very much, as you assistants can imagine. I have become humble and, yes, totally incompetent. This has helped us bond as a unit. In fact, we have changed our office motto from "The Area's Finest Eye Care Office" to "We Have No Clue Where Your Contact Lenses Went."

There are a few problems with trashing the lenses all over my desk:

1. If my office manager/wife finds out, I am in deep doo-doo. It would actually be better if she found out I am having 12 affairs than to lose one $42 refund on a box I was supposed to send back. Anybody can have affairs, but in her opinion, that $42 is *her* $42. She wants her dang money.

2. As soon as I trash them, someone will call and say they decided they loved the lenses they returned after all and need them back because they are leaving for the beach tomorrow. They want to order a ton of boxes and, well, I almost always forget to write the parameters of the lens on the chart, so I am in a mess. Now, computers have helped immeasurably, mainly because I can blame the computer for losing the information and not appear to be the nincompoop I actually am.

3. If I trash all the boxes on my desk, I will uncover all the ICD-10 stuff that I have been dreading.

In the end, I made the decision to pitch the whole contact lens cesspool into the trash, no matter what the cost or fallout. Well, I did keep one half-empty box. After all, it was close to my prescription! My wife gave me a 10% discount. ■

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LITR-819 REV A

Doin' the Dilation Lag

Patients with emergency presentations may not recognize the signs of Horner's syndrome, but you can, using these clinical pearls. **By Richard Mangan, OD**

A 36-year-old white male presents to your office with a chief complaint of intermittent pain on the right side of his head and orbital area along with redness and foreign body sensation in his right eye. As a construction worker, he's concerned that he may have gotten something in his eye.

External examination shows a mild ptosis (1.5mm) with minimal palpebral injection and no lid edema in the right eye. No foreign body of the cornea or bulbar conjunctiva is noted on a slit lamp examination, nor is any evident on lid eversion. The cornea is clear, the anterior chamber is well formed and quiet. His unaided visual acuity is 20/20 each eye.

Pupil testing reveals anisocoria greatest under dim illumination and most noticeable during the first few seconds after the lights were turned down. The right pupil showed a delay in dilation consistent with "dilation lag" found in Horner's syndrome. So, you perform bilateral tactile assessment of the forehead to discern potentially uneven sweating patterns. Anhidrosis is not evident and not reported by the patient.

But what if your office setting has no available pharmacological agents able to confirm or localize Horner's syndrome? Should the patient be referred for diagnostic testing, or should imaging be ordered? If so, what is the best approach to ordering imaging for acute onset Horner's syndrome?

This article offers several pearls on the differential diagnosis and timely management of acute-onset Horner's syndrome patients.

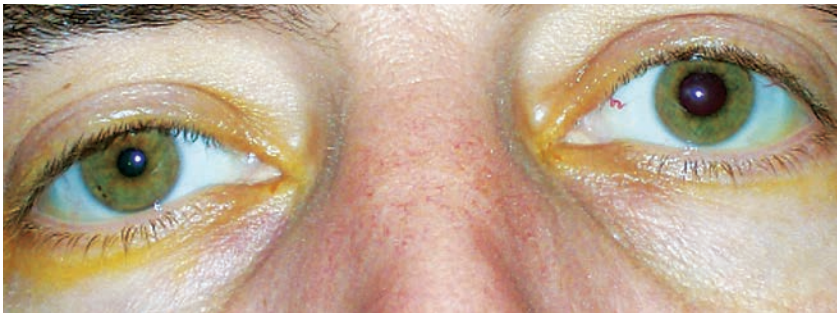

Photo: Alan G. Kabat, OD

Anisocoria in a patient with recent onset of Horner's syndrome.

The Classic Triad

Patients who present with unilateral ptosis, miosis and anhidrosis have the classic triad of signs associated with oculosympathetic paresis, also known as Horner's syndrome.[1] Because the oculosympathetic pathway of the eye is rather long and at times winding, the list of potential causes that can disrupt the efferent signal along this pathway is long.

When disruption along the oculosympathetic pathway occurs, the efferent signal to Mueller's muscle and the iris dilator muscle is adversely affected, resulting in pseudo-enophthalmos (1mm to 2mm ptosis of the upper lid coupled with reverse ptosis of the lower lid) and a miotic pupil, respectively. The ptosis and anisocoria can be quite subtle and may go unnoticed by friends, family and coworkers.

The pupil is miotic because the circumferential sphincter muscle of the iris, which is innervated by the parasympathetic pathway, is no longer equally opposed by the oculosympathetic pathway, which innervates the radial dilator muscle.[2] The resultant anisocoria is most noticeable under dim illumination. Dilation lag is a classic sign of Horner's syndrome—when the slit lamp beam is turned off, the anisocoria will be most noticeable during the first four or five seconds of viewing. The abnormal pupil will slowly dilate or "lag behind" over 10 to 15 seconds, making the pupil asymmetry less evident. This partially explains why some cases of Horner's syndrome are diagnosed during a routine eye examination.

Causes

Horner's syndrome is most often due to a benign etiology; but a few sinister causes require urgent diagnosis and care. These include, but are not limited to:

- Spontaneous or traumatic carotid artery dissection.
- Neoplasia in the neck.
- Lung apex (Pancoast tumor).
- Childhood neuroblastoma.

Establishing the underlying diagnosis of Horner's syndrome begins with a detailed clinical history,

Down, Boy.

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- Use of corticosteroids may result in posterior subcapsular cataract formation.
- Use of steroids after cataract surgery may delay healing and increase the incidence of bleb formation and occurrence of perforations in those with diseases causing corneal and scleral thinning. The initial prescription and renewal of the medication order should be made by a physician only after examination of the patient with the aid of magnification, and where appropriate, fluorescein staining.
- Prolonged use of corticosteroids may suppress the host response and thus increase the hazard of secondary ocular infection. In acute purulent conditions, steroids may mask infection or enhance existing infection.
- Use of a corticosteroid medication in the treatment of patients with a history of herpes simplex requires great caution. Use of ocular steroids may prolong the course and exacerbate the severity of many viral infections of the eye (including herpes simplex).
- Fungal infections of the cornea are particularly prone to develop coincidentally with long-term local steroid application. Fungus invasion must be considered in any persistent corneal ulceration where a steroid has been used or is in use.
- Patients should not wear contact lenses when using LOTEMAX® GEL.
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LOTEMAX is a corticosteroid indicated for the treatment of post-operative inflammation and pain following ocular surgery.

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Invert closed bottle and shake once to fill tip before instilling drops.

Apply one to two drops of LOTEMAX into the conjunctival sac of the affected eye four times daily beginning the day after surgery and continuing throughout the first 2 weeks of the post-operative period.

CONTRAINDICATIONS

LOTEMAX, as with other ophthalmic corticosteroids, is contraindicated in most viral diseases of the cornea and conjunctiva including epithelial herpes simplex keratitis (dendritic keratitis), vaccinia, and varicella, and also in mycobacterial infection of the eye and fungal diseases of ocular structures.

WARNINGS AND PRECAUTIONS

Intraocular Pressure (IOP) Increase

Prolonged use of corticosteroids may result in glaucoma with damage to the optic nerve, defects in visual acuity and fields of vision. Steroids should be used with caution in the presence of glaucoma. If this product is used for 10 days or longer, intraocular pressure should be monitored.

Cataracts

Use of corticosteroids may result in posterior subcapsular cataract formation.

Delayed Healing

The use of steroids after cataract surgery may delay healing and increase the incidence of bleb formation. In those diseases causing thinning of the cornea or sclera, perforations have been known to occur with the use of topical steroids. The initial prescription and renewal of the medication order should be made by a physician only after examination of the patient with the aid of magnification such as slit lamp biomicroscopy and, where appropriate, fluorescein staining.

Bacterial Infections

Prolonged use of corticosteroids may suppress the host response and thus increase the hazard of secondary ocular infections. In acute purulent conditions of the eye, steroids may mask infection or enhance existing infection.

Viral Infections

Employment of a corticosteroid medication in the treatment of patients with a history of herpes simplex requires great caution. Use of ocular steroids may prolong the course and may exacerbate the severity of many viral infections of the eye (including herpes simplex).

Fungal Infections

Fungal infections of the cornea are particularly prone to develop coincidentally with long-term local steroid application. Fungus invasion must be considered in any persistent corneal ulceration where a steroid has been used or is in use. Fungal cultures should be taken when appropriate.

Contact Lens Wear

Patients should not wear contact lenses during their course of therapy with LOTEMAX.

ADVERSE REACTIONS

Adverse reactions associated with ophthalmic steroids include elevated intraocular pressure, which may be associated with infrequent optic nerve damage, visual acuity and field defects, posterior subcapsular cataract formation, delayed wound healing and secondary ocular infection from pathogens including herpes simplex, and perforation of the globe where there is thinning of the cornea or sclera.

The most common adverse drug reactions reported were anterior chamber inflammation (5%), eye pain (2%), and foreign body sensation (2%).

USE IN SPECIFIC POPULATIONS

Pregnancy

Teratogenic Effects: Pregnancy Category C.

Loteprednol etabonate has been shown to be embryotoxic (delayed ossification) and teratogenic (increased incidence of meningocele, abnormal left common carotid artery, and limb flexures) when administered orally to rabbits during organogenesis at a dose of 3 mg/kg/day (35 times the maximum daily clinical dose), a dose which caused no maternal toxicity. The no-observed-effect-level (NOEL) for these effects was 0.5 mg/kg/day (6 times the maximum daily clinical dose). Oral treatment of rats during organogenesis resulted in teratogenicity (absent innominate artery at ≥5 mg/kg/day doses, and cleft palate and umbilical hernia at ≥50 mg/kg/day) and embryotoxicity (increased post-implantation losses at 100 mg/kg/day and decreased fetal body weight and skeletal ossification with ≥50 mg/kg/day). Treatment of rats with 0.5 mg/kg/day (6 times the maximum clinical dose) during organogenesis did not result in any reproductive toxicity. Loteprednol etabonate was maternally toxic (significantly reduced body weight gain during treatment) when administered to pregnant rats during organogenesis at doses of ≥5 mg/kg/day.

Oral exposure of female rats to 50 mg/kg/day of loteprednol etabonate from the start of the fetal period through the end of lactation, a maternally toxic treatment regimen (significantly decreased body weight gain), gave rise to decreased growth and survival, and retarded development in the offspring during lactation; the NOEL for these effects was 5 mg/kg/day. Loteprednol etabonate had no effect on the duration of gestation or parturition when administered orally to pregnant rats at doses up to 50 mg/kg/day during the fetal period.

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

Nursing Mothers

It is not known whether topical ophthalmic administration of corticosteroids could result in sufficient systemic absorption to produce detectable quantities in human milk. Systemic steroids appear in human milk and could suppress growth, interfere with endogenous corticosteroid production, or cause other untoward effects. Caution should be exercised when LOTEMAX is administered to a nursing woman.

Pediatric Use

Safety and effectiveness in pediatric patients have not been established.

Geriatric Use

No overall differences in safety and effectiveness have been observed between elderly and younger patients.

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment Of Fertility

Long-term animal studies have not been conducted to evaluate the carcinogenic potential of loteprednol etabonate. Loteprednol etabonate was not genotoxic *in vitro* in the Ames test, the mouse lymphoma tk assay, or in a chromosome aberration test in human lymphocytes, or *in vivo* in the single dose mouse micronucleus assay. Treatment of male and female rats with up to 50 mg/kg/day and 25 mg/kg/day of loteprednol etabonate, respectively, (600 and 300 times the maximum clinical dose, respectively) prior to and during mating did not impair fertility in either gender.

PATIENT COUNSELING INFORMATION

Administration

Invert closed bottle and shake once to fill tip before instilling drops.

Risk of Contamination

Patients should be advised not to allow the dropper tip to touch any surface, as this may contaminate the gel.

Contact Lens Wear

Patients should be advised not to wear contact lenses when using LOTEMAX.

Risk of Secondary Infection

If pain develops, redness, itching or inflammation becomes aggravated, the patient should be advised to consult a physician.

FOR MORE DETAILED INFORMATION, PLEASE READ THE PRESCRIBING INFORMATION.

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Is It Long Standing?

Long-standing cases are less concerning than acute cases. Inspecting old photographs may confirm long-standing Horner's syndrome and support the decision to monitor. The presence of iris heterochromia (affected iris is blue and the other brown) certainly indicates that the lesion occurred somewhere between birth and two years of age. The most common cause is birth trauma resulting in a brachial plexus injury.

Is It Iatrogenic?

Patients who have had head, neck or chest surgery may be experiencing a temporary or permanent side effect from the procedure. The preganglionic neuron is the most common site of injury for iatrogenic Horner's syndrome. Such procedures include coronary artery bypass surgery, carotid endarterectomy or stenting, insertion of a pacemaker, epidural anesthesia, chest tube insertion, as well as lung or mediastinal surgery. If the patient reports recent surgery involving one or more these anatomical areas, be sure to notify the surgeon of your findings.

Is There Ipsilateral Anhidrosis?

Anhidrosis, or loss of hemifacial sweating, may be seen in a subset of Horner's patients. The level of involvement ranges from barely detectable to striking. The area of involvement may include the brow, forehead or the entire side of the head and face, depending on lesion location. If a patient has a lesion near the common carotid artery, loss of sweating involves the entire side of the face. With lesions distal to the carotid bifurcation, the lack of sweating is typically confined to the medial aspect of the forehead and side of the nose.

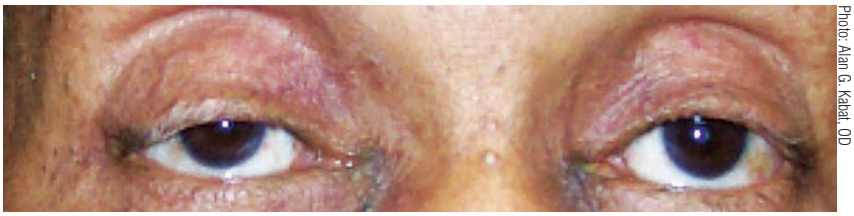

Photo: Alan G. Kabat, OD

New onset right ptosis (with miosis) in a patient who presented with Horner's syndrome secondary to a Pancoast tumor.

A patient may report an exertion-induced asymmetric reaction of the skin remaining pale and dry on the side of the face with the affected pupil. The presence of anhidrosis narrows the list of potential causes to the central and preganglionic pathways.

Pain Matters

If Horner's syndrome is associated with acute-onset ipsilateral head, orbital, face or neck pain, consider it an internal carotid artery dissection (ICAD) until proven otherwise.[3] Studies show that a partial-Horner's syndrome is found in 36% to 58% of all ICAD cases.[4] ICAD is rare, with an incidence of three in 100,000 and is classified as either spontaneous (i.e., connective tissue disease) or traumatic.[5] While the most common traumatic cause of ICAD is motor vehicle accidents, any exertional force that induces a twisting of the head or neck may cause the lamina intima of the ICA to tear secondarily to hyperextension, rotation, or both.[3,6] This can lead to bleeding into the laminar wall of the vessel, resulting in ischemia or thrombosis. If not treated with anticoagulation therapy in a timely fashion, the patient may suffer stroke or even death.[6,7]

If, on the other hand, Horner's syndrome is associated with a radiating pain from the shoulder to the ulnar side of the arm and hand, this suggests an apical lung tumor 55% to 60% of the time.[8] Pancoast tumors are also associated with neck pain as well as parasthesias of the hand.[9] More than 200,000 new cases of lung cancer are diagnosed every year, approximately 5% of which are Pancoast tumors.[10] Incidence is highest in males between the age of 40 and 60 with a history of smoking.[10] Pancoast tumors are a form of non-small-cell carcinoma, most commonly with squamous cell carcinoma (45% to 50%).[8]

No Cocaine? No Worries!

Pharmacologic testing may be helpful in diagnosing and localizing a Horner's syndrome lesion.

Four percent to 10% cocaine was historically used to check for sympathetic pupil denervation.[11] However, recent research suggests a more readily available product, apraclonidine, is a reliable (87% sensitivity) and practical solution.[13,14]

Apraclonidine is an ocular hypotensive agent that acts as a weak alpha-1 agonist and a strong alpha-2 agonist.[15] In Horner's syndrome, upregulation of alpha-1 receptors increases apraclonidine sensitivity and causes denervation and supersensitivity of the iris dilator muscle. This results in pupillary dilation and lid elevation on the abnormal side, with little to no effect—or even mild miosis on the normal side due to the alpha-2 activity. The apraclonidine test is considered positive if it reverses anisocoria

after bilateral instillation.

Twenty-four to 48 hours after applying 1% apraclonidine, the 1% hydroxyamphetamine test may further distinguish a postganglionic or third-order neuron lesion from central or preganglionic causes. Hydroxyamphetamine stimulates the release of stored endogenous norepinephrine from the postganglionic axon terminals into the neuromuscular junction at the iris dilator muscle.

Hydroxyamphetamine drops instilled into a Horner's syndrome patient's eye who has intact postganglionic fibers (i.e., first- or second-order neuron lesions) dilate the affected pupil to an equal or greater extent than they do the normal pupil. Therefore, the degree of anisocoria remains the same or decreases. However, hydroxyamphetamine drops instilled into an eye with Horner's syndrome with damaged postganglionic fibers (third-order neuron lesions) do not dilate the affected pupil as well as they do a normal pupil. Therefore, the anisocoria is likely to increase.

Pharmacological Testing vs. Stat Imaging

Now, apply these pearls to our 38-year-old construction worker. You could certainly do some more investigation into his general health, as well as surgical and recreational history. Does he smoke? Does he have a history of cluster headaches? With that said, and given what we know, this is a case where stat imaging takes precedence over trying to schedule pharmacological testing to localize the lesion—pain trumps pupil testing in this case.

While scheduling, communicate with the radiologist who will be running the tests. Radiologists can often act as a partner in designing an imaging plan based on the clinical insight you provide.

Take the time to educate your staff on the importance of bringing all unilateral ptosis patients to your attention before they are dilated. Horner's syndrome is not something you want to miss. ■

Testing Hierarchy for Horner's Syndrome

FIRST-ORDER NEURON LESIONS
Occurs uncommonly in isolation, usually one of a number of neurologic findings.

Associated Findings	Diagnostic Tests	Anatomic Sites	Imaging
• Dysphagia (difficulty swallowing) • Dysarthria (slowed or slurred speech) • Hemisensory loss • Ataxia (lack of muscle coordination) • Vertigo • Nystagmus	• Cocaine test (+) • Iopidine test (+) • Paredrine (+)	• Hypothalamus, thalamus, brainstem • Cervical spinal cord	• CT of the chest & CT / CTA of the head & neck *or* • MRI of the chest & MRI / MRA of head & neck

SECOND-ORDER NEURON LESIONS
Most often caused by trauma or tumor, including malignant tumors.

Associated Findings	Diagnostic Tests	Anatomic Sites	Imaging
• Hx of prior head, face, or neck trauma • Previous thoracic or neck surgery / chest tube or catheter placement • Any facial, neck, axillary, shoulder or arm pain • Chronic cough or hemoptysis (coughing up blood or blood stained mucous)	• Cocaine test (+) • Iopidine test (+) • Paredrine (+)	• Cervicothoracic spinal cord • Brachial plexus • Anterior aspect of the Neck • Lung apex • Mediastinum	• CT of the chest & CT / CTA of the head & neck *or* • MRI of the chest & MRI / MRA of head & neck

THIRD-ORDER NEURON LESIONS
Variable causes from benign to life-threatening.

Associated Findings	Diagnostic Tests	Anatomic Sites	Imaging
• Diplopia (i.e. VI N palsy) • V1 & V2 numbness • Pain	• Cocaine test (+) • Iopidine test (+) • Paredrine (-)	• Superior cervical ganglion • Internal carotid artery • Cavernous sinus • Orbital apex	• MRI / MRA of the head & neck

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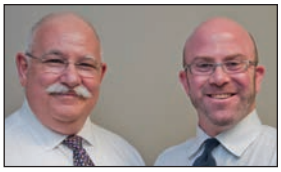

A Prism Disaster

Diplopia, headaches and blurry near vision made homework a chore for this young man. His current glasses didn't help. **By Marc B. Taub, OD, MS, and Paul Harris, OD**

Jonathon, an 18-year-old male, was referred by a local pediatric ophthalmologist. The patient had been having headaches, blurry vision at near and intermittent diplopia since first grade. Jonathon and his parents, who had accompanied him to the examination, were seeking a second opinion concerning surgery.

When questioned about previous treatment, Jonathon brought out a most interesting pair of glasses—Ben Franklin style "bifocals" in which each lens was made of two separate lenses of different prism powers. He was wearing a total prism power of 6Δ base in for distance (split 3Δ base in each eye) and 20Δ base in for near (split 10Δ base in each eye) with a +4.00D add!

Our patient's glasses had two different prism powers split into each lens.

The power of the prism had increased during the past four years since the pediatric ophthalmologist had referred the patient to an optometrist for prism glasses. After four years of power increases to the Rx, the family recently went back to the surgeon, who responded by saying that he was surprised that the patient had not come back sooner.

They discussed surgery, requested a second opinion and the surgeon referred him to our clinic.

Diagnostic Data

At our examination, Jonathon complained of headaches and blurry vision with near work and diplopia at near greater than distance. The family characterized his learning history as challenging. Reading was especially difficult—he mixed up small words, words ran together and he was seeing double. Mom recalled that she would copy assignments and increase the size of the materials to make it easier for him to read. This brought her to the verge of tears.

Jonathon was not able to keep up in a traditional classroom and was completing high school online. He was planning to attend a technical school in the fall. He had scored a 21 on the ACT (an average score); he knew that his visual issues were holding him back.

When questioned about the use of his glasses, he reported that they were of little help and that he never used them. His habitual spectacle prescription was:

+1.00 DS 3Δ base in at distance, 10Δ base in at near OD

+1.00-0.75 x 90 3Δ base in at distance, 10Δ base in at near OS

+4.00 add OU

His visual acuity was 20/20 OD, OS, OU at distance and 20/30 OD, 20/40 OS at near. Uncorrected acuity was 20/30 OD, OS, OU at distance and 20/100 OD, OS, OU at near. Stereopsis was measured at 25 seconds of arc on Wirt circles, and covert test was 2Δ exophoria at distance and 8Δ intermittent alternating exotropia at near. Near point of convergence was 10cm break and 14cm recovery, performed three times. Accommodative amplitudes were 13D OD and 14D OS. Confrontation fields, pupils and extraocular motilities were normal.

Retinoscopy:
OD: +1.75-0.50 x 105
OS: +1.75-0.75 x 055
Subjective refraction:
OD: +1.25-0.50 x 105 20/20
OS: +1.50-0.50 x 055 20/20

With the subjective refraction in place, the near visual acuity was 20/50 OD, OS, OU. Negative relative accommodation (NRA) was +2.00, and positive relative accommodation (PRA) was -0.75. The patient also reported diplopia.

With the subjective refraction in a trial frame, we attempted vergence ranges with a prism bar (which allows greater ability to observe the eyes). In this case, we used bar vergences instead of vergences in the phoropter due to the presence of the exotropia. At distance, the ranges were limited,

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showing x/6/4 for positive fusional vergence and x/6/1 for negative fusional vergence. At near, the patient was diplopic and required 10Δ base in for fusion.

Diagnosis and Rx

At this point, two things became clear. One, the patient needed plus at near, based on the visual acuity and imbalance of the NRA/PRA. Second, he also needed vision therapy.

Based on the approximate +0.50D imbalance, we trial framed that amount over the subjective refraction. His visual acuity immediately improved to 20/20 OD, OS, OU. We repeated the cover test at near, and he demonstrated great control with 4Δ of exophoria. After discussing the prescription options, we decided that a full-time prescription with the extra +0.50 was the right choice based on the patient's future technical trade, patient choice and the fact that when trialed, the patient continued to see 20/20 at distance.

The final prescription for full-time wear was +1.75-0.50 x 105 20/20 OD and +2.00-0.50 x 55 20/20 OS.

We presented the idea of vision therapy to all parties and discussed the potential improvement in symptoms and academic limitations. The parents were shocked to hear about such a treatment and that it was not recently invented. They questioned why Jonathon's previous doctors had not offered this option and instead allowed him to struggle, prescribing increasing amounts of prism and then turning to surgery to correct this common visual issue. Mom once again started to cry. We felt sorry that their son had struggled for so long, but were confident that with time and commitment, vision therapy along with the appropriate glasses prescription would successfully remediate the condition.

The patient is now enrolled in a vision therapy program and his symptoms have improved after just six sessions.

Discussion

A wealth of research directs us to the most appropriate treatment for convergence insufficiency, which affects 3% to 5% of the population.[1] In a comparison of base in prism (based on Sheard's criteria) vs. placebo glasses, base in prism reading glasses were found to be no more effective than placebo in alleviating symptoms, improving the near point of convergence or improving positive fusional vergence at near.[2]

In a randomized, double-blind, placebo-controlled study that compared office-based vision therapy, office-based sham (placebo) therapy and home-based pencil push-ups (HBPP), office-based vision therapy was more effective than office-based sham (placebo) therapy and home-based pencil push-ups in reducing signs and symptoms of convergence insufficiency.[3]

A second randomized, double-blind study investigated HBPP, home-based computer vergence/accommodative therapy and pencil push-ups (HBCVAT), office-based vergence/accommodative therapy with home reinforcement (OBVAT) and office-based placebo therapy with home reinforcement (OBPT). This study found a significant difference between the OBVAT treatment and all of the other treatments.[4] The research team concluded that 12 weeks of OBVAT compared with HBPP, HBCVAT and OBPT results in significantly greater:

- Improvement in symptoms
- Improvement in near point of convergence
- Improvement in positive fusional vergence
- Percentage of patients reaching the predetermined criteria of success

The "take home" message is that convergence insufficiency is most effectively treated with office-based vision therapy with home-based support. So, if your office does not offer this treatment, refer to a colleague who does. To find an optometrist in your area who offers vision therapy, visit the Optometric Extension Program Foundation (www.oepf.org/page/map) or the College of Optometrists in Vision Development (http://locate.covd.org). No child like Jonathon should be allowed suffer and not reach his true potential. Do not keep vision therapy—the most appropriate treatment for not only convergence insufficiency but also accommodative and ocular motor dysfunction—a hidden gem. It's time to share the wealth. ■

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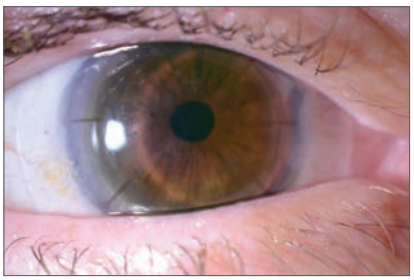

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Hazed and Confused

What's causing this patient's neovascularization and corneal haze? Think outside the cornea. **Edited by Paul C. Ajamian, OD**

Q I have a long-time contact lens patient who recently developed neovascularization and corneal haze. Could this be limbal stem cell deficiency?

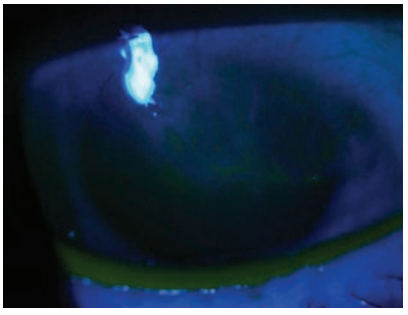
A "It absolutely could be—but it's not necessarily one of the first things to consider," says Will Smith, OD, of Advanced Vision Care in Los Angeles.

For instance, superior limbic keratoconjunctivitis (SLK) can have a similar appearance. "The way to differentiate between SLK and limbal stem cell deficiency is that SLK will stain with both fluorescein and lissamine green, but limbal stem cell deficiency will not stain with lissamine green," he says.

Why? It has to do with the disease itself, Dr. Smith explains. In limbal stem cell deficiency, something goes wrong with the stem cell development process in the limbus—instead of becoming avascular corneal cells, the limbal stem cells develop as conjunctival cells, producing that neovascular and hazy appearance on the cornea. The etiology of the problem can be congenital (e.g., aniridia), acquired (e.g., chemical or thermal burns) or inflammatory (e.g., Stevens-Johnson syndrome).

In any event, lissamine green stains damaged and dead epithelial cells, as in SLK. But the cells in limbal stem cell deficiency are not damaged—they're simply in the wrong location, so they won't automatically stain with lissamine green.

Also, be sure to rule out infectious causes. Dr. Smith relates the case of a patient referred to his office with a presumed diagnosis of interstitial keratitis, which is generally due to a systemic inflammatory disease such as syphilis. "But other bacterial infections—such as tuberculosis, Lyme, even parasitic infection like *Acanthamoeba*—should be considered, too. In such cases, order lab work to rule out these causes," Dr. Smith says.



A whorled, vortex-like keratopathy is a clue to a limbal stem cell deficiency.

In his patient's case, the lab work showed that she did not have interstitial keratitis. Instead, a whorled keratopathy pointed to a different etiology.

Generally, the diagnosis of limbal stem cell deficiency is made clinically, Dr. Smith says. It's often characterized by a whorling, vortex pattern on the cornea—which was indeed the case in this patient with presumed "interstitial keratitis."

If you wish to confirm the clinical diagnosis, the gold standard test is corneal impression cytology, in which filter paper is placed on the patient's anesthetized eye, then removed and sent off for histological analysis, Dr. Smith says.

Discontinue contact lens wear immediately. Contact lenses, especially for high myopes, can sit heavily on the limbus, which can foster hypoxia and mechanical irritation. (Indeed, contact lens wear can be one of the causes of limbal stem cell deficiency.)

Treatment involves maximum-strength, preservative-free anti-inflammatory therapy, such as preservative-free dexamethasone 0.1% or preservative-free prednisolone acetate 1% suspension, which must be ordered through a compounding pharmacy. Start at QID for one week, then BID for as long as necessary until the inflammation resolves.

Improvement will be slow. "I wouldn't expect to see a whole lot of difference until about a month out," Dr. Smith says. "We've had patients whose inflammation didn't start regressing for four months."

Upon resolution, vision usually returns to normal, although pannus and ghost vessels may remain on the cornea. If the patient wishes to return to contact lens wear, reconsider the lens modality, Dr. Smith says. Try a gas permeable lens or a scleral lens that vaults the limbus.

If the patient doesn't improve, or if the disease is already severe, consider a referral to an anterior segment specialist who is familiar with the surgical option of limbal stem cell transplantation.

In either case, follow these patients closely, as limbal stem cell deficiency often recurs, Dr. Smith says. ■

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New Surgical Options for PRESBYOPIA

By Sondra Black, OD, Paul Karpecki, OD, and Eric Brooker, OD

The corneal inlay is the latest in vision correction surgery for presbyopic patients; here's what you need to know about it, including the different styles, expected results, comanagement tips and more.

By 2020, there will be 2.1 billion presbyopes worldwide. These patients make up a significant portion of most optometric practices and are in many ways the ideal demographic group. Baby boomers and older Gen-Xers are at the peak of their careers, financially secure, and at a point in their lives where they can focus on their own health care and quality of life. Compared to earlier generations, today's presbyopes are less willing to accept outward signs of aging. They also have visual demands that didn't exist 20 years ago, such as smart phones, tablet and laptop computers, and vehicle navigation systems.

As these patients become presbyopic, they often transition into presbyopic versions of their distance correction: bifocal, trifocal, or progressive spectacles, multifocal contact lenses or contact lens monovision. Each of these conventional approaches to presbyopia has pros and cons—but the disadvantages for night driving and/or intermediate tasks can trouble even long-time spectacle and contact lens wearers. This has led many to pursue a surgical option.

Until recently, surgical options for presbyopia have been as unpalatable as non-surgical ones. For example, LASIK monovision has the same tradeoffs as contact lens monovision, with the added disadvantage that the degree of monovision can't be adjusted as presbyopia advances. Another option, refractive lens exchange with implantation of a multifocal or accommodating IOL, is a great alternative for older presbyopes and higher hyperopes, but many patients and practitioners consider it too invasive for a 45- or 50-year-old with a healthy crystalline lens.

The newest surgical option—corneal inlays—have been available since 2012 in Canada and, as of April 2015, are now available in the United States. The greatest advantages of inlays are that they are an additive technology and they can be removed in the event of patient dissatisfaction, a complication, or development of other conditions. Compared to lens surgery, the insertion procedure is less invasive. And, depending on the inlay, the near correction remains effective even as presbyopia advances.

In the pages that follow, we'll discuss how to effectively manage presbyopes' high demands for near and intermediate vision using corneal inlay technology.

Corneal Inlays

There are three different styles of corneal inlays, all designed for monocular implantation in the non-dominant eye: corneal reshaping inlays; refractive inlays; and small-aperture inlays. So far, only one (KAMRA small-aperture inlay, AcuFocus, Irvine, Calif.) is approved for use and commercially available in the United States. Therefore, much of this

Release Date: June 2015
Expiration Date: June 30, 2016
Goal Statement: The correction of vision in presbyopic patients has come a long way over the years, and the optometrist's role in comanaging these patients is increasing, so it is important for them to know what they can do, from educating patients, to counseling them and providing postoperative care. This activity takes a particular look at corneal inlays and explains how the everyday optometrist can turn the act of referring a patient for surgery into a practice builder through referrals and comanagement fees.

Faculty/Editorial Board: Sondra Black, OD, Paul Karpecki, OD, Eric Brooker, OD
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paper will focus on our clinical experience with that particular technology.

The KAMRA inlay (Fig 1) is a microperforated, opaque inlay that relies on the principle of pinhole optics to increase the eye's depth of focus by blocking unfocused light (Fig 2). It is 6.0 µm thick, with an overall diameter of 3.8 mm and a central aperture of 1.6 mm. This inlay is basically a focusing aperture or artificial pupil that is implanted into a small pocket in the cornea.

The procedure takes about 10 minutes to perform and requires only topical anesthesia. Better still, unlike LASIK, the KAMRA inlay can be removed. Worldwide, more than 20,000 have been implanted, and there are many published reports in the literature (see further reading, page 40).

Other inlays in the development and approval pipeline include the Raindrop Near Vision Inlay (ReVision Optics, Lake Forest, Calif.) and the Flexivue Microlens (Presbia, Irvine, Calif.) The Raindrop is a clear, 2.0-mm-diameter inlay that is positioned under a flap to reshape the central cornea for near vision. It is in late-stage clinical trials. The Flexivue Microlens is a refractive inlay. This 3.0-mm inlay has a small opening in the center to facilitate fluid exchange, surrounded by a central plano zone for distance and a refractive peripheral zone with variable add power. It is currently enrolling in phase III FDA clinical trials.

Although all three inlays share some similarities (e.g., monocular corneal placement, removability) there are significant differences among them that may affect patient selection, refractive targeting, surgical procedures, and results.

Inlay Results

It is important to know that the surgical procedure and postoperative care regimens have evolved considerably since the KAMRA inlay was first introduced in Canada, as is common with new technologies. For example, surgeons initially implanted the inlay under a thick LASIK flap, but have since moved to implantation in a deep stromal pocket created by an advanced femtosecond laser. The pocket approach has reduced the incidence of dry eye and improved refractive predictability and stability.

When best practices are followed (see sidebar) patients at Crystal Clear Vision (Dr. Black's practice) typically achieve J1 to J2 near and 20/20 to 20/25 intermediate and distance vision in the inlay eye, with excellent binocular uncorrected distance acuity.

Results from nearly 9,000 commercial pocket procedures performed around the world demonstrate that patients gain an average of three lines of near vision-

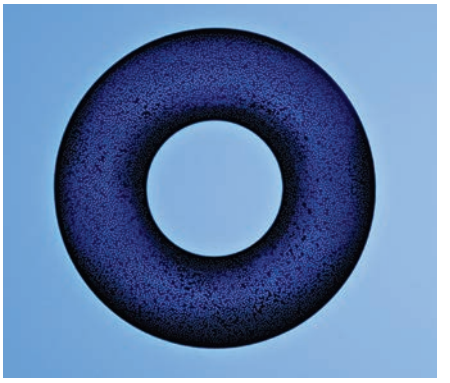


Figure 1: The KAMRA inlay is a microperforated, opaque inlay that relies on the principle of pinhole optics to increase the eye's depth of focus.

at one week and an additional line at one month, resulting in an average near UCVA of 20/28 (Fig 3).

With the pocket procedure, 95% of small-aperture patients in an international study were satisfied with the procedure and very few required reading glasses (Fig 4). It is a great source of comfort to patients and practitioners alike to know that the inlay can be removed if needed. That said, the removal rate is quite low. Globally, the removal rate is only 1.2% for pocket-based procedures, and studies show that 97% recovered their pre-inlay BCDVA within six months of removal.

Presenting Surgical Options

One of the most important things we, as optometrists, can do for our profession and for patients is to stay abreast of new technology. After all, with each new innovation comes the ability to better treat more patients. Plus, you never want a patient to learn about a technology somewhere else. It's always better for patients to hear about options from their own optometrist than to learn later from a friend or relative that there were surgical options their doctor didn't mention. Additionally, there is no one better suited for properly screening and selecting patients for an inlay procedure than optometrists who thoroughly understand their patients' needs, the binocular visual system and how it will be impacted by implantation of a presbyopic corneal inlay.

As referring optometrists, it is import-

Evolution of the Inlay Procedure

Since inlays were first introduced, both the surgical technology and the care procedures have evolved significantly. Elements of a state-of-the-art KAMRA inlay procedure today include:

Preoperative

- Rule out patients with even mild cataract. We use the AcuTarget HD instrument to objectively measure optical quality and rule out patients with optical scatter (OSI score >1.5) due to lens changes or retinal issues
- Prepare the ocular surface for surgery, usually with a course of topical cyclosporine or punctal occlusion
- Treat meibomitis and blepharitis aggressively

Intraoperative

- Ideal placement is in a deep pocket (200 µm or deeper)
- Pocket must be created with an advanced femtosecond laser to create a smooth stromal bed
- Ideal patient is between plano and -0.75 in the non-dominant eye with less than 0.75D of cylinder and close to plano in the dominant eye
- Use the AcuTarget HD instrument to facilitate precise centration
- Insert punctal plugs on the day of surgery

Postoperative

- Recommended postoperative steroid regime is a 3-month taper with additional time if the patient requires
- Emphasize ongoing use of cyclosporine and artificial tears to prevent dryness related problems

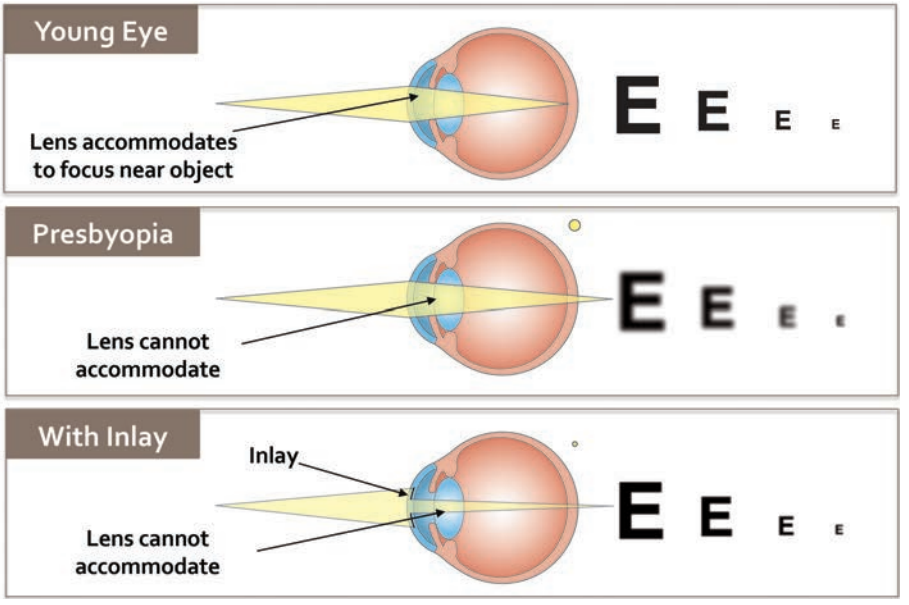


Figure 2: In a young eye (top image) the crystalline lens is able to accommodate to allow a patient to see clearly across a range of distances. As patients age and their crystalline lenses begin to lose their accommodative function (middle image) the range of vision is decreased and images become blurry. With a small-aperture inlay, only focused light rays are allowed to enter the eye, providing patients with a renewed range of vision and a reduction in blur (bottom image).

ant to understand the lifestyle needs and wants of our patients so we can educate them about the latest technological improvements that are available today to help them reduce their dependence on reading glasses. This positions the optometrist as a well-educated expert and patients appreciate the personalized care and approach from their doctor (Fig 5).

The difficulty with presbyopes is that their expectations of seeing well are extremely high. Active presbyopes, in particular, have very low tolerances for any visual impairment whatsoever. This presents a unique challenge because, on one hand, we want to present all of the options, but on the other hand, we don't want them to be disappointed with post-operative results.

In reality though, our hesitation to educate patients about the available options can do more harm than good. Presbyopes who have a desire to do away with their readers generally present with preconceived notions of what their options may be. Many have engaged in significant online research and they've gleaned random details from friends. As such, our first priority is to provide a real-world overview of the options, while reminding them that a thorough workup will be required to determine which procedure is appropriate for their particular situation.

As with all refractive surgery, it's important to set appropriate expectations before recommending a procedure, so assessing each patient's individual needs is essential. Consider the patient's career, hobbies and interests. As optometrists, we are generally well acquainted with the patient and are therefore ideally suited

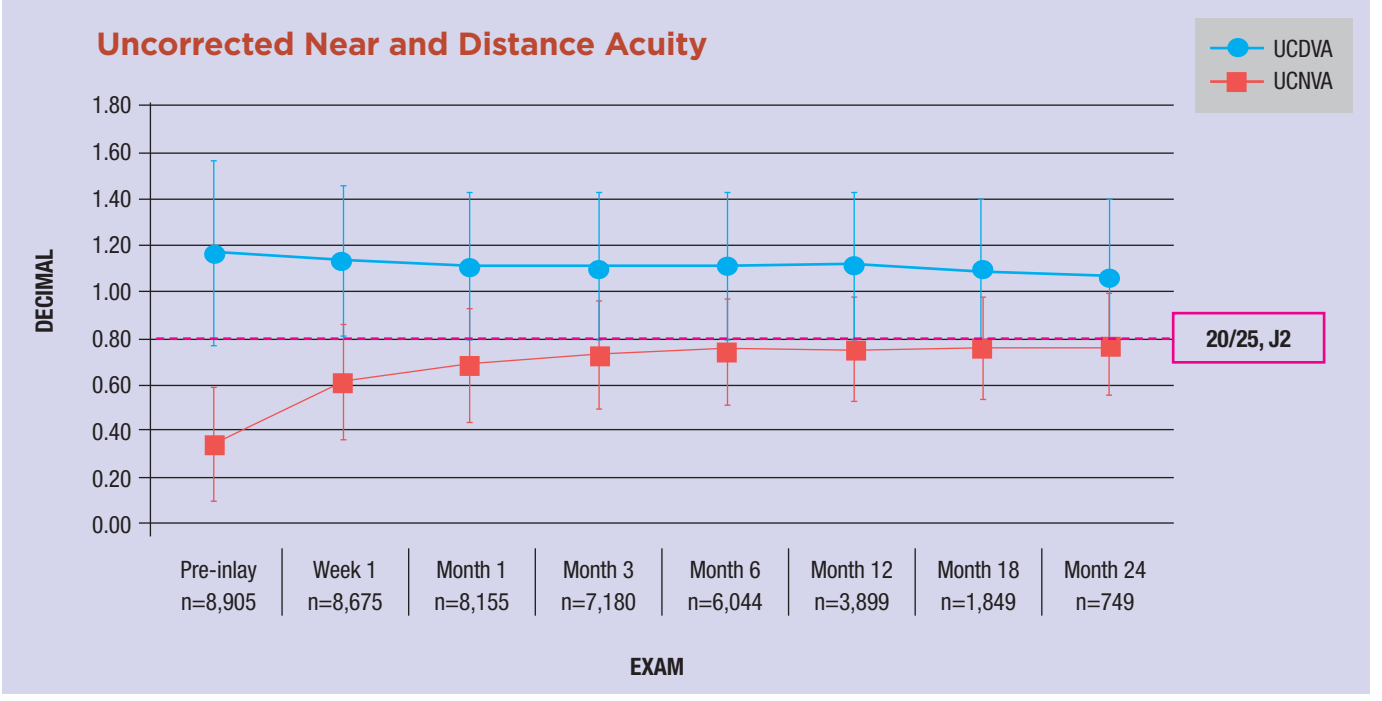


Figure 3: In 9,000 commercially performed procedures around the world, including more than 700 with two-year follow-up, the average UCVA is 20/28 (J2) for near and 20/20 for distance. Data from the Global KAMRA Data Registry.

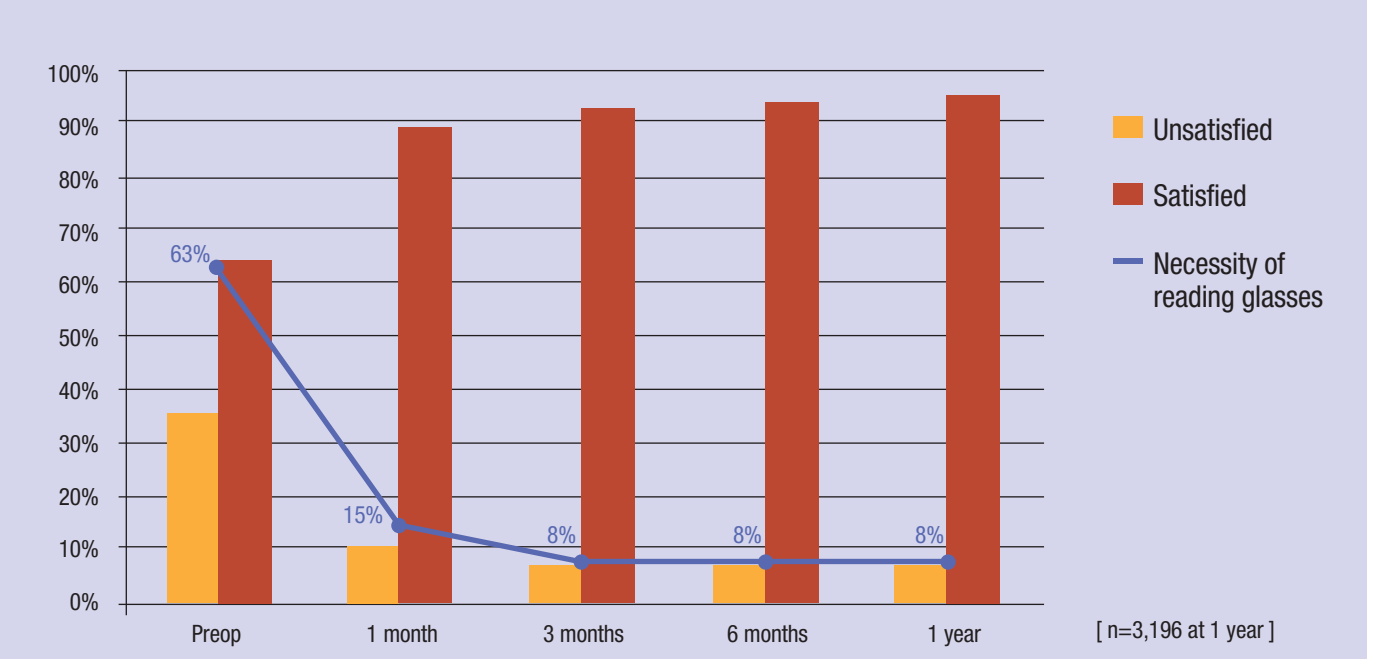


Figure 4: In a study conducted by Minoru Tomita, MD, 95% of the more than 3,000 subjects undergoing pocket implantation of the KAMRA inlay were satisfied with their vision at one year and only 8% still used reading glasses, most of them only occasionally (2%: often, 6%: sometimes). Data courtesy Minoru Tomita, MD.

to make appropriate recommendations based on lifestyle and personality. We also tend to already be aware of which patients are hypercritical and therefore poor choices for any elective procedure.

Educating patients about their options can be a strong practice builder. It will earn you the respect and loyalty of your patients. Reputable surgical practices will send patients back to you for follow-up and ongoing annual exams. Furthermore, the fees for comanagement of surgery can be significant—and the referrals from satisfied surgical patients even more so.

Pre-Operative Screening

In the United States corneal inlays are approved for the emmetropic presbyope. We look for similar criteria as for LASIK: good overall systemic and ocular health with no autoimmune disease; a healthy ocular surface and clear lens; and a healthy retina/macula that offers full visual potential. The ideal pre-operative refraction is between plano and -0.75. For someone who is just beginning to refer patients for this procedure and wants to start conservatively, it is a "slam-dunk" solution for 45- to 60-year-old emmetropes who want spectacle independence.

Generally speaking, if a patient is not a good LASIK patient, he won't be a good inlay patient. Basic requirements include a good ocular surface, a clear crystalline lens, sufficient corneal tissue, and normal topography. If a patient has an unhealthy ocular surface, it will affect the outcome. As such, you need to get the tear layer as good as it can get before referring a patient for any refractive procedure—including treatment with the KAMRA inlay.

If there is any dry eye disease, let the patient know that you need to optimize the ocular surface first and, in fact, will continue to do so after surgery. Look for any signs of dry eye disease and perform fluorescein staining and a blink test on KAMRA candidates. If tear quality is poor or dryness is present, treat it before referring the patient.

Additionally, if patients are suffering from blepharitis or meibomitis, this can significantly impact the tear film quality, which impacts the patient's vision. This should also be treated aggressively pre-operatively to improve the tear film prior to surgery and allow for a better post-operative outcome.

KAMRA inlay patients are viewing the world through a small aperture, so a good tear film is a prerequisite for high quality vision. Anything within that aperture—including small spots of punctate keratitis—can have a big impact on vision. At Crystal Clear Vision, we start patients on topical cyclosporine preoperatively, insert a punctal plug on the day of surgery, and recommend continued cyclosporine and artificial tears throughout the postoperative period.

Managing Expectations

To optimize outcomes and patients' satisfaction, it is important to properly screen and educate patients prior to KAMRA surgery. We advise you spend a fair amount of time counseling patients in order to appropriately set expectations.

The importance of preoperative education cannot be understated with all elective procedures. It is important not to promise miracles. No current technology gives presbyopic patients the ability to see exactly like a 20-year-old emmetrope, but we feel that corneal inlays come closer to that ideal than any other option.

Any symptom that you don't prepare a patient for pre-operatively will be perceived as a problem post-operatively. Talk to the patient about their goals and their visual demands and, based on that, make a recommendation.

Always explain the risks and recovery

period as well as the benefits. A good rule of thumb is to under-promise and over-deliver.

Once a decision has been made, set realistic, achievable expectations. This begins with making sure that the patient understands that age-related changes will continue to occur over the coming years and they may need to occasionally use reading glasses.

Let patients know that inlays can reduce their dependency on glasses, but they may still find that they will need glasses for very fine print or prolonged reading. They will need good lighting (a free flashlight app on their smart phone can usually take care of that).

Also, with any inlay, there is a longer healing time than with LASIK. Although some patients get an immediate postoperative "wow," most of them can expect to wait three to four weeks for full visual recovery. The recovery period will vary for each patient and is normally related to their healing speed and the efficiency of their endothelial function. Make sure that inlay patients understand that it will take time to achieve their best possible vision and they will need to take an active role in the healing and adaptation process by taking their drops and avoiding the use of reading glasses.

Comanaging an Inlay Patient: What to Expect

Due to longer healing times, postoperative care of an inlay patient is quite different from that of a LASIK patient. Patients at the longer end of the normal healing spectrum may need some reassurance that things are normal and will continue to improve with time. Remind them that the best vision is yet to come. It's also important to remind them to continue using their drops for best results.

You should also discourage patients from using readers or comparing their two eyes during the initial postoperative period, as this can slow down their neural adaption. Also check IOP at every postop visit. These patients are using topical corticosteroids so pressure spikes, while not common, are possible.

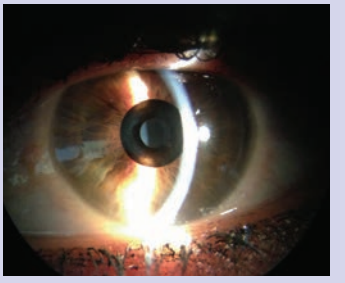
In cases where the patient is noncompliant with the postop regimen, tear film irregularities can put stress on the

Figure. 5: Presbyopic patients undergo extensive diagnostic testing, and an examination and consultation with Dr. Black to determine if they are good candidates for a corneal inlay.

The Doctor Becomes the Patient

By Sondra Black, OD

The quest for an effective presbyopic solution for my patients was greatly informed by my own experiences with presbyopia. I've treated more than 250 patients with small-aperture inlays and, by June 2013, I had seen enough to decide that the technology is a good fit for me personally, as well as professionally. As such, both myself and Crystal Clear Vision surgeon Jeff Machat, MD, have undergone KAMRA inlay implantation.



Post-op image of a corneal inlay in Dr. Black's eye.

In my case, I emphatically did not want to wear reading glasses. I had LASIK monovision around age 40 and did very well with that for several years, although I always kept distance glasses in my car for driving at night or in bad weather. The lack of binocularity bothered me, as did activities such as grocery shopping where the reading distance (labels on the shelves) was different from the near point I'd chosen for monovision. By my late 40s, I was getting headaches frequently. I found myself wearing readers for work more and more often, plus the driving glasses, which meant I was wearing glasses much of the time, and was not happy about that.

I had a KAMRA inlay implanted in my non-dominant eye. I blogged about the experience on our practice Facebook site and both Dr. Machat and I have continued to share our personal experience with patients. That firsthand experience certainly makes it easier for us to set accurate expectations and enhances our credibility.

Dr. Machat could see really well on the very first day, but it took me about six weeks to reach the point where I was comfortable with my visual acuity at all distances. We realized that part of the difficulty I was having was due to ocular surface problems. A course of topical cyclosporine therapy was needed to get me from 80% satisfied to 100% satisfied. Knowing that, we now put many patients on Restasis proactively.

Today I don't wear glasses at all. I do need good lighting to see well at near (in a dim restaurant, for example), but this is easily addressed with a smart phone flashlight app. My uncorrected distance acuity is 20/40 in the inlay eye and 20/20 with both eyes open. Binocularity is far superior and intermediate tasks are much easier than they ever were with monovision.

Courtesy Gunther Grabner, MD

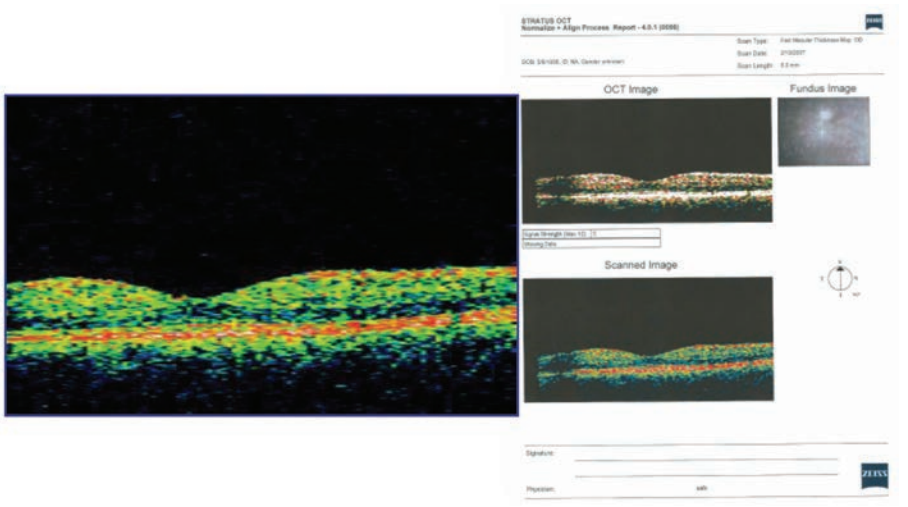


Figure 6. High resolution retinal imaging can be easily achieved even through a non-dilated pupil, as shown in these OCT images taken six months post-KAMRA inlay.

epithelial cells and cause a myopic shift and occasionally a "blue ring" or area of flattening over the inlay will be observed on topographical mapping. Another postoperative sign that optometrists should watch for in KAMRA patients is a "red ring" on topography. By itself, this is not cause for concern, but if accompanied by central flattening, haze formation, or a hyperopic refractive shift, then an enhanced corticosteroid treatment may be needed. With other inlay styles, postoperative haze may manifest with different signs due to the individual inlay designs and implantation depths.

Evaluating inlay centration and vision post-operatively

After inlay implantation, check patients' distance vision in a well-lit room and use an additional overhead light when measuring near acuity. It is important to know that autorefraction is unreliable in an inlay eye and will bias the measurement hyperopically. If refraction is necessary, a midpoint or red/green refraction is a better option given the patient's increased depth of focus. It is important to utilize the same refraction method at each visit so the patient can be monitored for relative changes to their refraction.

When viewing the inlay eye at the slit lamp, it is important to keep in mind that slit lamps do not provide a truly coaxial view, so the inlay will appear to be slightly off-center even when it is perfectly centered. The best method for understanding the positioning of the inlay is utilizing the advanced centration software on the AcuTarget HD diagnostic unit, which every surgeon will have in their office. The surgical center can inform you if there are any issues with centration of the inlay.

Examination through the inlay

It is possible to perform all aspects of the eye exam with an inlay in place. Lens opacities can be easily viewed with a dilated pupil. All retinal quadrants can be examined with a digital, wide-field lens or with Goldmann contact glass fundoscopy. Intraocular pressure measurements are no different in inlay-implanted eyes than at baseline, and gonioscopy imaging can be used to view the angle. Optic nerve and retinal nerve fiber layer evaluation is also easily achieved with the inlay in place.

Clinical studies suggest there is a slight overall decrease in visual field sensitivity after inlay implantation (~1.0 dB change from baseline) but measurements remain within normal limits, with no scotomas. This has been demonstrated with both Humphrey 30-2 field studies and with automated Goldmann perimetry utilizing the Octopus to compare inlay implanted eyes to patients' non-implanted eyes.

Future procedures

Retinal lasers may cause thermal damage to the inlay and surrounding tissue. Therefore, if laser retinopexy or focal or pan-retinal laser photocoagulation are indicated, the inlay should be removed prior to laser treatment.

Cataract surgery can be performed with the inlay in place, although manual

Further Reading

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surgery may be preferred over the use of a femtosecond laser for lens fragmentation. Fortunately, the choice of IOL is not limited by the inlay procedure. After the cataract is removed, the surgeon can implant a monofocal IOL and keep the inlay in place to maintain its benefits, or the inlay can be removed in favor of implanting a presbyopia-correcting IOL.

Summary

As primary care providers, it is our duty to educate patients and present them with vision correction options that might be a good fit. We are fortunate that these options have expanded with the recent approval of the KAMRA inlay in the United States. It truly is an exciting new option for presbyopic patients who want spectacle independence but aren't yet ready for cataract surgery. The visual results are very good, stereopsis is preserved, there are minimal compromises, and future exams and surgical choices are not significantly limited. Furthermore, corneal inlays are the only removable surgical option, and they have the great advantage of continuing to provide near vision even as presbyopia advances.

With current practices for implantation and postoperative regimens, optometrists will find that comanagement of inlay patients is fairly routine. Helping motivated presbyopes find a surgical solution that works for them represents a real opportunity for practice differentiation.

Dr. Black is vice president and clinical director at Crystal Clear Vision in Toronto, where she examines and counsels patients seeking refractive surgery, including laser, corneal and lenticular procedures. She spent 20 years in private optometric practice before joining a surgical practice in 2000.

Dr. Brooker is the founder of the Advanced Vision Institute in Las Vegas where he has focused his career on refractive surgery, presbyopia, corneal inlays, dry eyes, and medical eye care. He also is a consultant for several ophthalmic companies, providing innovative research and developing physician training programs worldwide.

Dr. Karpecki heads the ocular surface disease clinic and is director of clinical research at the Koffler Vision Group in Lexington, Ky.

A noted educator and author, he is the chief clinical editor of Review of Optometry. Dr. Karpecki is also past president of the Optometric Council on Refractive Technology and serves on the board for the charitable organization Optometry Giving Sight.

CE QUIZ

To obtain two hours of continuing education credit, complete the exam by recording the best answer to each self-assessment question online at: http://www.reviewoftoptometry.com/presbyopiaCE. Or, mail the Exam-ination Answer Sheet on page 42 to: Optometric CE, P.O. Box 488, Canal Street Station, New York, NY 10013. A minimum score of 70% is required to obtain a certification of completion. There is no fee for this course.

1. What distinguishes today's presbyopes from earlier generations?
a. They are wealthier
b. They are older
c. They have new intermediate-distance visual demands
d. They are less demanding

2. Which of the following surgical options for presbyopia is commercially available?
a. Multifocal ablation
b. Small-aperture inlay
c. Refractive inlay
d. Corneal reshaping inlay

3. Corneal inlays are intended for implantation in:
a. The dominant eye
b. The nondominant eye
c. Both eyes
d. The eye with the worse vision

4. Which of the following describes the Raindrop inlay?
a. Small-aperture inlay
b. Refractive inlay with power in periphery
c. Refractive inlay with power in centre
d. Changes corneal shape

5. Which of the following describes the Flexivue Microlens inlay?
a. Small-aperture inlay
b. Refractive inlay with power in periphery
c. Refractive inlay with power in center
d. Changes corneal shape

6. Which of the following describes the KAMRA inlay?
a. Small-aperture inlay
b. Refractive inlay with power in periphery
c. Refractive inlay with power in center
d. Changes corneal shape

7. The KAMRA inlay is best suited for implantation in which location?
a. Under a 100-µm flap
b. Under a 200-µm flap
c. In a 100-µm pocket
d. In a 200-µm pocket

8. In a large global registry study, how many lines of near vision were gained on average, following implantation of a small-aperture inlay?
a. One
b. Two
c. Three
d. Four

9. What is the removal rate for pocket-implanted KAMRA inlays?
a. 1.2%
b. 2.1%
c. 6.2%
d. 14.1%

10. For most patients, visual recovery following inlay implantation takes:
a. One to two days
b. One to two weeks
c. Three to four weeks
d. Three to four months

11. Which patients make the easiest first referrals for inlay surgery?
a. High myopes
b. Low myopes
c. Patients with glaucoma
d. 45- to 60-year-old emmetropes

12. Post-inlay implantation, a red ring on topography accompanied by hyperopic shift is an indication of what?
a. The inlay needs to be removed
b. Steroid treatment is needed
c. Ocular surface treatment is needed
d. The patient is developing corneal edema

13. Which of the following will be most accurate following inlay implantation?

CE QUIZ

a. Autorefraction
b. Standard manifest refraction
c. Cycloplegic refraction
d. Midpoint refraction

14. Which of the following can be performed through the inlay?
a. Gonioscopy
b. Fundoscopy
c. Retinal nerve fiber layer evaluation
d. All of the above

15. Which of the following cannot be performed with the inlay in place?
a. Panretinal photocoagulation
b. Visual field testing
c. Phacoemulsification
d. IOL implantation

16. What is the ideal refractive target for the inlay eye?
a. +0.25D
b. 0.00D
c. -0.25D
d. -0.75D

17. What is the ideal refractive target for the fellow eye?
a. +0.25D
b. 0.00D
c. -0.25D
d. -0.75D

18. How long is the postoperative steroid taper in the authors' practice?
a. One month
b. Three months
c. Nine months
d. Twelve months

19. Basic requirements to be considered for a KAMRA procedure include:
a. A clear crystalline lens
b. Sufficient corneal tissue
c. Normal topography
d. All of the above

20. In the authors' practice, how is the tear film treated in KAMRA patients?
a. Topical cyclosporine preoperatively
b. Punctal plug on the day of surgery
c. Post-op cyclosporine and artificial tears
d. All of the above

Examination Answer Sheet

Valid for credit through June 30, 2016

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New Surgical Options for Presbyopia

Directions: Select one answer for each question in the exam and completely darken the appropriate circle. A minimum score of 70% is required to earn credit.

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7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
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11.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
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17.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
18.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
19.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
20.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

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22. Related to your practice needs: ① ② ③ ④ ⑤
23. Will help you improve patient care: ① ② ③ ④ ⑤
24. Avoided commercial bias/influence: ① ② ③ ④ ⑤
25. How would you rate the overall quality of the material presented? ① ② ③ ④ ⑤
26. Your knowledge of the subject was increased:
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Rick Bay served as the publisher of The *Review* Group since 1991.

To those who worked for him, he was a leader whose essence was based in a fierce and boundless loyalty.

To those in the industry and the professions he served, he will be remembered for his unique array of skills and for his dedication to exceeding the expectations of his customers, making many of them fast friends.

Scholarships are awarded to advance the education of students in both ***Optometry*** and ***Ophthalmology***, and are chosen by their school based on qualities that embody Rick's commitment to the profession, including integrity, compassion, partnership and dedication to the greater good.

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Essential Procedures at the Slit Lamp:

'Capping Off' Cataract Surgery

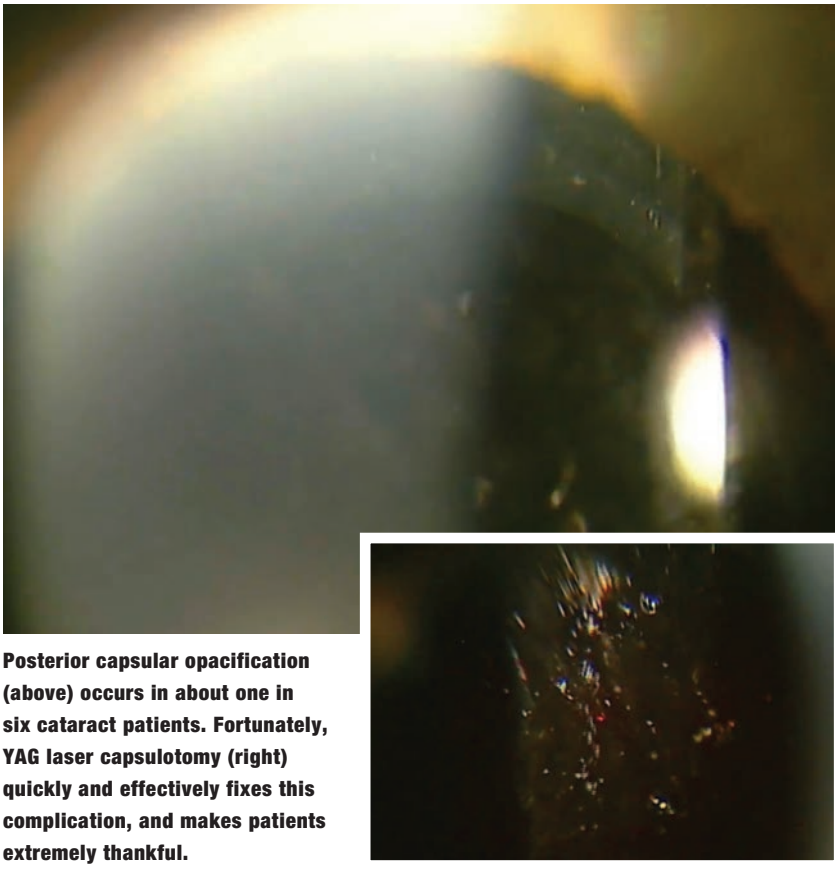
Posterior capsular opacification is the most common complication of cataract surgery. A YAG laser capsulotomy is often the fix. Here's how this quick and effective procedure is performed. **By Kelly Boucher, OD, Brittany Ellis, OD, and Nathan Lighthizer, OD**

"Doc, my cataract surgery went beautifully—or so I thought. I spent thousands of dollars on this premium lens implant, but I can't see well anymore. Can you help?"

Unfortunately, this patient has likely developed posterior capsular opacification, or PCO, behind the intraocular lens (IOL) implant. The good news is that a quick, simple, in-office laser procedure can fix this problem for good.

YAG capsulotomy is a relatively simple procedure that takes only minutes to perform and can make an enormous impact on a patient's vision. This article—the sixth in a six-part, print-and-video, how-to series—details the step-by-step procedure of a YAG laser capsulotomy.

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



Posterior capsular opacification (above) occurs in about one in six cataract patients. Fortunately, YAG laser capsulotomy (right) quickly and effectively fixes this complication, and makes patients extremely thankful.

Posterior Capsular Opacification

Modern cataract surgery involves making a hole in the anterior capsule to remove the lens, while leaving the posterior capsule intact. Unfortunately, this technique can leave behind lens epithelial cells on the anterior capsule and equatorial region. With time, these cells are believed to proliferate and migrate onto the IOL and the posterior capsule, creating a PCO. When enough of these cells build up, they become more opaque and can obscure or scatter the path of light entering the eye, causing patient symptoms. These cells can also cause wrinkling of the capsule, which can interfere with vision.[1]

Posterior capsular opacification is the most common complication of cataract surgery. Incidence rates used to be as high as 50%; but with improvements in surgical technique, IOL designs and IOL materials, the incidence has decreased to about 14% to 18%.[1]

The problem may not bother patients for months to years afterward, so they may forget that in-depth conversation you had prior to their surgery about the possibility of its development.

Blurry vision is just one of the symptoms that patients may experience. Other signs and symptoms include hazy or cloudy vision, decreased visual acuity, decreased contrast sensitivity, glare or halos around lights, double vision and asthenopia.

The current standard treatment for PCO is a capsulotomy using a laser.

YAG Capsulotomy

This technique is performed with a neodymium-doped yttrium aluminum garnet (Nd:YAG) laser to make an opening in the posterior capsule along the patient's visual axis. The Nd:YAG laser uses photodisruption to apply a series of focal ablations to disintegrate the offending tissues. The Nd:YAG laser does not have a thermal reaction or coagulative properties that may cause other complications.

Patient preparation involves instillation of dilating drops, an alpha-agonist to prevent intraocular pressure spike and proparacaine. The patient should be positioned comfortably at the slit lamp, with good fixation and head position.

Indications and Contraindications

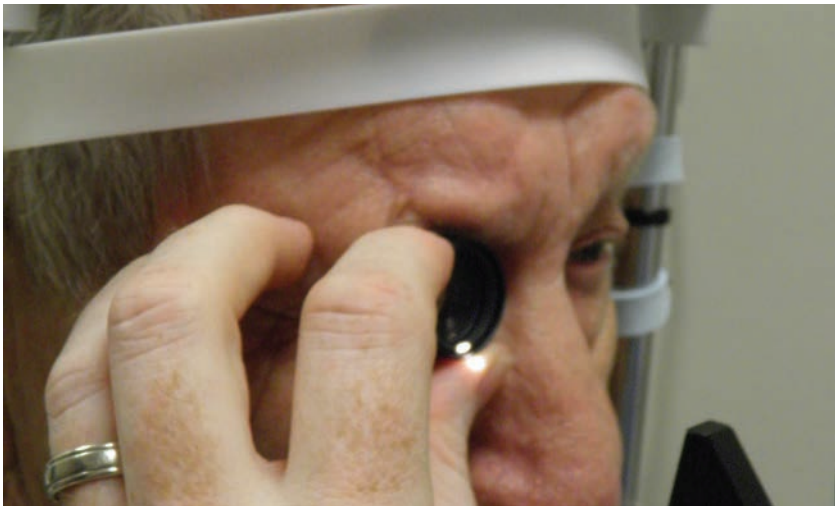
Consider a YAG capsulotomy when the patient's symptoms interfere with his or her activities of daily living or when the PCO restricts the ability to monitor or treat a patient's retinal condition. Thorough education of expectations after the procedure should be addressed with patients, especially if there is pre-existing retinal or macular disease.

Contraindications for YAG capsulotomies include corneal opacities, corneal scars, corneal edema, corneal surface irregularities, intraocular inflammation, cystoid macular edema, red eyes and patients who are unable to hold still. Retinal and/or macular pathologies—including macular hole, vitreomacular traction (VMT) and retinal tears—are other possible contraindications in which the risk-to-benefit ratio of the procedure needs to be considered.

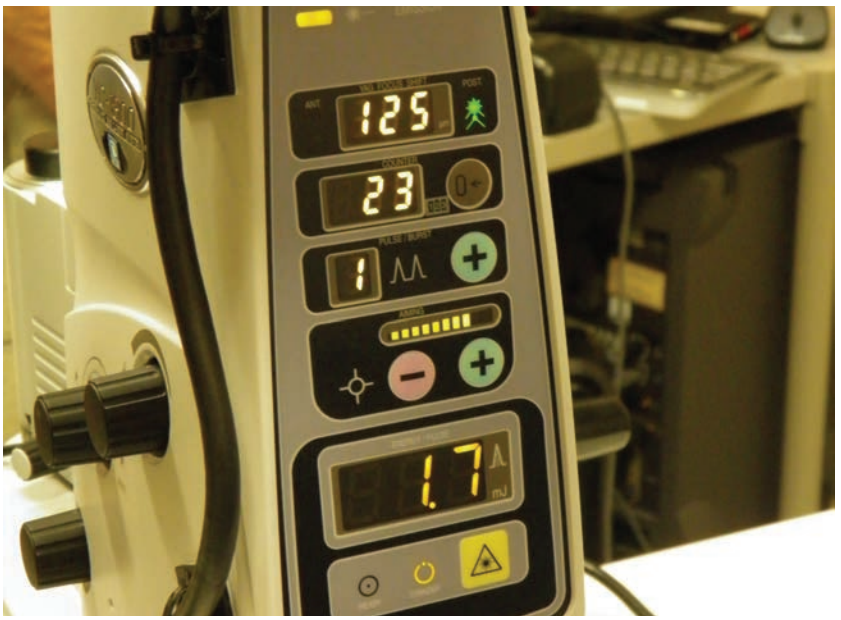
How YAG Capsulotomy is Performed

Prior to the YAG capsulotomy, perform a comprehensive exam including components such as best-corrected visual acuity, contrast sensitivity, glare testing and potential acuity. This should also include a thorough slit lamp exam and fundus evaluation including binocular indirect ophthalmoscopy. If there is concern about the state of the macula, a macular OCT is warranted.

Take a careful patient health history and physical information including allergies, current medications, respiration, pulse and blood



A capsulotomy lens may be used to stabilize the eye and allow for better lid control. It also magnifies the target and can allow for better visualization of the treatment area.



The Nd:YAG laser in action. This particular laser's settings display an offset of 125µm, a total number of 23 laser pulses used in the current procedure, a single pulse (1) per press of the button, and a power level of 1.7mJ.

pressure. A consent form reviewing indications, contraindications, risks and benefits, and alternative treatments should also be explained and signed by the patient.

After you've completed the examination and obtained the proper consent, the steps of a YAG capsulotomy include:

1. Instillation of dilating drops to get maximum dilation. Prior to dilation, the pupil position as well as the pupil size should be noted, in dim illumination. This is important for determining where and how large the capsulotomy should be.

2. Instillation of one drop of brimonidine or apraclonidine prior to the procedure to help control postoperative pressure spike.

3. Instillation of one drop of proparacaine in each eye to improve patient comfort and to minimize the blink reflex.

4. Adjustment of the laser settings:

• *Power/Energy* is initially set between 0.8mJ to 2.5mJ. Power can vary from laser to laser as well as from patient to patient and how dense the PCO is. The "golden rule" for any laser is to use the least amount of energy at the lowest setting to get the job done. Our typical starting energy is 1.2mJ to 1.8mJ.

• *Spot size and duration* are fixed, meaning they cannot be altered on a YAG laser.

• *Number of pulses per shot* should be one, which means one pulse of laser energy is fired at the capsule every time the button is pressed; however, this can be adjusted on a per laser and per patient basis. (For other laser procedures that employ the YAG laser, such as a laser peripheral iridotomy, the pulses may be increased to two or three, depending on the patient and the thickness of the iris.)

• *The offset of the laser* should be set between 125µm to 250µm posteriorly. The laser energy works anterior toward the front of the eye and therefore aiming slightly into the anterior vitreous will cause the energy to travel anteriorly and disrupt the posterior capsule and eliminate the PCO. So, the offset setting allows the operator to aim directly on the capsule with the helium-neon ("HeNe") aiming beams, yet in actuality the laser pulse will strike 125µm to 250µm posterior to that because of the offset.

Another benefit of the offset is it helps to prevent IOL pits.

5. The patient should be aligned comfortably in the slit lamp. Good

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Important Safety Information

Bimatoprost ophthalmic solution 0.03% has been reported to cause darkening (pigmentation) of eye color, eyelid skin, and eyelashes as well as increased growth of eyelashes. Pigmentation changes can increase as long as bimatoprost ophthalmic solution 0.03% is used. After stopping bimatoprost ophthalmic solution 0.03%, darkening of eye color is likely to be permanent, while darkening of the eyelid skin and eyelash changes may be reversible. The effects of increased darkening beyond 5 years are not known. When only one eye is treated, there is a possibility of eyelash changes in the eye treated with bimatoprost ophthalmic solution 0.03%. These changes may result in differences between the eyes in eyelash length, thickness, darkness, number of eyelashes, and/or direction of eyelash growth. These changes are usually reversible upon stopping bimatoprost ophthalmic solution 0.03% therapy. Patients should avoid allowing the tip of the dispensing container to contact the eye, surrounding structures, fingers, or any other surface in order to avoid contamination of the solution by common bacteria known to cause ocular infections. Serious damage to the eye and subsequent loss of vision may result from using contaminated solutions. In the event patients develop an intercurrent ocular condition (e.g., trauma or infection), have ocular surgery, or develop any ocular reactions, particularly conjunctivitis and eyelid reactions, they should immediately seek their physician's advice concerning the continued use of bimatoprost ophthalmic solution, 0.03%. Contact lenses should be removed prior to instillation of bimatoprost ophthalmic solution, 0.03% and may be reinserted 15 minutes following its administration. The most common side effects are conjunctival hyperemia, growth of eyelashes, and ocular pruritus.

Please see Brief Summary of Prescribing Information on the adjacent page.
For additional information, please visit www.lupinpharmaceuticals.com

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BIMA061115TRG

BRIEF SUMMARY OF FULL PRESCRIBING INFORMATION

BIMATOPROST Ophthalmic Solution, 0.03%

INDICATIONS AND USAGE

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

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

WARNINGS AND PRECAUTIONS

Pigmentation

Bimatoprost ophthalmic solution has been reported to cause changes to pigmented tissues. The most frequently reported changes have been increased pigmentation of the iris, periorbital tissue (eyelid) and eyelashes. Pigmentation is expected to increase as long as bimatoprost is administered. The pigmentation change is due to increased melanin content in the melanocytes rather than to an increase in the number of melanocytes. After discontinuation of bimatoprost, pigmentation of the iris is likely to be permanent, while pigmentation of the periorbital tissue and eyelash changes have been reported to be reversible in some patients. Patients who receive treatment should be informed of the possibility of increased pigmentation. The long term effects of increased pigmentation are not known.

Iris color change may not be noticeable for several months to years. Typically, the brown pigmentation around the pupil spreads concentrically towards the periphery of the iris and the entire iris or parts of the iris become more brownish. Neither nevi nor freckles of the iris appear to be affected by treatment. While treatment with bimatoprost ophthalmic solution, 0.03% can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly.

Eyelash Changes

Bimatoprost ophthalmic solution, 0.03% may gradually change eyelashes and vellus hair in the treated eye. These changes include increased length, thickness, and number of lashes. Eyelash changes are usually reversible upon discontinuation of treatment.

Intraocular Inflammation

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

Macular Edema

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

Bacterial Keratitis

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

Use with Contact Lenses

Contact lenses should be removed prior to instillation of bimatoprost ophthalmic solution, 0.03% and may be reinserted 15 minutes following its administration.

ADVERSE REACTIONS

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

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

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

USE IN SPECIFIC POPULATIONS

Pregnancy Category C

Nursing Mothers

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

Pediatric Use

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

Please see full Prescribing Information.

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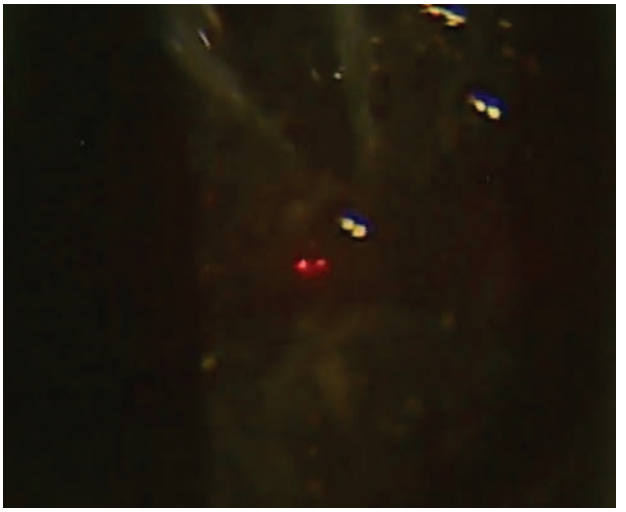
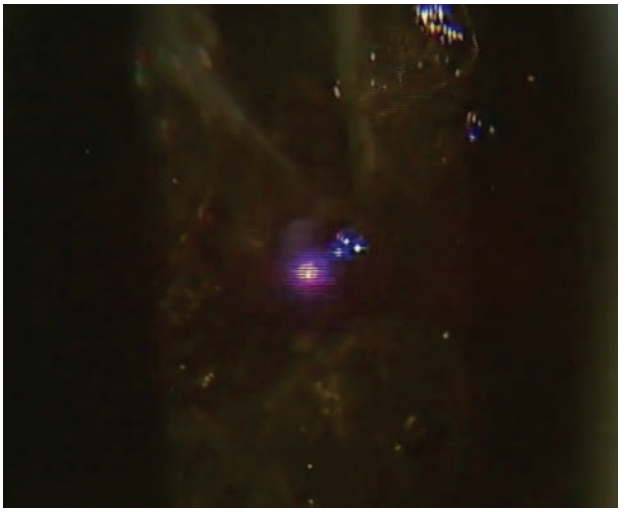
BIMA061115TRG

May 2015



The laser emits two separate beams (the double red dots) that are used for focusing just behind the posterior capsule.



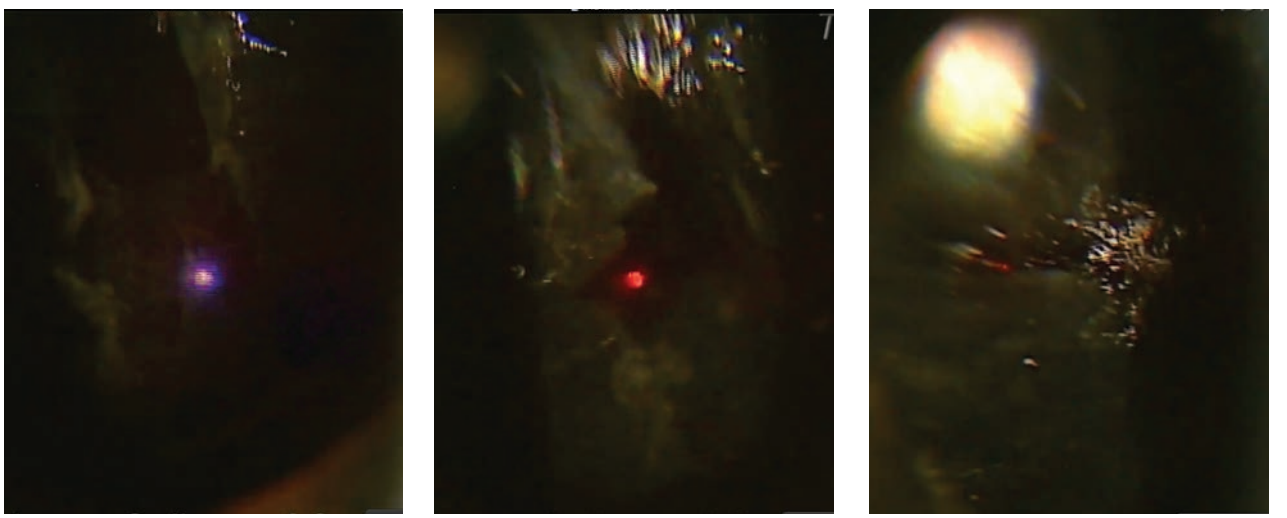
When the beams are focused, they appear as one. The pulse of laser energy hits the posterior capsule and ablates the tissue.

fixation and head position should be emphasized. Also, patients should be educated that they should not be alarmed if they hear small "snaps" or "claps" from the laser or see sparks of light during the procedure.

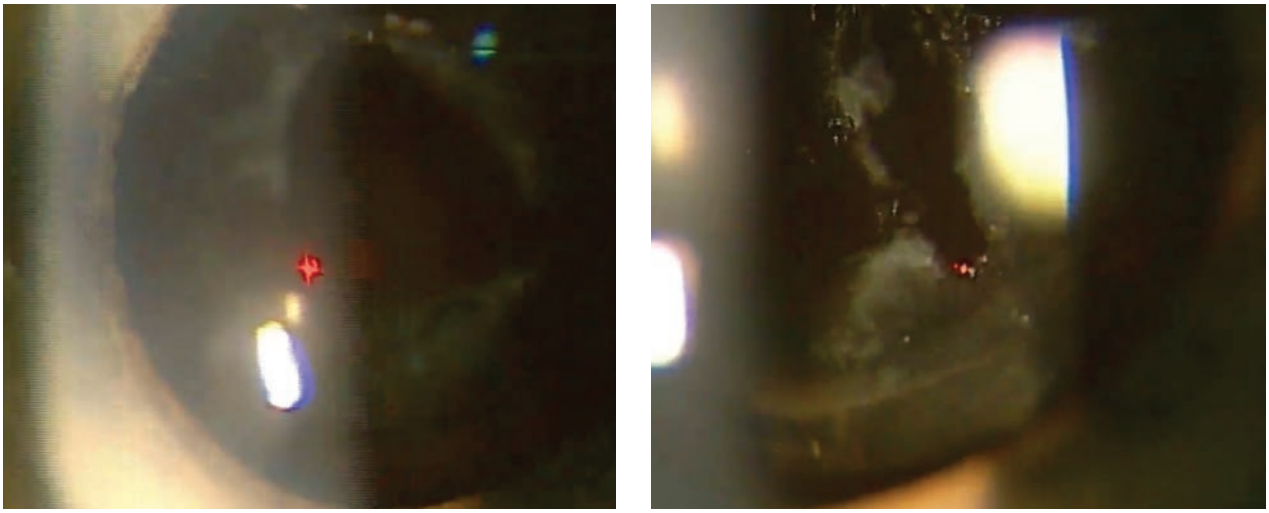
6. Some practitioners prefer to use a capsulotomy lens, which is a small lens similar to a flanged gonioscopy lens. The advantages of using the lens is to stabilize the eye and allow for better lid control; it also magnifies the target and can allow for better visualization of the treatment area. Just like with gonioscopy lenses, there can be bubble formation upon insertion and there may be reflections from the lens sometimes making the procedure more difficult. The choice of whether to use a capsulotomy lens or not is strictly up to the treating clinician and which laser is used.

7. With the laser turned on, the HeNe beams should be aimed at the posterior capsule. The HeNe laser emits two separate red beams that, when focused, become one. (Again, the laser energy will be applied 125µm to 250µm behind the posterior capsule because the offset of the laser is set behind the area of focus.)

8. Starting outside the visual axis, each laser shot is targeted such that one is placed adjacent to the next. There are multiple patterns in which the capsule can be opened;



Each laser shot is placed adjacent to the next. In this patient's procedure, the laser bursts are performed in a cross-like pattern—a simple and energy-efficient design—beginning from the top of the posterior capsule to the bottom, and then from left to right.



The posterior capsule should be inspected (left) without the laser lens and in retroillumination to ensure that there are no major strands or areas that need to be opened further. If so, the remaining "flaps" should be removed (right) to create a full central hole.

the most common is the cruciate, or cross-like, pattern, which creates a vertical and horizontal cross in the posterior capsule. Our preference with the cruciate pattern is to start at 12 o'clock and work our way down to 6 o'clock—this allows for good efficiency and usually lends itself to the least amount of laser shots and energy into the eye, and minimizes complications. During the procedure, no "laser burns" will be observed. Typically the examiner will just see an opening of the capsule as the procedure is performed. Once that is complete, we enlarge the opening in both the nasal and temporal aspects until a sufficient opening is created. Other patterns include the offset cruciate, horseshoe/arch shape, and circular.

9. Once finished, the posterior capsule should be inspected without the laser lens and in retroillumination to ensure that there are no major strands or areas that need to be opened further. Initially, the capsular bag may not appear fully open; but with time the flaps will settle out and expand, leaving a central hole in the bag. We typically try to make the opening about 4mm to 6mm, which is usually about the pupil size in dim illumination.

10. Thorough documentation with any procedure is vitally important. All the laser settings—including the number of shots, energy per shot, and total energy used for the procedure—should be documented.

11. Once finished with the laser, one drop of brimonidine or apraclonidine should be instilled to help control postoperative pressure spike.

12. Intraocular pressure should be checked again 30 to 60 minutes post-procedure to ensure that it is not rising. (Measuring IOP immediately after the procedure may give a false sense of security as IOP typically takes one to three hours to elevate, if it does rise post-procedure.) If IOP is acceptable and close to the preoperative intraocular pressure, the patient is sent home with a topical anti-inflammatory to be used for five to seven days to decrease inflammation after the procedure. Our preference is for maximal therapy, such as topical Pred Forte (prednisolone acetate 1%, Allergan) dosed QID.

Before leaving the office, the patient should be alerted to the potential for flashes of light, floaters, spots in their vision and/or drastically reduced vision, and educated to return to the clinic immediately if any of these occur.

13. The patient should be followed up in one week for a thorough dilated fundus exam as well as reassessment of visual acuity and IOP. If the desired outcome was reached at this visit, the other eye can be treated if necessary.

Complications

As with any ocular procedure, there are potential complications that the patient should be educated about. Increased intraocular pressure is a common finding after YAG capsulotomies. Increased IOP tends to maximize at one to three hours post-laser and return to close to normal within 24 hours due to the laser energy quickly dissipating from the eye. Patients with glaucoma or elevated intraocular pressures prior to the procedure are more likely to have a pressure spike and should be followed more closely in the postoperative period.

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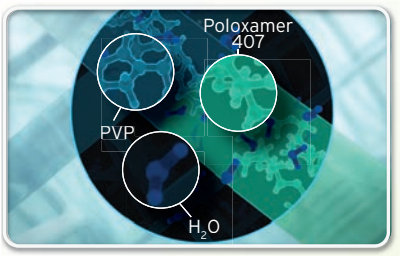

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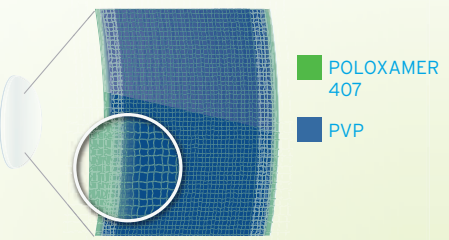

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REFERENCE: 1. Multiple-Packaged Lenses Comparison, Tyler's Quarterly – Professional Edition, September 2013 **2**. Twenty-two subjects participated in a randomized, double masked, contralateral eye study to evaluate water loss of Biotrue ONEday, 1-Day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

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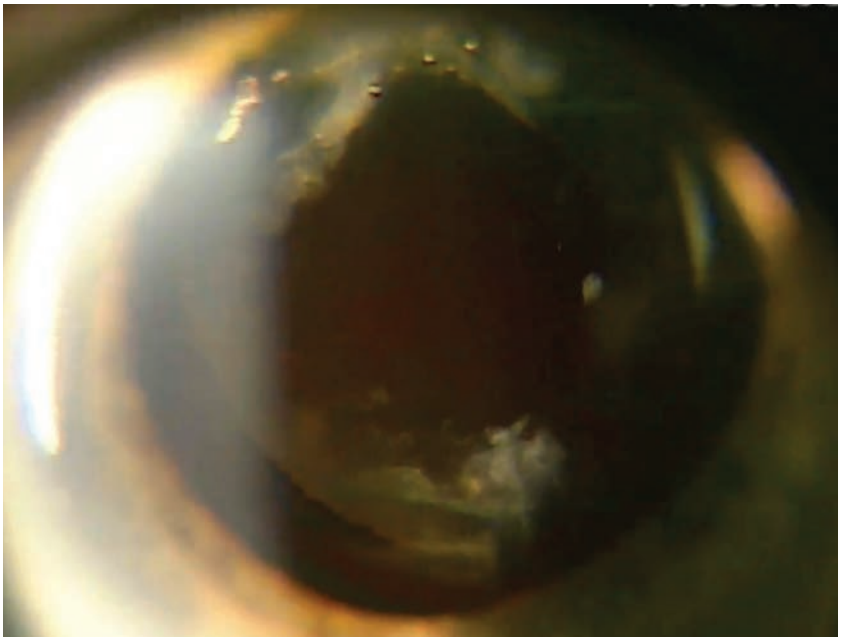

We typically make the final opening about 4mm to 6mm, which is usually about the pupil size in dim illumination. Thirty to 60 minutes post-procedure, the intraocular pressure should be rechecked to make sure that it is not spiking.

Other factors, such as increased total laser energy and larger capsulotomy size, are also associated with increased IOP.[2]

Postoperative inflammation in the form of an anterior uveitis is the second most common potential complication with any anterior segment laser procedure. This is managed with the topical anti-inflammatory for five to seven days after the procedure. However, it is incredibly rare to see a clinically significant iritis after a capsulotomy.

A more serious complication that can occur with YAG capsulotomy is a retinal detachment. While rare, it is important to perform a thorough dilated fundus exam prior to performing the procedure. Such a retinal detachment is believed to be caused from the loss of the intact posterior capsule or laser shockwaves, which induce vitreous changes.[2] Pre-existing retinal conditions such as high myopia or lattice with holes are risk factors for retinal detachments after surgery. Other factors such as larger capsulotomy size and higher total energy can also lead to increased risk for retinal detachments.[2]

Patients commonly experience an initial increase in floaters, but these should resolve within a few days as the tissues settle. Other complications that can occur after capsulotomy include cystoid macular edema, damage to the IOL in the form of pits, slight movement in IOL position, corneal edema, iris hemorrhage and vitreous prolapse. While complication rates are rare, it is essential to perform a comprehensive eye exam and to thoroughly explain all the risk and benefits to the patient prior to performing a YAG capsulotomy.[2]

Billing and Coding

The Current Procedural Terminology (CPT) code for the YAG capsulotomy procedure is 66821 with a 90-day global follow-up period. The International Classification of Disease (ICD-9) code for posterior capsule opacification is 366.53.

Posterior capsular opacification is a relatively common occurrence in the months to years after cataract surgery. It can leave patients frustrated and searching to find a solution.

A YAG laser capsulotomy is one of the most rewarding procedures that optometrists can be a part of. Optometrists in three states (Oklahoma, Kentucky and Louisiana) can now be certified to perform this procedure, and optometrists all across the country recognize and comanage the condition successfully with our ophthalmology colleagues.

A recent patient of ours came in for the one-week YAG-laser post-op, and nearly knocked us over with a bear hug stating that "she hadn't seen this well in years." Your patient will leave knowing that you gave them the best, most advanced care possible and will be raving about how you restored their vision to how it should be after cataract surgery. ■

Dr. Boucher is currently completing her residency in Ocular Disease and Family Practice at NSU Oklahoma College of Optometry.

Dr. Ellis is currently completing her residency in Optometric Management at NSU Oklahoma College of Optometry.

Dr. Lighthizer is the assistant dean for clinical care services, director of continuing education, and chief of both the specialty care clinic and the electrodiagnostics clinic at NSU Oklahoma College of Optometry.

1. Levin LA, Albert DM. Ocular Disease: Mechanisms and Management. Philadelphia: Saunders; 2010.
2. Aslam TM, Devlin H, Dhillon B. Use of Nd:YAG laser capsulotomy. Surv Ophthalmol. 2003 Nov-Dec;48(6):594-612.

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Final Sale: AVOIDING GLASS CHECKS

Glass checks and the inevitable spectacle remakes that follow can be frustrating. Here are 12 strategies to keep patients happy on the first try. **By Bill Potter, OD**

I recently chatted with an old friend, a fellow 30-plus year practitioner. Despite our varied experiences, we came to the same conclusion: spectacle prescription checks and remakes were some of the most frustrating and challenging situations encountered in practice. They are a constant reminder that refraction is more of an art than you might think.

Often, it can be challenging to reconcile a patient's subjective and objective findings to get the right prescription, despite the help of newer technology such as autorefractors. Some might assume that the refraction in the phoroptor is the refraction you should prescribe, but this philosophy might be the driving force behind many of the glass checks and spectacle remakes you see in your office.

Most seasoned clinicians have found some good answers—as much for our sanity as for economic survival—including some that go against the grain of traditional refraction. Abandoning the "white-knuckle" phoropter refraction experience, with its litany of choices and rapid changes in quarter-diopter steps, has helped put patients at ease and increased their confidence.

Handheld flippers at +0.50/-0.50 show the patient a proposed prescription.

Here are 12 strategies to help you avoid glass checks and spectacle remakes—some of which might challenge your traditional approach to refraction.

Do you agree or disagree? We welcome your comments. Write to ROeditorial@jobson.com with your thoughts on the strategies below.

1. Treat the Patient, Not the Refraction

Everyone in the medical field is aware of the step-wise approach to therapy: start with the simplest option and move forward from there. The same might be said of refraction, so carefully weigh patients' difficulties with adaptation to their new prescriptions.

While each practitioner has his or her own approach, I have found that adding more than one diopter of plus at near, or +0.75 at distance, unless there is a significant binocular disruption that high plus will solve, can be problematic. Your best retinoscopy, autorefraction and subjective measurement may all indicate a diopter or more of change, but in my experience your chances for prescribing success diminish as you make spherical changes that don't follow the above rule. Assure that your distance prescription improves visual performance over the prior prescription.

Also consider if the patient needs a prescription change to meet motor vehicle standards and job requirements. Although we have to prescribe with patients' adaptation in mind, it is mandatory to update prescriptions in a manner that allows qualification for state vision standards. We have an implied duty to warn patients who would not pass a state motor vehicle examination. Be sure to know your state's regulation on this. For example, in New Jersey it is a violation of patient confidentiality to report vision exam results without consent. This may vary tremendously in other states.

2. "Soften" Your Cylinder Axis and Power

We find that a very common cause of glass checks and remakes is when subjective cylinder axis and powers are prescribed without compromise. But a small change can help; I seldom change a cylinder axis by more than 20 degrees, even if it improves acuity. Spatial distortion increases as the cylinder axis is changed, especially in cylinders above +1.00D. Higher cylinder powers tend to make axis changes more difficult, so be conservative in changing cylinder axes if powers are high, and test to ensure that vision is improved.

3. Don't Overtest

The new prescription depends as much on the old one and history as it does on findings. Patient fatigue can be a significant problem in the classic subjective refraction, especially for some senior and systemically ill patients. Although subjective refraction takes little more than five minutes, the burden of accuracy you place on the patient can quickly become overwhelming

Glass Checks: A Team Effort

Glass checks are an excellent opportunity to collaborate closely with your opticians. In fact, their expertise can often resolve glass check issues without crowding your already busy schedule.

Patient Education

Whether it's difficulty adjusting to new progressive lenses, improper measurements, or a frame selection issue, opticians are a crucial first step in getting to the heart of the patient's problem.

"Education is a key factor, and discussing why one frame is better than another is critical. Weight, shape and size play a major role," says Ross Cappuzzo, MBA, president of Millennium Eye Care in West Freehold, NJ.

Difficulty adjusting to new progressive lenses, for example, is one of the main reasons Mr. Cappuzzo sees patients for glass checks. To help them better understand the changes and the role their frame selection plays, Mr. Cappuzzo uses the example of today's entertainment advancements.

"Using terms like digital and focal improvement can help patients understand," he says. "I like to compare today's digital lenses with the advances in HD digital TVs. The technology once worn (or viewed, as in the case of the new TVs) is dramatic" compared with what we have today.

Turning Trouble Into Success

Although you and your opticians may provide great patient education about lens changes and frame choices, some patients will inevitably return unsatisfied. And when that happens, the best solution is the same for all glass checks: exceptional customer service. Anything can be fixed, and ensuring patients they are understood and that solutions are available will go a long way to securing them as future customers.

Mr. Cappuzzo strongly recommends all practices have a glass check protocol on hand to help make this happen.

"Having a glass check protocol office policy in place that everyone in the practice understands is the key," Mr. Cappuzzo says. "It's a written policy of how we are to treat the customer and handle the glass check. We treat each glass check as an opportunity to demonstrate our skill level in the field of optics."

The first step in the protocol is ensuring the patient is directed to the right person—the optician. They can get to the bottom of the patient's issues and either solve them with proper education, expert frame adjustments or simply some much-needed reassurance. But the protocol also reminds staff not to tell patients "'try to get used to the glasses.' They can always decide to do that themselves."

Instead, staff should listen to the patient's complaint and respond accordingly—and always remember to document the encounter. Even when the patient needs to—or insists on—seeing you, the optician's documented glass check can make the appointment run a lot smoother when you are finally face-to-face with the patient.

and intimidating. I advocate for a brief event, with only a few demonstrations on each eye. Quickly go to a trial frame or flippers if results are in doubt. Leave the white-knuckle refractions back in training.

I have found that the most indecisive patients on subjective refraction tend to have more difficulty with changes in their prescription. At the same time, hypercritical patients are more likely to return with issues as well. Again, target the change to the patient's needs, and be cautious in changing if test results are vague. For example, a patient with no complaints and 20/25 acuity with a spherical prescription may actually get 20/20 by adding -0.50 of cylinder. But, patients have variable responses to the Jackson Cross Cylinder method of determining axis, with repeated contradictions. This would be a great case to defer cylinder prescription.

Halberg clips offer convenient demonstration of prescription options by holding the desired trial lenses in place.

4. Test With Trial Lenses Like Crazy

What a great technique. Establish binocularity and best-corrected acuity as is customary, then use trial lenses, preferably held over the habitual eyewear, in a waiting area or outdoors.

Lighting is a big issue, especially glare, and trying lenses in different lighting can solidify—or reject—your exam room results. Simulate habitual lighting whenever possible, especially for reading and computer work. That additional +0.75 that was so strongly accepted in the exam room may blur license plates and street signs.

Trial lenses with Halberg Clips (Keeler Instruments) allow you to demonstrate a new prescription over the habitual glasses without the strain of holding the lenses in place. Trial frames can sometimes be cumbersome and time consuming—small changes are often easily demonstrated with flippers and Halberg Clips.

5. Remember: Hyperopia is a Different Animal

How often have you had a hyperopic patient who was completely asymptomatic with an uncorrected distance acuity of 20/50? It happens to me almost every day. Pupil size, accommodation and visual demands all play a role in mandating less plus, or none, compared to our intuition. Better said, don't push plus on asymptomatic patients.

I believe that we tend to prescribe glasses for too many people based solely on classic refraction principles, with the resultant glass checks for patients who might struggle with adaptation.

Uncorrected hyperopic patients notoriously reject plus for distance. Trial frame a plus prescription that does not exceed a spherical equivalent of +0.75 for best adaptation and acceptance, but only if the patient is symptomatic and might be in danger of failing a motor vehicle vision exam.

6. Prepare for Progressive Design Changes

Digital progressives, with increasing patient customization, are greatly affecting our patient's comfort. However, the transition from an older design to a newer one is not always smooth.

Counseling, in advance of purchase, can make a huge difference in acceptance of the new glasses. I discuss an expected adaptation period of 10 to 14 days and emphasize the positive aspects. Being able to view distance, intermediate and near without having to remove or change eyewear is a real plus. The newer design may offer a wider, less distorted area for intermediate vision, for example. The perception of distortion will diminish as the patient's success grows.

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REFERENCES: 1. Data on file. Bausch & Lomb Incorporated. Fit Data. Q4 2014 **2.** Results from a 22-investigator, non-masked multi-site switching study of Bausch + Lomb ULTRA® contact lenses with MoistureSeal® technology, on 327 current silicone hydrogel lens wearers. After 7 days of wear, subjects completed an online survey. Subjects rated performance across a range of attributes. Preference comparisons represent only those subjects expressing a preference. Ratio is based on the average across the silicone hydrogel lenses represented in the study.

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Halberg clips minimize fatigue for both patient and doctor.

with a professional optician. He or she may have a better perspective on which progressive design produces the best results when converting from an older or lower-quality version.

Do your best to find the lens that produces less peripheral distortion, or perhaps offers an occupation-specific benefit.

7. Take into Account a Patient's Personality and Past History

This can be intuitive for some, but quite difficult for others. More precise patients can be resentful when we prescribe a change—even a small one—if they were asymptomatic. Adaptive difficulties will be attributed to the tiny prescription change, not the real culprit such as changing progressive design, frame size or base curve.

Demonstrating the prescription change via a trial frame, Halberg Clips or flippers can help tremendously in the patient's decision.

A patient's history of prior adaptation difficulties will sensitize you to be cautious on large changes in progressive design, frame or prescription.

Some patients have chronic difficulties in adapting to new glasses, and it is often appropriate to limit any changes in any aspect of the lenses. Often, difficult patients appreciate the improvement with incremental changes, and advising the patient on these issues demonstrates your thought process and will make for a more adaptable patient.

8. Respect Non-Habitual Wear

A patient who wears the prescription intermittently may have a harder time adapting to the changes.

Let's say the patient agreed to your +0.75 prescription last year. Driving is the only time the prescription is used, and we've fallen from 20/20 to 20/30 at this year's

Clinical Pearls

Courtesy of Joy Gibb, ABOC, Daynes Eye and Lasik, Bountiful, Utah

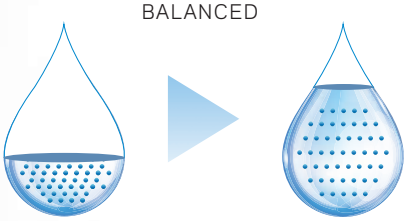
- Take the patient's lifestyle into consideration to understand how he or she wants the product to perform. For example, some patients may need a hypoallergenic material or a lightweight material because of thinning skin around the nasal area.
- If a patient chooses a frame that you know won't work, be honest and tell them why it's not a good choice and give them better options.
- Be up front about potential changes and create realistic expectations. If you take the customer from a large frame to a smaller framer, for example, you may need to change to a short corridor progressive lens, which can change how and what the customer can see and accommodate.
- Pre-adjust frames before taking measurements. Once the patient has put on the frames, ask if that is where they will be wearing them.
- Make sure temples are long enough and that the bridge fits well.
- Check the pantoscopic tilt of the frame before measuring.
- If you have repeat re-do offenders, invite them to participate in coming to an amicable solution. This can help them understand they have some responsibility in the choices they are making, particularly if they go against your specific recommendations.
- If you are noticing a lot of remakes, check your lab's monthly reports for clues. There may be a lack of training in the dispensers, issues coming from the lane during refraction, etc.
- Have standard operating procedures—such as a glass check protocol—in place to help reduce glass checks and remakes.

Dry eye relief starts with restoring balance

Elevated tear film osmolarity (osmolarity imbalance or hyperosmolarity) is one of the primary causes of dry eye symptoms.[1]

TheraTears® Dry Eye Therapy with Osmo-Correction™ corrects osmolarity imbalance to restore comfort with a unique hypotonic and electrolyte balanced formula that replicates a healthy tear.

WITH
OSMO-CORRECTION™
HYPOTONIC & ELECTROLYTE BALANCED



HYPEROSMOLARITY

Increased concentration of the tears leads to irritation and potential damage to the ocular surface.

HEALTHY TEAR

TheraTears® Dry Eye Therapy replicates the electrolyte balance of natural tears to restore comfort.

RESTORE + CLEANSE + NOURISH

thera tears®
THERAPY FOR YOUR EYES™

DRY EYE THERAPY
Restores Eyes Natural Balance™

theratears.com

AKORN Consumer Health

Reference: 1. Research in dry eye report of the Research Subcommittee of the International Dry Eye WorkShop (2007). *Ocul Surf.* Apr 2007; 5(2): 179-193.

exam—would you give a +1.75? Regardless of the patient's subjective response during the exam, this particular one diopter increase is a formula for a remake. The quarter-diopter per line of vision change is a good principle to start with, but it does not always correspond to the reality of the prescription acceptance or rejection.

9. Help Select the Right Frames

Proper frame selection is a key component in avoiding remakes. Modern spectacle prescribers are seldom involved in this process, and the disconnection sometimes leads to poor frame choices. Frame size and shape influence the nature of the fabricated lenses, and often can create visual discomfort. Did the patient have a fashionable, smaller eye size in the previous glasses? This might lead to disconcerting distortion if the patient chooses an aviator style for prescription sunglasses. Did the first-time progressive lens wearer select a frame that requires a short corridor design? This limits vision and creates maladaptation.

You shouldn't tell the patient to avoid changing frames, just to be sensitive to this issue in frame selection. A brief note on the prescription can give the optician extra sensitivity. I'll typically say, "Please note small habitual frame size when selecting new frame."

Communicate with the fabricating optician about any frame change issues. We comment on the prescriptions themselves with suggestions such as "minimize frame dimensions, as able," or "note computer monitor position in selecting progressive design." These simple comments have resulted in better communication that can dramatically limit remakes.

10. Consider a Patient's Working Distance

Modern computer workstations have created a challenge to spectacle prescribers. Variations in patient posture, monitor height, desk design and distance to screen can lead to extremely different lens designs and powers for patients with the identical prescription.

A patient may need a +2.25 add for a test distance of 16", but the reality of an eye-level computer monitor at 27" will produce a

rejected prescription unless you take the work distance into consideration.

A separate "computer" prescription, customized for a patient's workstation features, is tremendously helpful if patients are willing to purchase a dedicated pair of glasses for this purpose.

11. Focus on Patient Education

No matter what your approach to refraction may be, patient education is a must. It is important to demonstrate prescription changes to patients and let them decide if they perceive a benefit from the changes.

Explain the adaptation period necessary with some prescriptions, especially PALs for new presbyopes. This will ensure each patient leaves your office with a prescription he or she has agreed to, as well as an understanding of how you reached that decision together.

12. Remember: Perfection Can Be Subjective, Too

Refraction involves a physical measurement of a biological system. Just like any laboratory test or medical imaging, there can be variation and vague results. Accepting this will help you aim for a result that is perfect for your patient, even if it's not perfect from an objective testing standpoint.

This is the essence of optometric practice in terms of vision correction. No matter what modality is used, patient expectations must be shaped in order to promote better acceptance of the new prescription.

Avoiding glass checks and changes obviously benefits both the patient and your practice, but it can be harder than you think. Just as we are careful not to overmedicate drops and oral medications, we should take the same approach with eyeglass powers, axes, adds and lens designs. Although this may challenge some tenets of the traditional philosophy of refraction, most patients will appreciate conservative changes and will be less likely to return with complaints. ■

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6th Annual Retina Report

The Road to RETINAL WELLNESS

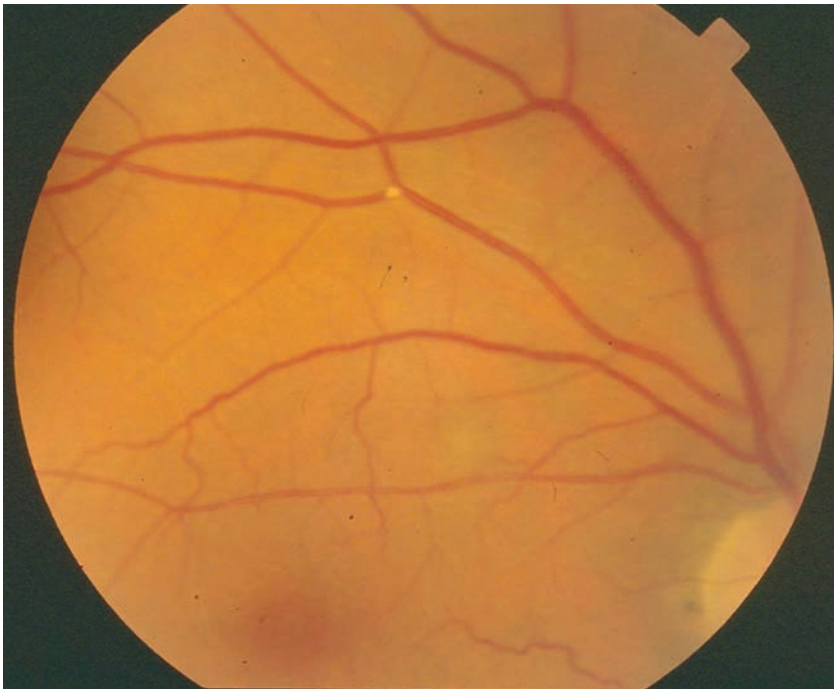
Optometrists can protect vision by talking about a healthy lifestyle. Join in the preventative care movement with these tips. **By Kimberly Reed, OD**

Patient communication and education is a cornerstone of a successful optometric practice. Consider the amount of time you spend each day discussing disease diagnosis and management with your patients. How much of that patient "face time" are you dedicating to promoting wellness and disease prevention?

Your patient base's demographics will certainly influence how you answer. But, if your practice habits mirror those of the overall trends in health care in the United States, you probably spend a lot more of your time and effort treating disease than providing education and strategies to prevent it. In fact, 88% of the US health care budget is spent on disease treatment, compared with a paltry 4% on prevention.[1]

Optometrists, as primary care providers, are in an ideal setting to encourage wellness. Specifically with regard to retinal health, factors such as obesity, diabetes, smoking and family history have a profound, direct impact on disease outcomes.

This article takes a closer look at why—and how—optometrists should widen our focus to incorporate disease prevention.



This fundus image shows a Hollenhorst plaque, which can develop in patients with high cholesterol.

Busting Wellness Myths

Shifting the focus to emphasize wellness instead of disease management isn't an easy task. Many of the obstacles come from health care providers themselves. Let's review, and debunk, some of the more common myths:

- *Myth:* "We should be emphasizing and educating patients about the best treatments for their diseases—talking about 'prevention' isn't as beneficial in the big picture."

Fact: About 50% of our health status is determined by lifestyle choices and wellness strategies. Genetics and environment only

contribute about 20% each, and access to care makes up the remaining 10%.[2] Consider this: If the average adult body mass index (BMI) is lowered by only 5% (for example, losing about 20 pounds for a 210lb, 5'10" tall person), obesity-related health care savings will amount to an estimated $29.8 billion in five years, and a staggering $611.7 billion in 20 years.[3]

Obesity and smoking combined contribute to nearly a million deaths in the United States alone each year.[4,5] These statistics represent our patients in our practices.

• ***Myth:*** "Anything a doctor says won't really make a difference. People are just going to continue eating what they want and avoiding exercising no matter what doctors tell them."

Fact: Studies show patients who receive counseling in a primary care setting, regarding better eating and exercise habits, more often took positive steps than patients who didn't receive counseling. And those steps led to more weight loss and more exercise in the patients receiving counseling.[6]

• ***Myth:*** Issues of weight and obesity are inappropriate topics of discussion for an optometrist and are best left to a general primary care physician.

Fact: Maybe. But, although virtually every set of clinical guidelines in existence recommends that physicians counsel overweight and obese patients about nutrition and weight management, this only occurs about once in every eight visits, according to a recent survey. Worse, only one-fourth of physicians who responded to that survey said they felt they had adequate training to provide diet and exercise counseling.[7] A mere one-third of medical schools meet the minimum number of hours in nutrition education recommended by the National Academy of Sciences.[8]

Also, many patients who are classified as overweight or obese avoid visiting a PCP for fear that they will be judged or ostracized for their weight.[9] At the other extreme, a separate study found only 39% of obese patients surveyed had even been told by a physician that they were classified as obese.[9] The gap between what needs to be done and what currently is being done must be filled. This is an "all hands on deck" public health crisis.

• ***Myth:*** "As an optometrist, I don't feel qualified to make dietary or weight loss strategy recommendations to my patients—and I'm not even sure I'm permitted to do so in my state."

Fact: If you feel under-qualified, that's okay! We, as primary health care providers, aren't expected to diagnose and manage every condition with which our patients present. We do, however, have the responsibility to connect patients to an appropriate care provider when a condition (or suspected condition) is outside of our scope of practice. A patient who has a high blood pressure reading along with retinal arteriolar attenuation isn't walking away from your office with a prescription for a diuretic and a systemic beta-blocker. Instead, she leaves knowing she may be at risk for ocular and systemic consequences, and in the

Retinal Wellness By the Numbers

• **$210 billion:** Current direct cost to the United States health care system every year for obesity-related diseases.[16]

• **$4.3 billion:** Current cost of obesity-related job absenteeism.[17]

• **$48 billion to $66 billion:** The estimated increase per year to care for people with obesity-related illness.[18]

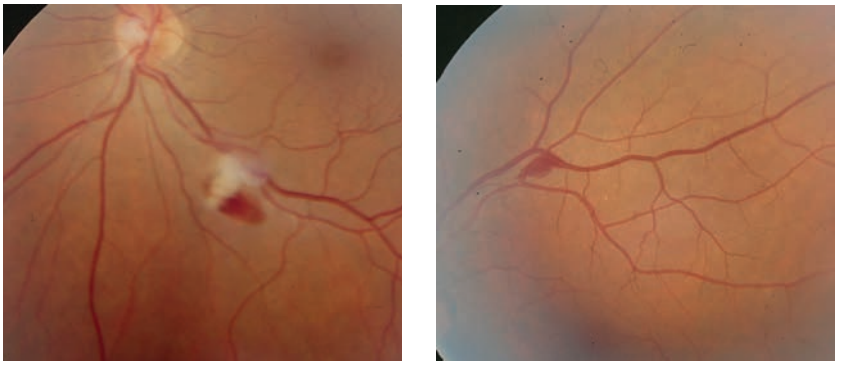
• **One-third:** The proportion of cancers that are directly or indirectly attributable to obesity or lack of physical activity.[19]

• **7.8 million:** The number of Americans diagnosed with diabetes mellitus type 2 (DMT2) in 1993.[20]

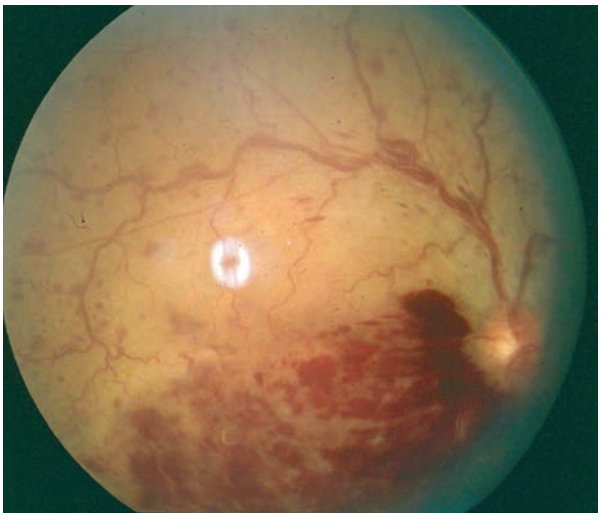
• **25.8 million:** The number of Americans diagnosed with DMT2 in 2012.[20]

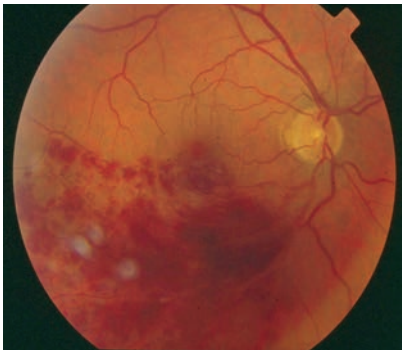
• **75%:** The proportion of patients with hypertension that can be attributed to obesity or overweight status.[21]

• **$580 billion:** The estimated total cost of loss of productivity due to obesity-related illness by 2030.[18]



These fundus images show retinal hemorrhages, a result of hypertension on the retina.





Retinal vein occlusions, shown in these fundus images, are most often seen as a complication of hypertension, or less commonly, diabetes.

best-case scenario, she already has an appointment with a primary health care provider to confirm the diagnosis and manage it. Likewise, a patient with a BMI of 33 and retinal arteriolar attenuation isn't visiting your optical to get appetite suppressants, a map to the gym and a two-week diet meal plan. However, that patient should leave with full knowledge of his ocular and systemic health risks, along with, ideally, an appointment with a qualified nutrition counselor, registered dietician or other medical or para-medical specialist who will recommend an appropriate eating and exercise plan.

Endocrinologists, retinologists, dermatologists and neurologists are among our referral targets—and obesity specialists should be as well.

• ***Myth:*** "Smokers already know the health risks. By me mentioning that they should stop smoking, I'm not only stating the obvious, but I also risk offending them."

Fact: A large meta-analysis of 41 studies of more than 31,000 smokers found an improved quit rate for smokers who were counseled to do so by a physician.[10] While the rate for unassisted quitting was 2% to 3%, counseling improved that rate by 1% to 3%. That might not sound like much as a percentage, but considering that tens of millions of Americans still smoke, that small percentage adds up to a meaningful number of your patients. The intervention by a health care provider does not need to be lengthy or involved to be successful—the same study found no difference in success based on the duration of discussion. Merely mentioning online support resources or suggesting a quitting technique such as nicotine gum was helpful.[10]

And remember, most people who smoke are ultimately successful in quitting only after multiple attempts.[11] So don't give up on your patients who fall off the wagon frequently.

• ***Myth:*** "Nobody who is overweight or obese wants to have it pointed out to them."

Fact: Few topics are as awkward to discuss with a patient as excess body weight. Many patients who have an unhealthy BMI want desperately to lose weight. They may feel isolated, frustrated or ashamed about their failure to do so.[12-15] Handled with compassion and sincerity, this is an opportunity to build a strong trust relationship with your patient, rather than to alienate him.

Identifying At-Risk Patients

For many patients who need a referral to another doctor, the protocols and practices are clear and long established. For example, say a 62-year-old male patient complains of severe headaches increasing in frequency, along with a stationary blurry gray spot in the upper right portion of the visual field. Even before the dilated exam and automated visual field, you already have a plan to refer this patient to neurology for imaging and further care.

But for patients with excess adiposity absent any evidence of ocular findings, the path is less clear. How do you broach the topic of discussion? On what exam finding are you basing your pronouncement that the patient needs additional care? For what, and to whom, are you referring the patient?

Bear in mind, a high BMI is an independent risk factor for central geographic atrophy in AMD.[22] Further, for patients with Type 1 diabetes, BMI is an independent risk factor for severity of diabetic retinopathy.[23] Several studies show a correlation between BMI and cataract.[24] While these conditions have complex and multifactorial causes, each presents a valid opportunity to open the dialogue about healthy weight with your patients.

Prevention Resources

To integrate wellness promotion into your practice, a bit of preliminary

Rx Specify 0.01% ☑ DAW/DNS

LUMIGAN® 0.01%
(bimatoprost ophthalmic solution) 0.01%

There is no FDA-approved generic version of LUMIGAN® (bimatoprost ophthalmic solution) 0.01%[1]

Important reasons to choose LUMIGAN® 0.01%— and to make sure your patients get it at the pharmacy

1. Proven efficacy in treating elevated intraocular pressure in a large clinical trial.[2]
2. Established tolerability with low discontinuation rate.[2]
3. LUMIGAN® 0.01% is the #1 dispensed branded glaucoma medication.[3]

Make sure patients get the treatment you've selected— specify 0.01% on every LUMIGAN® 0.01% prescription

INDICATION

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

IMPORTANT SAFETY INFORMATION

WARNINGS AND PRECAUTIONS

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

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

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

ADVERSE REACTIONS

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

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

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

ALLERGAN

LUMIGAN® 0.01%
(bimatoprost ophthalmic solution) 0.01%

LUMIGAN® 0.01%
(bimatoprost ophthalmic solution)

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

INDICATIONS AND USAGE

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

CONTRAINDICATIONS

None

WARNINGS AND PRECAUTIONS

Pigmentation: Bimatoprost ophthalmic solution has been reported to cause changes to pigmented tissues. The most frequently reported changes have been increased pigmentation of the iris, periorbital tissue (eyelid) and eyelashes. Pigmentation is expected to increase as long as bimatoprost is administered. The pigmentation change is due to increased melanin content in the melanocytes rather than to an increase in the number of melanocytes. After discontinuation of bimatoprost, pigmentation of the iris is likely to be permanent, while pigmentation of the periorbital tissue and eyelash changes have been reported to be reversible in some patients. Patients who receive treatment should be informed of the possibility of increased pigmentation. The long term effects of increased pigmentation are not known.

Iris color change may not be noticeable for several months to years. Typically, the brown pigmentation around the pupil spreads concentrically towards the periphery of the iris and the entire iris or parts of the iris become more brownish. Neither nevi nor freckles of the iris appear to be affected by treatment. While treatment with **LUMIGAN®** (bimatoprost ophthalmic solution) 0.01% can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly *[see Patient Counseling Information (17.1)]*.

Eyelash Changes: LUMIGAN® 0.01% may gradually change eyelashes and vellus hair in the treated eye. These changes include increased length, thickness, and number of lashes. Eyelash changes are usually reversible upon discontinuation of treatment.

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

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

Bacterial Keratitis: There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products. These containers had been inadvertently contaminated by patients who, in most cases, had a concurrent corneal disease or a disruption of the ocular epithelial surface *[see Patient Counseling Information (17.3)]*.

Use with Contact Lenses: Contact lenses should be removed prior to instillation of **LUMIGAN®** 0.01% and may be reinserted 15 minutes following its administration.

ADVERSE REACTIONS

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

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

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

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

USE IN SPECIFIC POPULATIONS

Pregnancy: Pregnancy Category C

Teratogenic effects: In embryo/fetal developmental studies in pregnant mice and rats, abortion was observed at oral doses of bimatoprost which achieved at least 33 or 97 times, respectively, the maximum intended human exposure based on blood AUC levels. At doses at least 41 times the maximum intended human exposure based on blood AUC levels, the gestation length was reduced in the dams, the incidence of dead fetuses, late resorptions, peri- and postnatal pup mortality was increased, and pup body weights were reduced.

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

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

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

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

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

OVERDOSAGE

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

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

NONCLINICAL TOXICOLOGY

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

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

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

PATIENT COUNSELING INFORMATION

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

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

Handling the Container: Instruct patients to avoid allowing the tip of the dispensing container to contact the eye, surrounding structures, fingers, or any other surface in order to avoid contamination of the solution by common bacteria known to cause ocular infections. Serious damage to the eye and subsequent loss of vision may result from using contaminated solutions.

When to Seek Physician Advice: Advise patients that if they develop an intercurrent ocular condition (e.g., trauma or infection), have ocular surgery, or develop any ocular reactions, particularly conjunctivitis and eyelid reactions, they should immediately seek their physician's advice concerning the continued use of **LUMIGAN®** 0.01%.

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

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

APC87BO14 based on 71807US14.

Rx only

ALLERGAN®

work is required. Find your local smoking cessation and weight management centers. A number of not-for-profit organizations with online resources are offered free to the public.

Many hospitals have free or low-cost smoking cessation programs. Many well-established weight reduction programs are available almost everywhere, either "live" or online. Some insurance programs cover weight and nutrition counseling. You can research these benefits for the plans that are prevalent in your area.

Specialty weight loss centers are featured at many hospitals, although many of these specialize in bariatric surgery. In most cases, to qualify as a candidate for surgery, patients must meet certain criteria (BMI over 35 or 40, for example, or a combination of a minimum BMI with one or more comorbid diseases).

Use caution when recommending weight loss centers that aren't based on sound scientific principles. Opt instead for physicians or facilities that offer a combination of behavioral modification, menu planning with sustainable and realistic food choices, physical activity and pharmacologic support where needed.

Supplements

Educate yourself, if you haven't already, regarding the various eye-specific supplements available. Remember, disease prevention and wellness promotion isn't a one-size-fits-all program. Nutritional counseling, including the recommendation of specific supplements, is an important part of the optometric practice and should not be overlooked. In addition to the familiar practices of recommending omega-3 supplements for dry eye and carotenoids for AMD, there are also diabetes-specific supplements that may greatly benefit your diabetic patients. Patients with multiple systemic conditions may have higher micronutrient intake needs. Also, consider that many medications deplete certain key nutrients.[25–27]

Who to Counsel About Weight and Physical Activity?

- Patients with cardiovascular or respiratory disease (DM, hypertension, high cholesterol, etc.).
- Patients with BMI >30 regardless of ocular or systemic health or history.
- Patients with BMI >25 with an obesity-related health condition (metabolic syndrome, sleep apnea, diabetes, CHD, hypertension, some cancers, etc.).
- Patients with BMI >25 and a family history of obesity-related conditions.
- Patients with BMI >25 with an AMD or a family history of AMD.
- Women with a waist circumference >35 inches even with BMI <25.
- Men with a waist circumference >40 inches even with BMI <25.

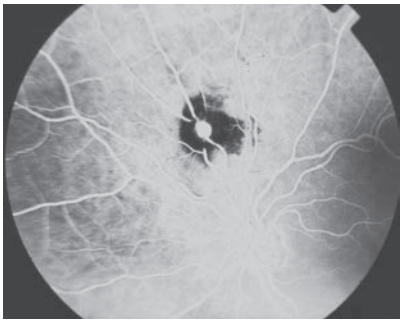
Talking Wellness

Case One

Here's how I handled a few actual case examples in which wellness promotion was appropriate.

Presentation: A 24-year-old Hispanic male presented with complaints of distance and near blur for six months. He reported "good health," and took no medications or supplements. His most recent medical exam was more than eight years ago. Family history was positive for Type 2 diabetes and hypertension in both parents (and his father had suffered a mild MI at age 50). His calculated BMI was 44. He had mild myopic astigmatism and was correctable to 20/20 in each eye. The remainder of the examination was without pathology.

Patient education: After addressing this patient's visual evaluation, I politely requested to address his weight issues. "The health status of your eyes today is good," I said. "However, you fall into a higher-risk category for developing several other eye and systemic diseases based on your BMI. Is it okay if we talk about that?"



Retinal macroaneurysm, as seen in this fluorescein angiography image, is another possible consequence of uncontrolled hypertension.

At this point, the patient acknowledged his weight problem and admitted the reason he didn't see his doctor for regular checkups; he was afraid the doctor would "yell at him" for being so heavy. He said he had made several attempts at weight loss and, despite losing up to 50 pounds each time, always gained the weight back. He said he had given up on it.

I resumed my counseling: "Would you like to talk with someone about the methods that you have tried before and see if there are other techniques that might work better for you? There are many medications now that can be beneficial for weight loss. You might also be a candidate for weight loss surgery that your insurance may even cover under certain conditions. Can I give you some information to take home and consider? Whatever you decide, please know that we are here to support you and your health, so if you decide against

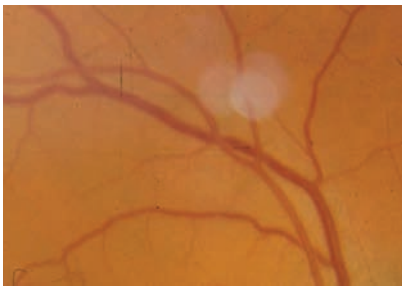
seeing one of these doctors, we still want you to come back for your eye checkups. This is a judgment-free zone."

The key to successful communication in this delicate area is to focus upon health risks rather than body size. Establish the reasons for your concern first, and then seek agreement from your patient to discuss the topic. This important step is essential to a positive outcome. Not every counseling attempt is going to result in an immediate positive result. Give patients a safe space to partner with you in evaluation their health needs. Remember, you may be the only health care practitioner who has made a meaningful, compassionate effort to help address the issue. In my own experience, patients are usually relieved and grateful that someone took an interest, instead of being resentful or defensive.

Nearly always, once the subject is broached, I gain important additional history and insight into the patient's health status that otherwise would not have been revealed. (For more tips, visit www.stopobesityalliance.org/wp-content/themes/stopobesityalliance/pdfs/STOP-Provider-Discussion-Tool.pdf)

Case Two

Presentation: A 55-year-old black female presented with a complaint of sudden vision loss in the left eye. She had high cholesterol and hypertension, and reported good compliance with a daily "water pill." BCVA was 20/20 OD and hand motion OS. She had full to finger counting visual fields in both eyes, but central vision was absent in her left eye. Blood pressure was 150/102. Her right eye's anterior and posterior segment health was unremarkable, aside from arteriolar constriction and A/V nicking; the



Arteriovenous nicking, seen in this fundus image, is seen as one of the earliest signs of hypertensive retinopathy.

left fundus showed a large (2.5 by 2.5 DD) central area of intra- and subretinal blood covering the central posterior pole adjacent to an apparent retinal arterial macroaneurysm superior-temporal to the macula. Her BMI was 31.

Patient education: In a case like this, focus on the impact to the patient's vision. Here's what I told her: "The vision loss you are experiencing in your left eye is likely not going to be permanent. The blood that is partially blocking your vision will most likely clear over time. The more important issue is the cause of the bleeding, which is most likely your high blood pressure, and it is high today. With your permission, I'm going to write a note to your doctor to let her know what we found today, and to suggest that in addition to considering ways to lower your blood pressure, that she work with us to help lower your BMI to a healthier ratio. I know you told me before that it seems like you have so much weight to lose that it's too much of a challenge, but believe it or not losing just 5% of your body weight will decrease your risk of disease consequences."

Case Three

Presentation: A 54-year-old white female presented with complaints of diplopia in upgaze for four days. She was hypertensive but unmedicated. She has smoked a pack of cigarettes per day since high school, and had tried many times to quit, but it never "stuck." She displayed signs consistent with an incomplete third nerve palsy without pupil involvement. Her blood pressure was 174/100.

Patient education: "I know this is something you've heard before, but the most important thing we can do for you overall is to help you succeed in smoking cessation. I have several resources for you, including other doctors who can prescribe helpful medications. Today, the more immediate problem is to make sure there isn't something more serious than high blood pressure causing your eye problems. To be on the safe side, I'm going to order some imaging studies of your head and eye areas. Assuming that comes back normal, we will work together with your doctor to lower your blood pressure and help you kick the smoking habit for good."

Healthy Patients

Finally, don't overlook your healthy patients who do maintain a BMI in the healthy range. A few words of encouragement and acknowledgement of their healthy habits may reinforce their hard work and dedication, and increase the odds of maintaining those habits for a lifetime.

"Promoting wellness" in the optometric practice encompasses a variety of factors, including smoking cessation, achieving and maintaining a healthy body weight, proper nutritional intake, regularly getting adequate sleep, stress reduction and avoidance of high risk activities. Shifting to a prevention perspective instead of waiting for

disease to develop before treating it might seem awkward at first, but ultimately can result in better patient health outcomes. ■

Dr. Reed is an associate professor at Nova Southeastern University College of Optometry in Fort Lauderdale, Fla. She teaches and writes extensively about ocular disease, ocular pharmacology and nutrition.

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Imaging the Choroid?

There's an App for That

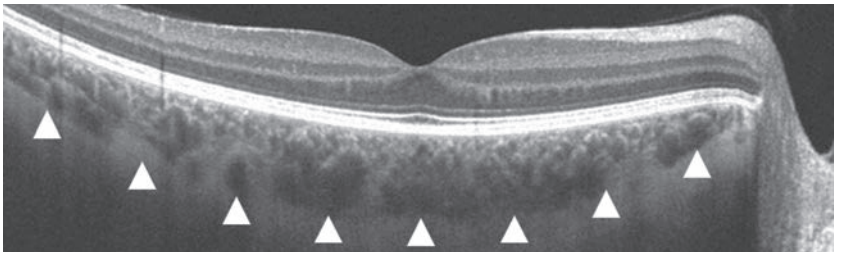
EDI-OCT technology brings this structure into focus, giving doctors the chance to diagnose diseases earlier and with greater precision.

By Carolyn E. Majcher, OD, Andrew S. Gurwood, OD, Joseph J. Allen, OD

Until recently, the choroid's inaccessibility—essentially buried beneath the retina—has made it a little understood anatomical structure. But this thin layer between the sclera and the retina, the "middle coat" of the eye, is vital to ocular health. It supports the outer layers of the retina by supplying nutrients and removing waste. Choroidal melanocytes absorb intraocular scattered photons (light). The superfluous flow of blood through the choroid assists in the removal of heat derived from the metabolism of phototransduction. Also, the suprachoroid lamina provides a safe route of travel for the long posterior ciliary arteries and nerves as they course toward the anterior aspect of the globe.

Choroidal analysis, in addition to retinal imaging, can provide supplemental information regarding disease progression. Thinning of the choroid is an indicator of advancing stages of nonexudative age-related macular degeneration (AMD) and correlates with the rate of visual field loss in eyes with normal tension glaucoma (NTG).[1,2]

Today, we're able to image deeper into the eye than ever before, allowing us the opportunity to evaluate choroidal thickness and morphology both for the benefit of patient treatment and for a better understanding of retinal diseases.



Gray-scale EDI-OCT of a healthy eye. The white arrows correspond to the choroid/scleral junction.

The technology making this possible is enhanced depth choroidal imaging, a function of optical coherence tomography (EDI-OCT). The clinical uses of EDI-OCT include detection and monitoring of pathologic alterations in choroidal thickness, differentiation between diseases with similar clinical features, and precise choroidal measurements. As this technology finds its way into our exam lanes, we can take on a greater role in patient management, one previously in the sole purview of the retina MD.

This article outlines the capabilities of EDI-OCT and the research that's helped us get to this level of care.

The Development of EDI

EDI was pioneered by ophthalmologists Ron Margolis, MD, and Richard F. Spaide, MD, in 2009.[3] Before their work, OCT imaging of the choroid was virtually impossible because of poor light source penetration through the densely pigmented retinal pigment epithelium, light scatter by the choroidal vasculature itself, limited axial resolution and motion artifacts.[3] Using the Spectralis (Heidelberg Engineering), Drs. Margolis and Spaide found that they could more effectively view the choroid by inverting the image.[3,4] Originally,

they accomplished this by simply positioning the patient slightly closer to the machine.[3,4] By convention, the zero delay line is located at the top of the imaging screen and represents the area of most precise focus.[3,5] By inverting that image, the choroid/sclera interface is placed closer to that zero delay line, improving the scan quality of the deeper, posterior structures.[3] EDI-OCT penetrates an additional 500µm to 800µm deeper compared with traditional OCT imaging.[6]

EDI-OCT can now be easily performed with just the click of a button on most OCT models that have new software upgrades, including the Spectralis, Cirrus HD-OCT (Carl Zeiss Meditec), and RTVue (Optovue).[6] The highest quality image of the choroid that can be obtained using commercially available machines is accomplished by combining EDI-OCT with image averaging.[6] When using the Cirrus HD-OCT, this involves combining the five lines of the HD 5-line Raster scan into one line (0mm spacing).[7]

Other Choroid Imaging Modalities

In addition to OCT-EDI, swept-source OCT (SS-OCT) and image averaging OCT techniques are used to improve visualization of the choroid.[1]

SS-OCT imaging uses a variable wavelength, frequency-swept laser light source.[1,2] Adding a longer-wavelength source allows even deeper tissue penetration without sacrificing the resolution of vitreoretinal structures that are better imaged at shorter wavelengths.[1] Another advantage of SS-OCT imaging is that interference patterns are more efficiently detected by photodiodes as opposed to conventional spectrometers.[1,2] The result is an axial resolution of 5.3µm with an acquisition speed of 100,000 to 400,000 A-scans per second.[3]

Image averaging refers to the technique of overlying multiple B-scan images of the same retinal location.[2] Since noise is random, combining many scans effectively cancels out the "speckle" or "static" present in each image.[2,4] This increases the signal-to-noise ratio, which sharpens and enhances retinal and choroidal features.[2] The quality of image averaging is improved with either eye tracking (Spectralis, Heidelberg Engineering) or Selective Pixel Profiling software capable of evaluating all of the pixel data to construct the best possible image (Cirrus HD-OCT, Carl Zeiss Meditec).[4-7] When using the Cirrus HD-OCT, image averaging is maximized by combining the five lines of the HD 5-line Raster scan into one line (0mm spacing).[7]

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Normal Choroidal Morphology

The thickness of the choroid varies throughout the posterior pole. In healthy eyes, the choroid is typically thickest beneath the fovea, where its average thickness ranges from 262µm to 354µm.[3,8-10] The surrounding superior and inferior choroidal quadrants within the macular region are generally thicker than the nasal and temporal quadrants.[10] The temporal choroid is thicker than the nasal choroid, which decreases rapidly toward the optic nerve.[3,8,10,11] Generally, the superior choroid is thicker than the inferior choroid.[10] This thinning of the inferonasal macular choroid marks the location of the embryonic optic fissure and may represent a normal "relative coloboma" or "knitting area."[10,12-14] Additionally, the thinning of the choroid between the fovea and the optic nerve may predispose the formation of peripapillary atrophy.[11] In the optic nerve, the peripapillary choroid is thinnest inferiorly and may contribute to the pathogenesis of glaucoma in susceptible cases.[4,11,12]

Influencing Choroidal Health

Many factors influence choroidal thickness—most significantly, age.[10] In patients older than about 60, choroidal thickness progressively decreases by 4µm to 5µm each year.[8,15] Although great variation exists between individuals, the resultant mean subfoveal thickness in individuals older than age 60 is 197µm.[8,10] Age has little effect on choroidal thickness in younger patients.[5] This age-related thinning of the choroid is likely mirrored by a reduction in oxygen and metabolite supply to the retinal pigment epithelium (RPE) and outer retina, which may play a role in the development of AMD and other degenerative retinal conditions.[3,8,11] Studies have already demonstrated a correlation between macular choroidal thickness and best-corrected visual acuity, highlighting the functional dependence of the photoreceptors on choroidal support.[1,15,16]

On average, healthy males have a 7% greater choroidal volume, which may explain the greater incidence of AMD among females who have thinner choroids at the outset.[11,15]

Other influential factors on choroidal thickness are refractive error

and axial length.[10,15] Studies have demonstrated a negative correlation between choroidal thickness and degree of myopia.[8,10,15] Similarly, increasing axial length is also associated with a decrease in choroidal thickness.[10,11] In eyes with myopia of greater than 1.00D, subfoveal choroidal thickness is expected to decrease by approximately 15µm per diopter of myopia and 32µm for each 1mm increase in axial length.[15]

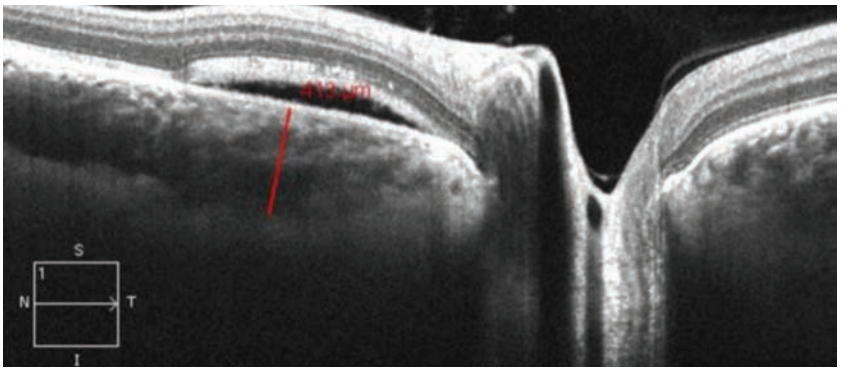
Other biometry measures associated with a decrease in choroidal thickness include shallower anterior chamber, thinner lens and steeper cornea.[15] Interestingly, neither hyperopic refractive error nor retinal thickness correlate well with choroidal thickness.[9,12,15]

It is well known that the choroid exhibits a pattern of diurnal fluctuation that may be related to fluctuations in choroidal blood flow given that the choroid is not autoregulated.[17,18] Investigators measured choroidal thickness over a 24-hour period and found that the choroid was generally thicker between 3 a.m. and 9 a.m. and thinnest between the hours of 3 p.m. and 9 p.m.[18]

A change in posture at night likely causes a gravitational elevation in venous hydrostatic pressure, resulting in engorgement of choroidal veins and an increase in choroidal blood volume.[19] No correlation between intraocular pressure (IOP) and choroidal thickness has been authenticated.[18]

Outer Retinal Diseases

• ***AMD.*** The invaluable functions of the choroid (waste removal from the RPE and photoreceptor nutrient supply) suggest that choroidal dysfunction is likely involved in the pathogenesis of AMD.[20,21] For instance, a 2011 study reported a decrease in mean subfoveal choroidal thickness in patients with exudative AMD (195µm) and nonexudative AMD (213µm) compared with age-matched healthy eyes (272µm).[22]



EDI-OCT showing central serous chorioretinopathy and neurosensory retinal detachment demonstrating an increase in nasal peripapillary choroid thickness (413µm).

Similarly, a 2013 study of choroidal thickness in eyes with nonexudative AMD found that worsening of the disease is associated with a progressive decrease in subfoveal choroidal thickness.[1] As expected, this study also reported a negative correlation between the subfoveal choroidal thickness and visual acuity, highlighting the importance of choroid health and visual function.[1]

Additional research measured choroidal thickness in eyes with early AMD either with or without reticular pseudodrusen, an interlacing yellowish pattern often found in the superior perifovea in some AMD patients.[23,24] The authors reported that the presence of reticular pseudodrusen is associated with a decrease in subfoveal choroidal thickness by approximately 19%.[23] This suggests that both the presence of reticular pseudodrusen and decreased choroidal thickness may be risk factors for progression to advanced AMD.[23,24]

Using laser Doppler flowmetry, new studies demonstrate a decrease in foveolar choroidal blood flow in eyes with nonexudative AMD correlating directly with the severity of the disease.[21,26,27] In fact, according to one study, decreased choroidal blood flow precedes conversion to exudative AMD and vision loss.[27] This decrease in choroidal blood flow and volume may account for the decrease in choroidal thickness found using EDI-OCT.[21] These findings may indicate the need for serial measurements in newly discovered and advancing cases.

Analysis of choroidal thickness not only plays a role in assessing the risk of developing AMD and AMD progression, but may also aid in differentiating between AMD and other diseases of choroidal circulation such as central serous chorioretinopathy (CSCR), polypoidal choroidal vasculopathy (PCV) and adult-onset foveomacular vitelliform dystrophy with fluid accumulation.[28-30] EDI of the choroid has increased our understanding of the pathogenesis of AMD and, with the advent of automated OCT choroidal thickness measurement, has the potential to reduce the time and labor costs associated with AMD management.[31,32]

• ***Central serous chorioretinopathy.*** CSCR is caused by increased choroidal hyperpermeability and breakdown of the outer blood retinal barrier, which leads to exudative/serous detachment of the neurosensory retina from the RPE.[33] Several studies using EDI-OCT documented significant increases in choroidal thickness in eyes affected with acute CSCR.[34-36] One study

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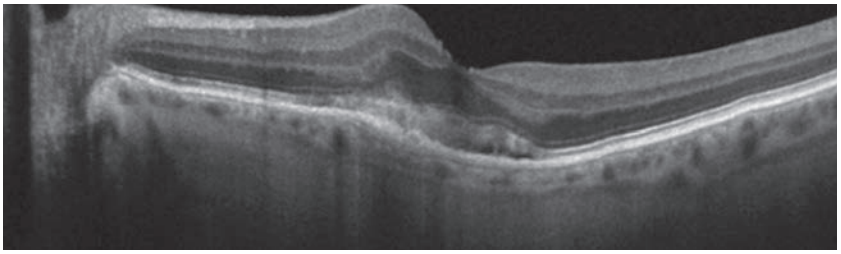

Gray-scale EDI OCT of an eye with exudative AMD and a perifoveal choroidal neovascular membrane with overlying subretinal fluid. Note the severe thinning of the subfoveal and nasal choroid.

found a 125µm increase in mean subfoveal choroidal thickness in eyes with CSCR compared with age-matched controls.[35] These findings are also supported in principle by investigators who found an 85% increase in choroidal thickness compared with age-matched controls.[35] This increase in choroidal thickness suggests that elevated hydrostatic pressure within the choroid may be a key pathophysiological factor in CSCR.[37]

In cases of chronic CSCR, treatments of laser photocoagulation or photodynamic therapy (PDT) or both may become necessary. Indeed, a 2010 study reported a 59µm decrease in mean choroidal thickness at one month following PDT treatment in eyes affected with CSCR.[38] A later study, using half-dose PDT, reported a mean decrease of 74µm at one month that was sustained to one year in 13 CSCR patients.[39,40]

With the use of EDI-OCT technology, choroidal thickness can be a useful parameter to assess CSCR activity both before and after treatment.

• ***Polypoidal choroidal vasculopathy.*** PCV is a primary disease of the choroid resulting in recurrent exudative and hemorrhagic pigment epithelial detachments with associated serous macular detachment.[40-43] The definitive diagnosis is made by indocyanine green angiography, which reveals multifocal choroidal hyperfluorescence, dilated and hyperpermeable choroidal veins, and a branching choroidal vascular network with adjoining polypoidal vessel dilatations.[40-44] Although the clinical manifestations and genetic mutations associated with PCV are similar to AMD, PCV is more common among Asian and black patients as compared with white patients.[42,43,45] PCV is less aggressive than AMD and the visual prognosis is better than that of exudative AMD.[40,46]

Several studies have shown the subfoveal choroid is bilaterally thicker by 80µm to 210µm in patients with PCV compared with healthy controls.[40,41,46] Further, the choroidal thickness decreases following treatment with PDT.[47] Additionally, investigators found a positive correlation between the diameter of the largest choroidal vessel lumen and the increase in choroidal thickness.[41] This suggests that thickening of the choroid in PCV is likely due to vessel dilation and engorgement rather than extravasation of fluid into the interstitial stroma via hyperpermeability.[40,41]

Some postulate that the dilation of large choroidal vessels may be caused by venous stasis, an abnormality in autonomic innervation to the choroid, or an increase in ocular perfusion pressure.[40,41,46] Research demonstrates that the mean ocular perfusion pressure, defined as the difference between mean arterial blood pressure and venous pressure, is significantly elevated in eyes with PCV.[46] These findings agree with previous research suggesting that systemic hypertension, which elevates ocular perfusion pressure, is a risk factor for PCV.[46,48] Increased ocular perfusion pressure puts mechanical stress on the fragile choroidal vessels, contributing to vessel dilation and increased hydrostatic pressure within the choroidal vasculature favoring fluid flux out of the vessels and into the interstitial stroma.[46,49]

Evaluating subfoveal choroidal thickness using EDI-OCT technology may be useful as a method to differentiate between PCV and AMD (associated with choroidal thinning).[50] In one study, eyes with a subfoveal choroidal thickness of 300µm or greater were five to six times more likely to have PCV.[50] Given the discrepancy in choroidal thickness values, it is likely that AMD and PCV have diverse etiologies and that PCV is not a subtype of AMD as previously thought.[40,41]

In addition to an increase in choroidal thickness, EDI-OCT often shows, in cases of PCV without hemorrhage, a separation of the RPE from Bruch's membrane referred to as the "double-layer sign."[41] The space between the RPE and Bruch's membrane is moderately hyperreflective and may be composed of vascular networks, polypoidal lesions, sub-RPE hemorrhage or cloudy fluid.[41,51]

In non-hemorrhagic cases, research shows Bruch's membrane remains intact.[41] Researchers postulate that hemorrhaging in PCV may be caused by ruptures of Bruch's membrane.[41]

• ***Retinitis pigmentosa.*** Retinitis pigmentosa is a genetic disease of variable inheritance causing destruction of the outer retina and RPE.[16]

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It classically results in night blindness and constriction of the peripheral visual field.[52] Several studies employed EDI-OCT to investigate choroidal thickness in eyes with retinitis pigmentosa. The results showed a decrease in subfoveal choroidal thickness by 27% to 36% compared with unaffected controls.[16,52] One group found subfoveal choroidal thickness significantly correlated with acuity and duration of the disease.[16] They, and others, postulate that the decrease in choroidal thickness may be due to a reduction in choroidal blood flow velocity and volume found in eyes with retinitis pigmentosa.[16,52,53]

Inner Retinal Disease

• ***Diabetic retinopathy.*** Diabetic retinopathy is the leading cause of new cases of blindness among adults ages 20 to 74.[54] Clinically, the pathophysiology of diabetic retinopathy is attributed to vascular changes within the retina. However, many histological studies identify similar signs of vascular damage within the choroid.[55-58] One study describes a significant decrease in choroidal blood flow within eyes of Type 2 diabetics, particularly those with macular edema.[59] EDI-OCT technology has helped to reveal decreased choroidal thickness in various stages of the disease.[60,61]

One study described significant thinning in patients with diabetic macular edema and in patients with treated proliferative diabetic retinopathy.[60] However, no changes in choroidal thickness were observed in patients with nonproliferative diabetic retinopathy.[60] Another found choroidal thinning in all stages of the disease when compared with age-matched controls.[61] These findings underscore the potential for analysis of the choroid as a method of monitoring the severity and progression of retinopathy.

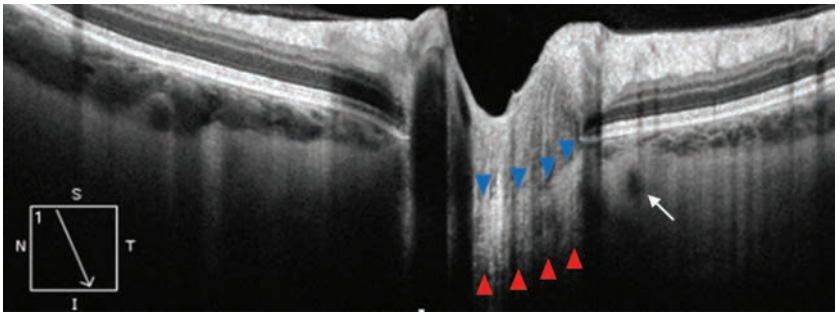
EDI-OCT for Glaucoma

With the advent of EDI-OCT, imaging of the peripapillary choroid as well as other deep papillary structures—such as the lamina cribrosa, short posterior ciliary arteries, central retinal artery, central retinal vein and the subarachnoid space around the optic nerve—is now possible.[62]

• ***Thinning of the lamina cribrosa.*** Recent evidence suggests that the primary site of ganglion cell axon injury is likely within the lamina cribrosa.[62-64] The biomechanical theory of glaucoma centers around the idea that an increase in IOP in the setting of reduced cerebrospinal fluid pressure causes posterior bowing and distortion of the lamina cribrosa, leading to crush damage to the axonal fascicles that pass through it.[63,64] EDI-OCT of the lamina cribrosa shows the lamina tends to be thinner in eyes with primary open angle glaucoma (POAG) compared with normal eyes.[65,66] As theorized, investigators have demonstrated the lamina is thinnest, approximately half that of normal eyes, in eyes with NTG.[65] Researchers suspect the unusually thin lamina cribrosa found in NTG eyes is highly susceptible to deformation, such that it occurs even in the presence of normal IOP values.[65] Combined with the possibility of poor vascular perfusion, this finding reinforces the biomechanical theory of glaucoma.

Several studies have shown a correlation between laminar thinning and the degree of structural and functional glaucomatous damage.[65,67] One found mean laminar thicknesses of 234µm in eyes with early stage glaucoma, 179µm in eyes with moderate stage glaucoma and 156µm in eyes with advanced stage glaucoma.[65] Additionally, the lamina was significantly thinner in NTG eyes with disc hemorrhage compared with those without.[65] The investigators hypothesized that a disc hemorrhage was likely a result of failure in capillary-containing laminar beams that led to rupture of prelaminar capillaries.[65] Research shows a significant correlation between decreasing laminar thickness and worsening visual field mean deviation.[65,67] This suggests the laminar thinning plays a role in progressive glaucomatous vision loss.[65,67]

Regional variation and changes in laminar pore size, as well as changes in pore shape, may be linked to glaucomatous progression too.[62,68] A recent study found variations in laminar pore shape and size as well as focal areas of partial laminar tissue loss in several glaucomatous eyes



EDI-OCT of deep papillary structures in a healthy eye. The blue arrows outline the anterior border of the hyper-reflective lamina cribrosa, while the red arrows outline the posterior border. The small hyporeflective circle adjacent to the laminar optic nerve represents the vascular lumen of the circle of Zinn-Haller (white arrow).

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when evaluated with EDI-OCT.[62] In eyes with localized nerve fiber layer loss, corresponding focal laminar defects were evident.[69]

Until further research is completed, it remains uncertain whether thinning and other structural changes of the lamina cribrosa are causes or consequences of glaucomatous optic nerve damage.[66]

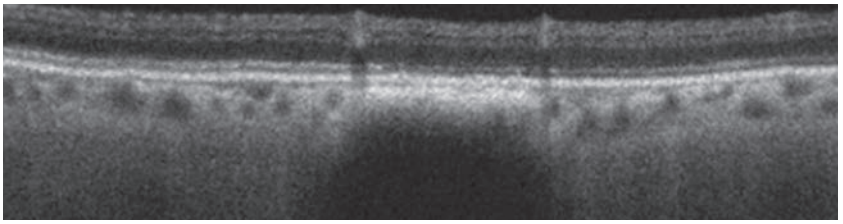
• ***Choroidal thickness in OAG and NTG.*** Evidence evaluating the choroidal thickness in eyes with glaucoma is conflicting. Some reports suggest choroidal thickness is less strongly correlated with the degree of glaucomatous damage compared with lamina cribrosa thickness. These reports, using EDI-OCT, show subfoveal and peripapillary choroidal thickness is not significantly different between eyes with OAG and OAG suspect eyes.[70,71]

To the contrary, choroidal thickness in the temporal peripapillary location (between the macula and nerve) is thinner in NTG patients than normal eyes and is correlated with the rate of visual field loss.[2] These findings can be interpreted to mean that hemodynamic alterations occur in the short posterior ciliary arterial supply to the posterior choroid along with the laminar/prelaminar optic nerve head.[2,72]

• ***Choroidal thickness in angle closure.*** A 2003 study proposed that choroidal expansion may contribute to the pathogenesis of primary angle closure.[74] The authors theorized an increase in choroidal volume would lead to an increase of posterior segment volume and pressure, inducing anterior displacement of the lens and shallowing of the anterior chamber.[74,75] Mechanisms that may induce choroidal expansion include:

1. An increase in episcleral and orbital venous pressure.

2. An increase in the osmotic pressure of the extravascular choroidal stroma due to leakage of large proteins that favor fluid movement into the extravascular space.[74]



Gray-scale EDI OCT of a small choroidal nevus with attenuation of the choriocapillaris and deep posterior choroidal shadowing. Note the preservation of the overlaying retina and absence of elevation or subretinal fluid.

The authors believe that primary angle closure may represent choroidal expansion in already smaller, anatomically predisposed eyes.[74,76]

EDI-OCT studies examining the choroid in patients with variants of primary angle closure support the hypothesis of choroidal expansion. A 2013 study evaluated subfoveal choroidal thickness in four patient subgroups of angle closure: primary angle-closure suspects, acute primary angle closure, primary angle closure and primary angle-closure glaucoma. Compared with the non-glaucomatous control group, the choroid was thicker in all subtypes of angle closure.[75] The mean subfoveal choroid was thickest in the acute primary angle-closure group—62µm thicker than control eyes.[75] A similar study found an increase in subfoveal choroidal thickness of approximately 60µm compared with controls in the fellow eyes of patients with a history of acute primary angle closure.[77]

A thick choroid may be another anatomical characteristic associated with primary angle closure that increases the risk of this entity, along with shallow anterior chamber depth, shorter axial length and increased lens thickness.[75]

Choroidal Tumors

• ***Choroidal nevus and choroidal melanoma.*** Choroidal melanoma is the most common primary adult ocular malignancy.[78] Risk factors for choroidal melanoma include orange pigment (lipofuscin), subretinal fluid, tumor thickness greater than 2mm via ultrasonography, symptoms of flashes, floaters or blurred vision and proximity to the optic nerve.[79, 80]

EDI-OCT allows for easy measurement of choroidal tumor thickness and detection of associated retinal edema and subretinal fluid.[79] Several authors have found that traditional ultrasonography tends to overestimate choroidal tumor thickness, sometimes more than two times greater than OCT measures.[81-83] In 2012, investigators compared EDI-OCT with ultrasonography in the evaluation of 51 cases of choroidal nevi. The authors found that EDI-OCT features of choroidal nevi included overlying choriocapillaris thinning, RPE atrophy and partial or complete shadowing posterior to the nevus.[81] Compared with ophthalmoscopy, the technique detected the presence of subretinal fluid in twice as many eyes.[81]

In another study comparing EDI-OCT imaging of small choroidal melanoma with similar sized choroidal nevus, several characteristics were more commonly associated with melanoma: increased tumor thickness, subretinal fluid, subretinal lipofuscin deposition, RPE atrophy, intraretinal edema, loss of the external limiting membrane and the inner segment/outer segment junction,

Advertorial

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BY BRAD OATNEY, O.D.

Our practice has seen a sharp increase in the number of contact lens wearers during the past year, opening the door to discuss the latest advances in technology with my patients. I've learned to have these conversations because my patients are incredibly motivated to learn about and try innovative products. I'm a firm believer that if you invest the time to educate lens wearers, they're much more likely to take action—such as being first to experience a new product, or to explore new options for contact lens wear.

While the lens material is critical to overall comfort, I try to educate patients about other possible factors affecting their overall lens-wearing experience—in particular, the role that their disinfecting solution can play. Current hydrogen peroxide-based contact lens disinfecting products contain surfactants that act not only as cleaning agents, but may also help retain moisture in contact lenses if they remain on the lens, keeping them hydrated longer.

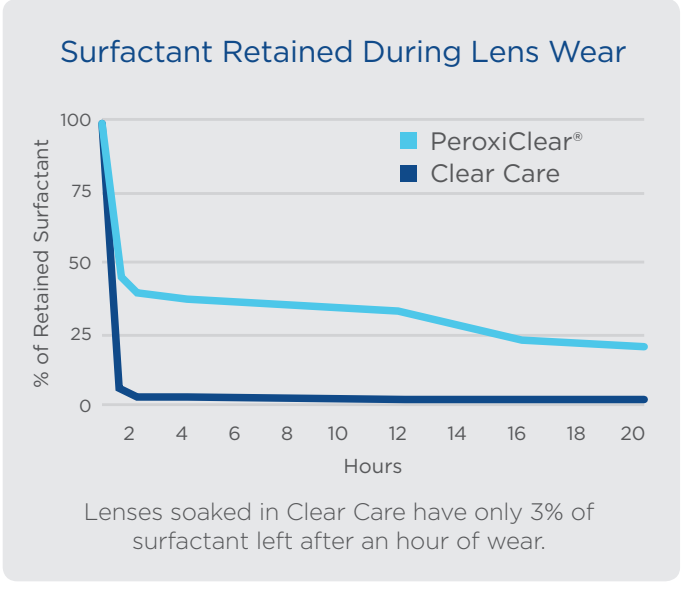
As new peroxide-based lens solutions have come to market, I've taken the time to educate myself on the benefits of these products. I've learned that among the new advances in hydrogen peroxide solutions, Bausch + Lomb PeroxiClear® solution is specifically designed to maintain moisture on the lens, and offer a more comfortable lens-wearing experience compared to other hydrogen peroxide systems.

PeroxiClear® utilizes Triple-Moist Technology®—a combination of three different moisturizing agents that work together to attract, spread and retain moisture on the lens surface throughout the day. One of these ingredients, carbamide, is a natural moisturizer that helps prevent dehydration. It also has a second unique role as a platinum-modulating compound (PMC) that allows for a faster overall disinfection rate, while still maintaining safe residual peroxide levels at the end of the neutralization and disinfecting cycle.[1] Additionally, studies have shown that the surfactant in PeroxiClear® remains on the lens to deliver moisture even after 20 hours of wear.[2]

The more I recommend PeroxiClear®, the more I observe its advantages over other hydrogen peroxide solutions. Most of my patients who have switched from Clear Care to PeroxiClear® say their lenses feel more comfortable immediately on insertion and stay moister and cleaner throughout the day. For patients who've been unsuccessful contact lens wearers because of discomfort or dryness, I've introduced PeroxiClear® with great success. Some of my patients who had sworn off contact lenses for years now tell me how exciting it is to wear lenses again.

When I educate contact lens wearers about the benefits of a new product, most are eager to try it—and they're certainly not shy about giving honest feedback. I was pleased that many patients who have switched to PeroxiClear® are reporting back a great lens-wearing experience. One such patient, whose lens dryness and discomfort was tied to her 14-hour workdays on a computer, followed my advice to switch to PeroxiClear®. When she came back for her annual exam a year later, she offered unsolicited, positive feedback that any eye care professional enjoys hearing: my recommendation transformed her contact lens-wearing experience. There is no better reward for an eye care professional than seeing a new product deliver benefits that result in a happier, more satisfied patient. ■

After neutralization, surfactant remains to deliver moisture for up to 20 hours of wear[2]



Lenses soaked in Clear Care have only 3% of surfactant left after an hour of wear.

[1] Millard KA, Groemminger SF, Hoteling A, Hook D, Wygladacz K. Surface characterization of the interaction between peroxide neutralizing disks and platinum modulating compounds. Scientific paper presented at: Association for Research in Vision and Ophthalmology (ARVO) 2014 Annual Meeting; 2014 May 4-8; Orlando, FL.[2] Hoteling A, Nichols W, Schafer J, Orsborn G. Retention of hydrogen peroxide solutions' surfactants on silicone hydrogel lenses. Poster session presented at: American Academy of Optometry (AAO)'s 92nd Annual Meeting; 2013 Oct 23-26; Seattle, WA.

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irregularity of the inner plexiform layer and the ganglion cell layer, photoreceptor loss, and "shaggy" swollen photoreceptors that appeared irregular and elongated.[83]

• ***Choroidal metastasis.*** Choroidal metastases are frequently a consequence of either lung or breast cancer.[79] Lesions commonly appear within the posterior pole and macular regions and may be multifocal or even bilateral as is common in cases of breast cancer.[79] Clinically, choroidal metastases appear amelanotic with indistinct margins.[79]

Researchers imaged 24 choroidal metastatic lesions with EDI-OCT and found that the majority demonstrated a plateau-shaped tumor with low internal reflectivity, overlying choriocapillaris thinning, shaggy photoreceptors, and subretinal fluid with high-reflective speckles.[82]

Additional associated features included RPE abnormality, photoreceptor abnormality, loss of the external limiting membrane or the inner segment/outer segment junction, and irregularity of the inner plexiform or ganglion cell layers.[82]

Five of the smaller tumors, which were undetected by ultrasonography, were identified and measured with EDI-OCT.[82]

Expanding Capabilities

Advancements in OCT technology, specifically the inclusion of enhanced depth imaging, allows for deeper evaluation of choroidal thickness and morphology. Emerging research suggests that choroidal dysfunction may play a role in the pathogenesis of many ocular diseases, even those initially thought to affect only the inner retina. The clinical uses of EDI-OCT include detection and monitoring of pathologic alterations in choroidal thickness, differentiation between diseases with similar clinical features, assessment of lamina cribrosa thickness in glaucoma patients, and precise measurement of choroidal tumor thickness. ■

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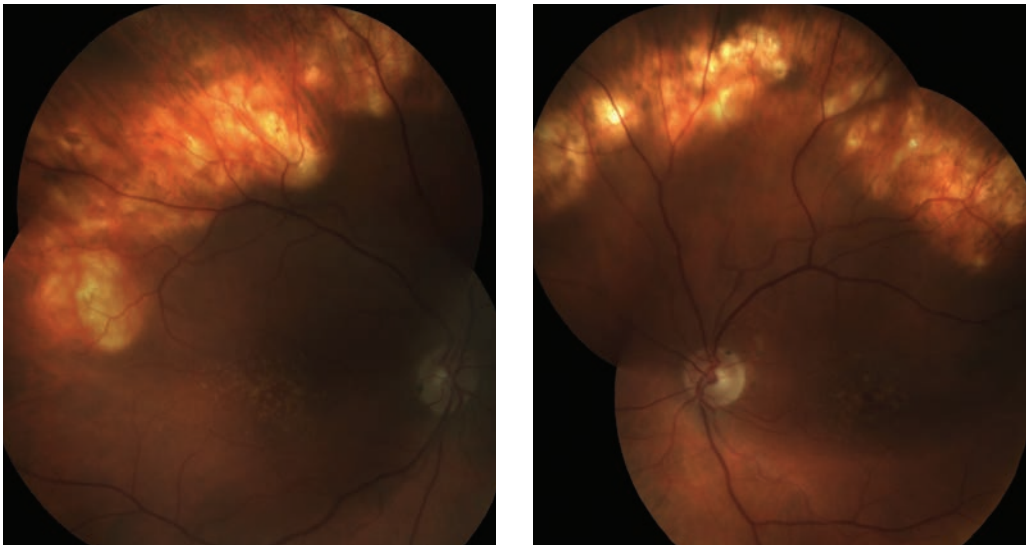
6th Annual Retina Report

'Doctor, I Have Mountains in My Eyes'

Suspicious retinal lesions indicate idiopathic sclerochoroidal calcification in an elderly patient. **By Sarah V. Cordes, OD, and Trina C. Perkins, OD**

Idiopathic sclerochoroidal calcification is a rare, benign condition generally affecting older to middle-aged whites.[1-7] This condition is characterized by calcific, yellow-white elevated lesions commonly found in the superior temporal fundus. Although these benign lesions are commonly outside the foveal zone and usually have no impact on the patient's vision, it's important to differentiate them from presentations with a similar appearance, such as choroidal neoplasms, inflammatory lesions and lesions associated with systemic conditions.[1-4,6-8]



This patient had significantly elevated, denticulated, yellow, mid-peripheral lesions located just beyond the superior temporal arcade in both eyes.

This case report describes a patient who presented to the clinic with this unique and rare condition. It also includes an overview of idiopathic sclerochoroidal calcification and the process by which differential diagnoses and systemic associations are ruled out.

History

An 81-year-old white male presented for a second opinion of his



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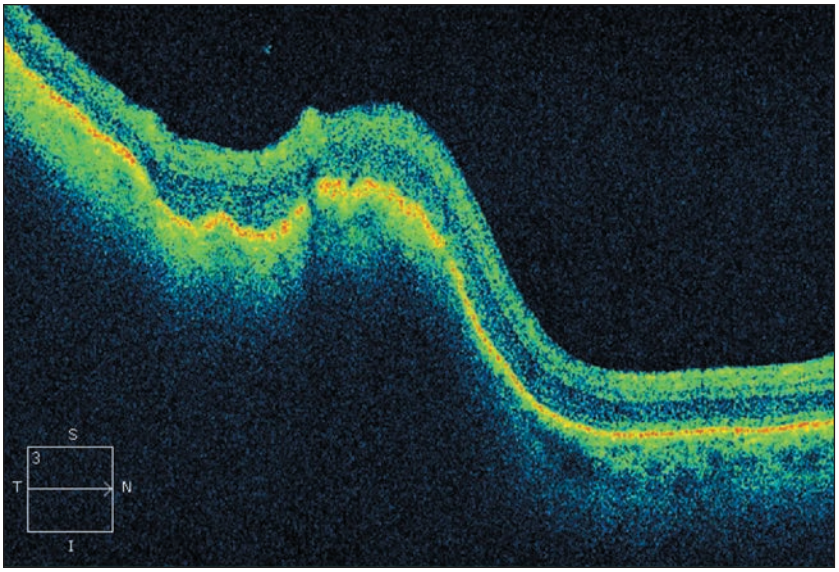

Ocular coherence tomography demonstrated the contour of the lesions seen upon initial ophthalmoscopy, and we understood why the patient described his condition as "mountains" in his eyes.

ocular condition. He reported that his local optometrist told him he has "mountains in his eyes." He complained of mild blur only at intermediate distances and occasional ocular itching.

His medical history was significant for Type 2 diabetes, hyperlipidemia, hypertension and aortocoronary bypass surgery due to coronary artery disease. His medications included hydrochlorothiazide, metformin, nitroglycerin, losartan and simvastatin.

Diagnostic Data

The patient's best-corrected visual acuity was $20/20^{-1}$ OD and 20/20 OS with compound hyperopic astigmatic correction.

Intraocular pressures measured 15mm Hg OU. Anterior segment examination was unremarkable. The patient's posterior chamber intraocular lens implants were centered bilaterally, without posterior capsule opacification.

Dilated fundus examination revealed multiple hard and soft confluent drusen of the macula consistent with non-exudative age-related macular degeneration. Peripheral ophthalmoscopy revealed significantly elevated, denticulated, yellow, mid-peripheral lesions at the level of the retinal pigment epithelium (RPE), located just beyond the superior temporal arcade in both eyes. Fundus photos and ocular coherence tomography, obtained at this initial visit, demonstrated the contour of these lesions. In addition, B-scan ultrasonography showed highly reflective choroidal elevations ranging in thickness from 1mm to 3.5mm with acoustic shadowing. There was no intra-retinal fluid, and the RPE and neurosensory retina were intact.

After reviewing these images, we understood why these elevated ridges were described to the patient as "mountains in his eyes."

Diagnosis

The bilateral, symmetric, superior temporal location of these lesions, and their elevated calcific appearance without inflammation, helped to differentiate this condition from choroiditis and choroidopathies. The highly reflective choroidal elevations with echogenicity and acoustic shadowing, along with the lack of intra-retinal fluid on B-scan ultrasonography, led to the provisional diagnosis of sclerochoroidal calcification.

However, an evaluation by a retinal specialist and fluorescein angiography were further indicated to help rule out possible intraocular tumors. Lab testing was also needed to rule out systemic etiology.

Treatment and Follow-Up

We referred the patient to a local retinal specialist for further evaluation and fluorescein angiography. Fluorescein angiography showed early phase hypofluorescence of the lesion, with late stage hyperfluorescence without leakage at any stage. No secondary circulatory patterns were present, which helped to rule out a possible intraocular neoplasm. The retinal specialist confirmed our diagnosis of sclerochoroidal calcification.

Following this, we ordered lab testing to screen for systemic associations. Tests included complete blood count, erythrocyte sedimentation rate and purified protein derivative (PPD). Serological evaluations for human immunodeficiency virus and syphilis were ordered to differentiate infectious etiologies. Blood and urine testing for calcium, phosphorus, potassium and magnesium were completed to rule out metabolic disorders. Serum testing for parathyroid hormone and calcitonin levels were also performed.

All testing was negative, further defining the diagnosis as idiopathic sclerochoroidal calcification.

No treatment was indicated for this patient, as no metabolic dis-

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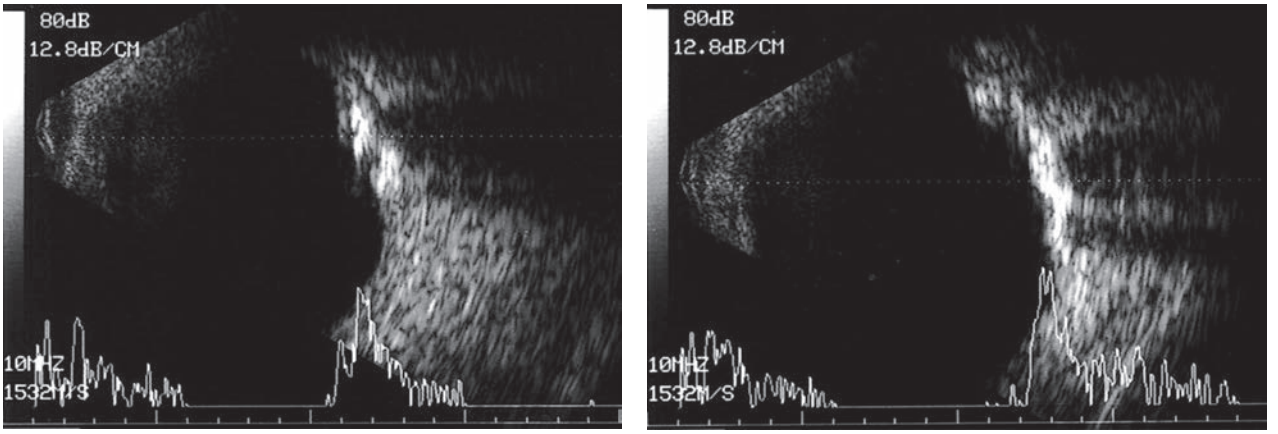

B-scan ultrasonography shows highly reflective choroidal elevations ranging in thickness from 1mm to 3.5mm with acoustic shadowing. There was no intra-retinal fluid, and the RPE and neurosensory retina were intact. These findings led us to a diagnosis of sclerochoroidal calcification.

order was present. Follow-up was scheduled in six months to monitor for unlikely choroidal neovascular membrane formation and retinal detachment. The patient was educated on the condition and given an Amsler grid for home monitoring.

Discussion

The term *idiopathic sclerochoroidal calcification* was first coined in the literature in 1989.[8] Limited data exists on the prevalence of this condition. A recent retrospective study led by Carol L. Shields, MD, at Wills Eye Hospital found reports of only 179 eyes affected with sclerochoroidal calcification between 1983 and 2014.[1] Most reported cases (79%) have been deemed idiopathic, and most occur in older whites.[1-7]

The lesions caused by sclerochoroidal calcification are often geographic and multifocal but can also be small, round and isolated.[1,4] They are typically raised or placoid with no overlying retinal involvement. Occasionally they are surrounded by choroidal atrophy. They are easily identified with ultrasonography or computerized tomography. On ultrasound, they are highly reflective with acoustic shadowing posterior to the lesion.[1-4,6-8]

Fluorescein angiography can also be helpful in making a diagnosis. The typical fluorescein pattern has early phase hypofluorescence followed by gradual late phase hyperfluorescence without leakage.[2,7,10] Due to disruption of the choroid and RPE, choroidal neovascularization and serous retinal detachment have been associated with this condition.[2,3,5,6,9-15] Biannual retinal evaluation is recommended to monitor for these rare complications, and patients should be advised to self-monitor vision with a home Amsler grid.[3,4]

Sclerochoroidal calcification can be further classified as metastatic or dystrophic. These terms are used to describe pathological calcification of body tissues.[4,16] Metastatic calcifications are lesions that form secondary to irregular calcium and phosphorous metabolism.[2-4,16-18] Dystrophic lesions are found in patients with normal serum calcium and phosphorus levels and are typically found in areas of organ tissue damage or necrosis.

Idiopathic sclerochoroidal calcification occurs in patients with normal calcium and phosphorus metabolism and is therefore termed a dystrophic calcification.[6] This calcification is pathologically identical to the anterior calcification at the insertion of the horizontal rectus muscles known as senile scleral plaques, seen in older individuals. Researchers hypothesize that idiopathic sclerochoroidal calcification has a similar mechanism, and is secondary to chronic movement of the oblique muscles at their corresponding insertion sites.[2,4-6]

Other causes of dystrophic sclerochoroidal calcification include chronic inflammation, trauma and senile degeneration of the sclera.[4,6,18,19]

Although the majority of sclerochoroidal calcifications are considered idiopathic, rare systemic associations do occur.[1-7] These calcifications have been attributed to hyperparathyroidism, parathyroid adenoma, vitamin D toxicity, pseudohypoparathyroidism, pseudogout, Bartter syndrome and Gitelman syndrome.[1,2,4,6,11,14,20,21] Initial laboratory testing to rule out these disorders may include serum and urine magnesium, potassium, calcium, alkaline phosphatase and phosphorus levels. Parathyroid hormone and calcitonin

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levels are also indicated.[1] In the case of abnormal test results, an endocrinology referral may be indicated.[22,23]

Differential Diagnosis

The differential diagnosis of sclerochoroidal calcification includes:

• ***Metastatic carcinoma*** must be ruled out, especially in patients with prior history of cancer. These lesions tend to be dome-shaped, creamy yellow, multifocal and are usually found inside the vascular arcades. They often have overlying serous retinal detachments. On diagnostic ultrasound, these lesions do not show signs of calcification, which include high reflectivity and significant shadowing beyond the surface of the lesion.[2-4,6,7,9,24]

• ***Choroidal melanomas*** and nevi are typically pigmented, unlike sclerochoroidal calcifications that are lacking in pigment. Although a melanoma may be amelanotic, it does not typically have overlying RPE dropout. Lack of calcification on ultrasonography is again the major diagnostic difference of these lesions. Melanomas also have internal vascularity visible on fluorescein angiography. Overlying retinal detachments are common in melanomas.[2-4,6,7,9,24]

• ***Choroidal osteoma*** is a common misdiagnosis of sclerochoroidal calcification. These orange-yellow placoid masses are found in younger adults and children. They are well defined and often located adjacent to the optic disc. The most notable differential of these lesions is that they are unilateral 80% of the time.[2-4,6,7,9,24-28]

• ***Intraocular lymphoma*** can present as raised multifocal or diffuse yellow lesions at the level of the RPE and therefore are sometimes confused with sclerochoroidal calcification. These patients typically have some form of systemic lymphoma and usually have an accompanying vitritis.[3,4,6,7,24, 26,27,29,30]

Other differentials of sclerochoroidal calcification may include inactive chorioretinitis, retinoblastoma, retinal astrocytic hamartoma, choroidal hemangioma and eccentric choroidal neovascular membrane. Patient demographics, the calcified nature of the lesion, ultrasound characteristics, lack of leakage on fluorescein angiography and typical location are all helpful in ruling out other diagnoses.[2,4,6,7,9,24]

Idiopathic sclerochoroidal calcifications are benign, but must be distinguished from more serious vision- or life-threatening choroidal neoplasms and inflammatory conditions. Detailed fundus examination, B-scan ultrasonography, fluorescein angiography and serial photography are typically needed to differentiate these conditions. Systemic metabolic abnormalities should also be ruled out with laboratory testing.

Routine ocular follow-up is needed to watch for possible serous retinal detachments and choroidal neovascularization. The prognosis is good as these retinal lesions typically have few complications and minimal visual significance.[1,2,4] ■

Dr. Cordes and Dr. Perkins are attending optometrists at The Villages VA Outpatient Clinic, in The Villages, Fla., which holds academic affiliations with the Illinois College of Optometry, Indiana University, Nova Southeastern University and Western University of Health Sciences.

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6th Annual Retina Report

Know Your RETINAL Breaks, Tears and Holes

Learn to better recognize posterior segment damage and minimize patient risk.

By Marlon Demeritt, OD, and Diana Shechtman, OD

Retinal tears, breaks and holes are commonly encountered in clinical practice. As they predispose a patient to a retinal detachment (RD) and optometrists are often the first clinician to examine such patients, prompt diagnosis and proper comanagement is a necessity.

This article will review the overall clinical picture of retinal holes, tears and breaks. It will discuss the classifications of the different types—specifically, horseshoe (flap) tears, operculated retinal breaks and atrophic holes—the propensity towards the development of RD, signs and symptoms and risk factors.

Posterior Vitreous Detachment Basics

Retinal tears, breaks and holes are often complications of posterior vitreous detachment (PVD). Understanding PVD, as well as recognizing its symptoms, can lead to early detection of retinal damage.

The vitreous humor is a transparent gel composed mostly of water. Other components include collagen fibrils and glycosaminoglycans (GAGs) or mucopolysaccharides (primarily hyaluronic acid). Collagen fibrils are structural proteins that add support and shape to the vitreous. They, along with extracellular matrix protein, help enhance vitreoretinal adhesion. In addition, hyaluronic acid provides stability to the collagen fibrils and is associated with the viscoelastic properties of the vitreous gel.

With age, the dynamics of collagen fibrils and hyaluronic acid break down, resulting in liquefaction, shrinkage and collapsing of the vitreous. Sequentially, small holes develop within the posterior vitreal cortex, causing liquefied vitreous to gain access to the subhyaloid space. This, along with a weakening of the vitreoretinal adhesion, leads to a separation of the vitreous from the retina's internal limiting membrane, resulting in PVD.

Release Date: June 2015
Expiration Date: June 1, 2018
Goal Statement: Prompt diagnosis and proper comanagement of retinal tears, breaks and holes is a necessity. This article discusses the classifications of the different types of retinal tears, breaks and holes, the propensity towards the development of RD, signs and symptoms and risk factors.
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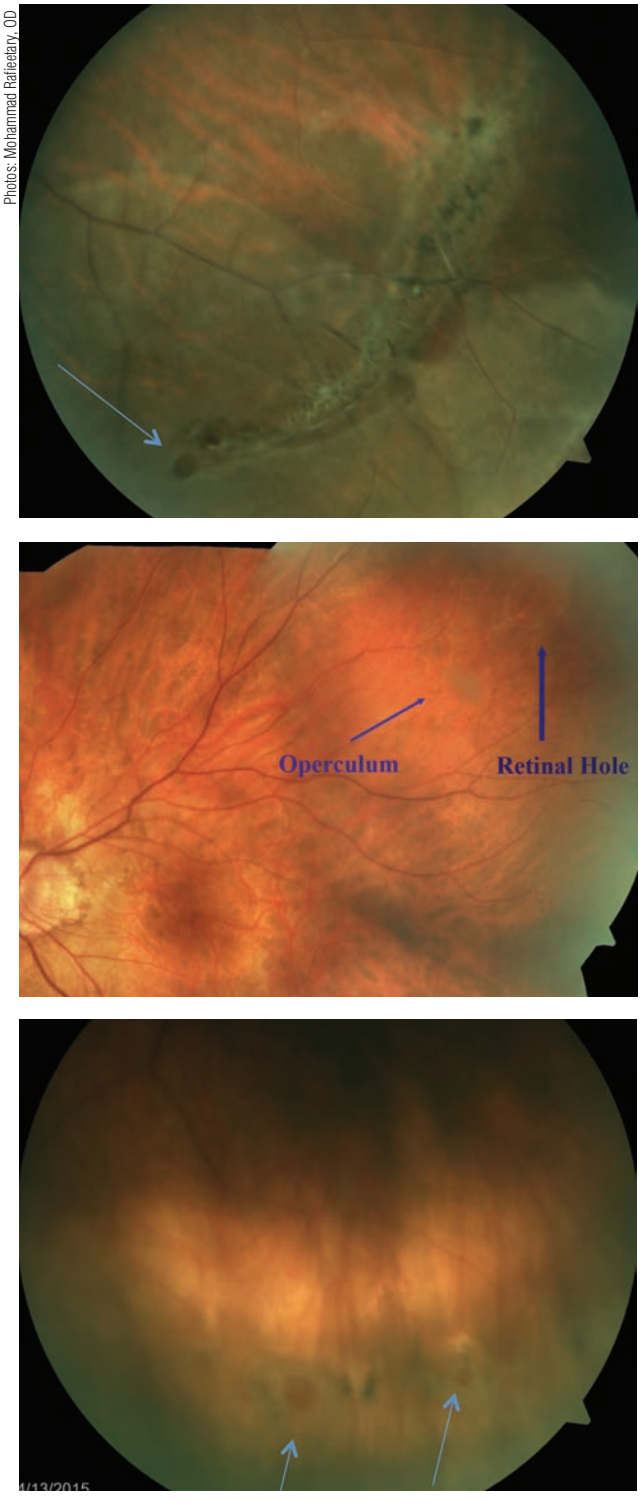


Tears are often found at the edge of lattice degeneration where the retina is thinner (top); the operculum appears as a whitish, disc-shaped floater over the retinal break (middle); atrophic holes are commonly found in the peripheral retina and within lattice retinal degeneration (bottom).

Sixty-five percent of people over the age of 65 will experience a PVD.[1,2] The condition typically occurs between the ages of 45 and 65 in the general population; however, the posterior vitreous may detach earlier in patients with retinal vascular diseases, trauma, aphakia, inflammation, vitreous hemorrhage or myopia.[3-7] A characteristic PVD is described as a ring shape overlying the optic nerve, referred to as a Weiss ring.

Up to 26% of patients with an acute PVD will present with a concomitant retinal break at the time of the initial presentation.[8-12] The chances of developing a retinal break following the initial presentation of an acute PVD is 2% to 5%.[10,13,14]

PVD Symptoms

Symptoms associated with a posterior vitreous detachment include flashes or floaters, or both. Patients typically report the characteristic flashes (photopsias) as an arc-shaped light perceived in their peripheral vision, which is most noticeable in the dark. Such photopsias are likely the result of vitreous traction on the peripheral retina, stimulating the underlying photoreceptors. In addition, it is also possible that the typically encountered arc-shaped flash is due to strong adhesion of the vitreous to the margin of the optic nerve head. As traction develops on this adhesion site, all the fibers that enter the optic nerve in that area become mechanically stimulated, resulting in an arc-shaped flash.

Flashes seem to represent a more ominous symptom than floaters. Though floaters are commonly associated with PVDs, they may be linked to other vitreal abnormalities such as blood or condensation of vitreous collagen. Floaters simply represent the casting of shadows onto the underlying retina.

Anomalous PVD

Complications of PVD are more likely to occur in eyes

Table 1. Complications of Age-Related Posterior Vitreous Detachment[16]

MACULAR CONDITIONS	PERIPHERAL CONDITIONS
Epiretinal membrane	Rhegmatogenous retinal detachment
Macular holes	Operculated retinal break
Myopic tractional maculopathy	Vitreous hemorrhage
Vitreopapillary traction	Retinal or optic disc hemorrhage
VMA/VMT	Flap tear

Table 2. Associated Findings that Increase Risk of Retinal Detachment

Fluid cuff or subclinical retinal detachment
Persistent vitreoretinal traction
Lattice degeneration with flap tear at lateral or posterior edge of lattice
Persistent symptoms
Posterior capsular rupture
Vitreoretinal degeneration

when accelerated vitreous liquefaction occurs prior to the weakening of the vitreoretinal adhesion, a phenomenon known as an anomalous PVD. This entity leads to traction at the vitreoretinal interface due to incomplete separation. Predisposed areas of strong vitreoretinal adhesions include the vitreous base, edge of lattice degeneration, blood vessels, optic nerve, macula and chorioretinal scars. Anomalous PVD can lead to numerous complications affecting the peripheral retina and the posterior pole (*see Table 1*).[15]

Peripheral Retinal Breaks

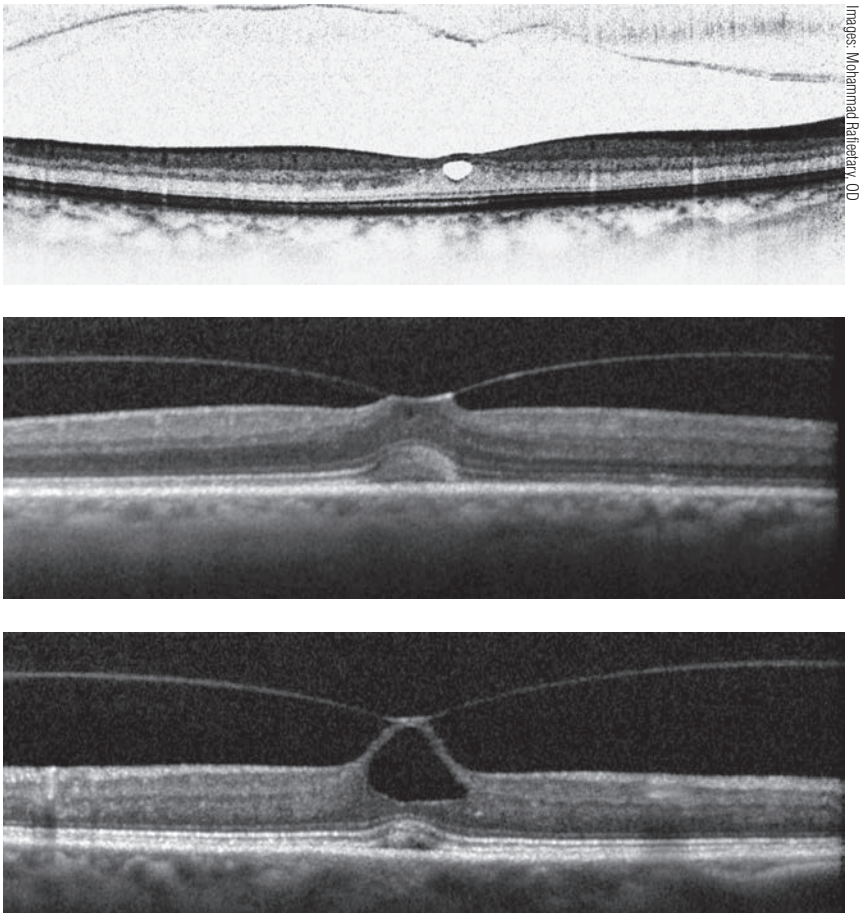
There are three common classifications of retinal breaks to look out for when evaluating patients:

An ***operculated retinal tear*** occurs following significant vitreous traction in a small, discrete area of the retina, resulting in increased vitreoretinal adhesion with an avulsed piece of retinal tissue (an operculum) overlying a small hole. Operculated holes are described as a plug of sensory retina overlying a round retinal break. It is often asymptomatic and results from a PVD pulling onto a vitreoretinal tuft.[17]

The operculum, which is seen as a whitish, disc-shaped floater over the retinal break, moves with eye movements due to its attachment to the posterior vitreous cortex. The operculum often shrinks with time, becoming smaller than the overlying break, indicating chronicity.[17] As a result, larger opercula are likely more acute in nature.

Operculated tears are usually round and appear redder than the surrounding retina since they are a full-thickness retinal defect. Although they can occur in any region of the retina, they are generally located between the ora serrata and the equator.[17] Operculated breaks usually do not progress to rhegmatogenous retinal detachment (RRD) because of release of the vitreous traction to the involved area during the formation of the operculum.[17] Traction on an incomplete operculated hole may increase the propensity to develop a retinal detachment. This presentation is often associated with symptomatic operculated breaks.[18,19] Thus, determining the presence of traction or symptoms of flashes and floaters in such cases becomes important in guiding treatment.

When significant localized vitreous traction exists, a ***horseshoe (flap) tear*** can develop. This is also known as a U-shaped tear or incomplete break. Horseshoe tears occur



Images: Mohammad Rafieetary, OD

OCT shows a complete PVD overlying a small macular defect (top); examples of anomalous PVD, represented by vitreomacular traction (middle and bottom).

as a result of incomplete vitreous traction pulling a horseshoe-shaped thin curvilinear flap of sensory retina towards the vitreous cavity.

The apex of the flap tear usually points toward the posterior pole, while the base points to the anterior retina. Although flap tears can exist in any region of the peripheral retina, they are most often found near the posterior margin of the vitreous base and at the edge of lattice degeneration, because the retina is thinner in the periphery and in areas with lattice degeneration.[17] Although lattice retinal degeneration has a very low risk of developing into RRD, patients with lattice and tractional-related flap tears associated with PVD have significantly increased risk of developing a rhegmatogenous retinal detachment.[20,21]

Flap tears are the most ominous type of tears due to their persistent traction on the retina. Approximately 50% of those with symptomatic flap tears will develop an RRD.[20,22,23] If retinal detachment is to follow, it usually does so within a few weeks after tear formation.[18] Horseshoe tears discovered in asymptomatic fellow eyes are less likely than symptomatic ones to develop an RRD. Thus, symptoms may be an important element in determining treatment of such tears. Patients presenting with acute retinal breaks or symptomatic retinal breaks should be referred to a retinal specialists for evaluation and management.

By far the most common presentation of retinal tears is ***atrophic holes***. Although they are not a result of tractional forces, they can present as a risk factor for developing a rhegmatogenous retinal detachment. An atrophic retinal hole is described as a full-thickness retinal break, commonly round-shaped with or without surrounding pigment. Associated pigment is a sign of chronicity and carries a very low

Case Study: Horseshoe Tear Follows Acute PVD

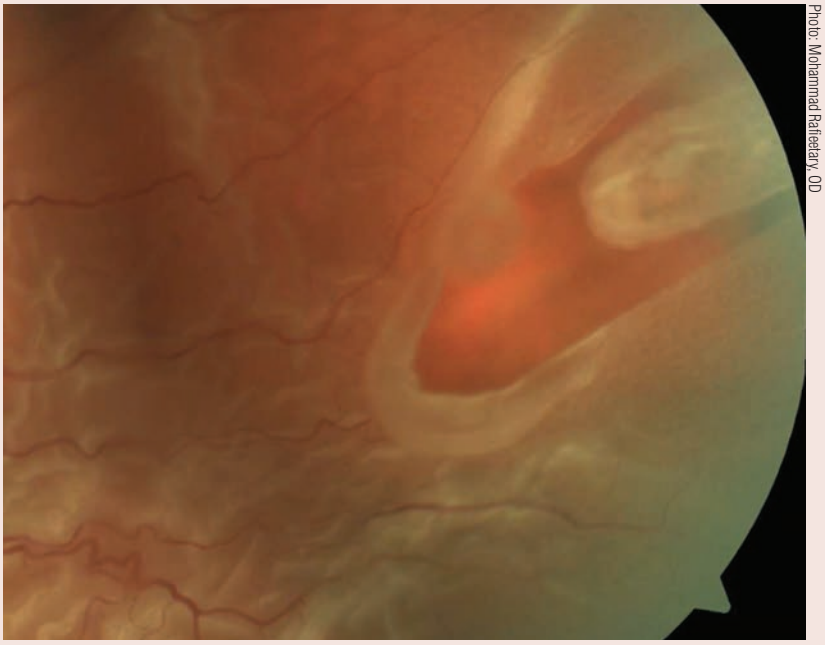


Photo: Mohammad Rafieetary, OD

Because of their persistent traction on the retina, horseshoe tears (such as this one in a similar patient) are the most ominous type of tears. Approximately 50% of those with symptomatic flap tears will develop an RRD, as seen here.[20,22,23]

A 60-year-old white male presented with two-day onset of complaints of flashes and floaters affecting the right eye. He reported that the symptoms occurred suddenly. His ocular history was remarkable for trauma in the left eye many years ago, but his medical history was unremarkable. Upon examination, his uncorrected visual acuity measured 20/50 (improving with pinhole to 20/30) OD and 20/25 OS. All preliminaries, including confrontation visual fields, extraocular motilities and pupillary testing, were unremarkable. Examination of his anterior segment was also unremarkable in both eyes. Both eyes' IOP measured 12mm Hg.

A dilated fundus exam of the vitreous revealed only a posterior vitreous detachment in the right eye, in addition to trace syneresis in the left eye. We saw no evidence of hemorrhage or pigmented cells in the vitreous of either eye. Optic disc cupping was 0.3/0.3 with pink, distinct neuroretinal rims in both eyes. Evaluation of both maculae revealed normal uniform color with no associated thickness or abnormalities. The peripheral retina was evaluated with scleral depression. No holes, tears or detachments were visible in either eye. We made the diagnosis of acute PVD in the right eye, advised the patient of the findings and educated him on new signs and symptoms that might occur. If they did, the patient was asked to return to the clinic immediately; otherwise, we recommended the patient return for a follow up in a week.

The patient returned in a week as advised. His case history revealed stability of the flashes and floaters with no changes in his signs or symptoms. His uncorrected visual acuities and preliminary testing remained unchanged from the previous visit. His anterior segment was unremarkable. IOP was 14mm Hg OD and 15mm Hg OS. Dilated fundus exam revealed vitreal pigmented cells and a Weiss ring in the right eye.

Trace vitreal syneresis was noted in both eyes. Peripheral findings of the left remained unremarkable, however a shallow rhegmatogenous retinal detachment associated horseshoe tear was observed in the temporal retina of his right eye with no macular involvement noted.

We immediately referred this patient to a retina specialist for treatment.

Table 3. Other Patient Factors that Increase of Retinal Detachment

Retinal detachment in the fellow eye
Trauma
Inflammation
History of ocular surgery, including YAG capsulotomy
Family history of retinal detachment
Occupation (e.g., contact sports)
Myopia

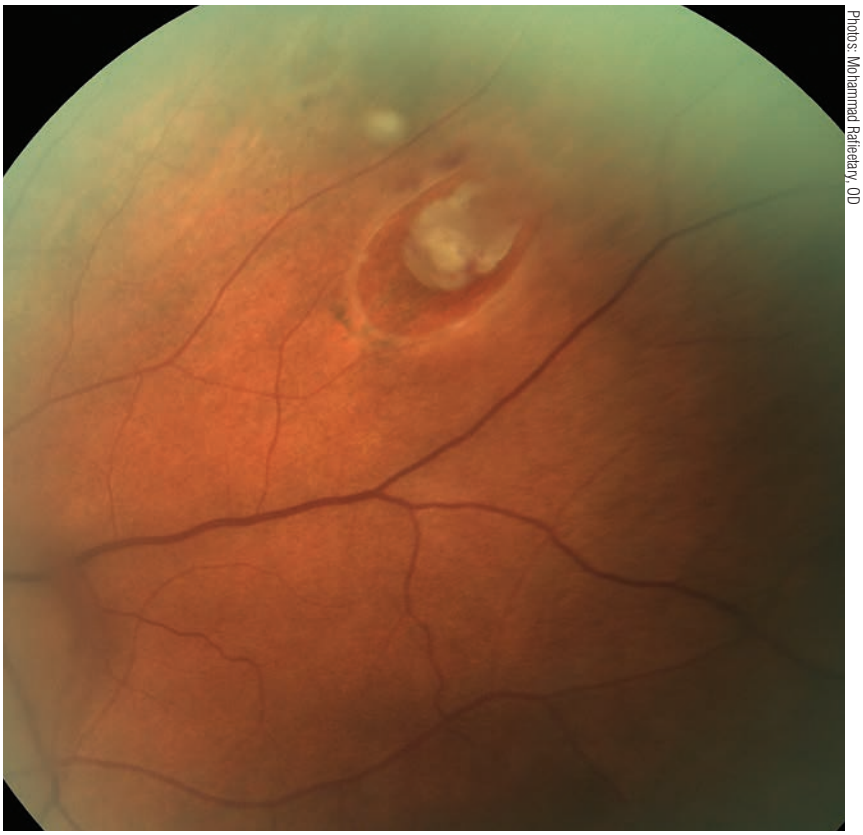
risk of developing an RRD. Atrophic holes are a result of an atrophic process associated with a thinning retina or vascular insufficiency. They are more commonly found in the peripheral retina and within lattice retinal degeneration. Holes within lattice retinal degeneration may increase the propensity of progressing towards an RRD, although the risk is still quite small. The sizes of these holes vary from pinpoint to greater than a disc diameter.[24]

While the pathogenesis of atrophic holes isn't related to vitreoretinal traction, vitreous fluid could potentially percolate underneath the hole, resulting more likely in a subclinical RRD. A subclinical RRD is defined as a >1DD extension of fluid surrounding the hole (larger than a cuff of fluid) but does not extend >2DD into the posterior pole. About one-third of subclinical RRD will progress towards a clinical RRD.[25]

Evaluation

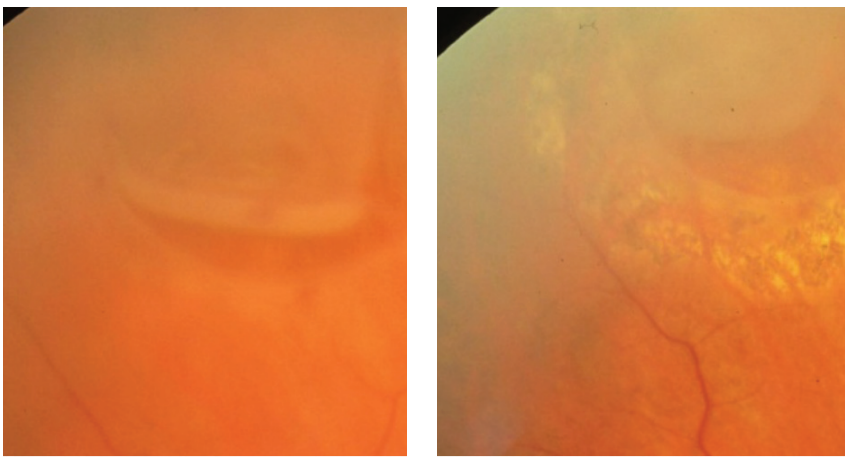
The initial evaluation of a patient with an acute PVD includes a careful case history. Since symptoms may carry a greater propensity towards the development of an RRD, determining associated symptoms related to flashes and floaters is critical.

Because PVDs associated with flashers carry a higher risk of developing a concomitant retinal break, it is prudent to reevaluate any increase in signs or symptoms during the follow up of a PVD.[26]



Photos: Mohammad Rafieetary, OD

Patient with recent onset PVD with secondary vitreous hemorrhage and horseshoe tear seen clearly, as well as operculated retinal hole seen in the upper left corner of the image.



The apex of a horsehoe tear usually points toward the posterior pole and the base points to the anterior retina (left). Patients with horseshoe tears associated with PVD have significantly increased risk of developing an RRD unless treated by laser prophylaxis, as seen here (right).

During the course of the examination, pay careful attention to the vitreous. Evaluate not only for the

Table 4. Management Options for Retinal Breaks[26]

Type of Lesion	Management Options
Acute symptomatic horseshoe tears	Treat promptly
Acute symptomatic operculated tears	Treatment may not be necessary
Acute symptomatic dialyses	Treat promptly
Traumatic retinal breaks	Usually treated
Asymptomatic horseshoe tears (w/o subclinical RD)	Often can be followed without treatment
Asymptomatic operculated tears	Treatment is rarely recommended
Asymptomatic atrophic round holes	Treatment is rarely recommended
Asymptomatic lattice degeneration without holes	Not treated unless PVD causes a horseshoe tear
Asymptomatic lattice degeneration with holes	Usually does not require treatment
Asymptomatic dialyses	No consensus on treatment and insufficient evidence to guide management
Eyes with atrophic holes, lattice degeneration, or symptomatic horseshoe tears where the fellow eye has had a RD	No consensus on treatment and insufficient evidence to guide management

presence of PVD, but any hemorrhages or pigmented cells within the vitreous. Fifty percent to 70% of vitreous hemorrhages associated with a PVD are also associated with a concomitant retinal break. Retinal breaks or RRD are often associated with pigmented cells in the vitreous. These cells represent pigment liberated from the underlying RPE due to tractional forces.

Since retinal breaks are often found in the peripheral retina, evaluation of the peripheral retina should include careful dilated assessment with indirect ophthalmoscopy. Scleral depression and 3-mirror contact fundus lens can also be helpful in the evaluation of the peripheral retina.

Various auxiliary tests may be useful when evaluating a patient presenting with an acute PVD.

In addition, OCT can be used to aid the clinician in evaluating the stage of the PVD, as well as any associated maculopathy. While a complete PVD may be noted using DFE or OCT, it does not preclude the possibility that associated peripheral vitreoretinal disease may coexist. B-scan ultrasonography has also been found valuable when attempting to assess a fundus that is obscured due to media opacities or vitreous hemorrhage.

Management

Referral for treatment of a retinal break varies based on type of break, associated findings (*see Table 2*), symptomology and risk factors (*see Table 3*). Because most atrophic and operculated holes do not have a high propensity towards progression to an RRD, most are followed. On the other hand, if patients display increased risk factors, such as a pseudophakic patient with a posterior capsular rupture, there may be a higher concern for the progression to an RRD. Since 30% of symptomatic retinal breaks progress towards developing an RRD, they should be referred to a retina subspecialist. Remember that symptomatic flap tears carry the highest risk of developing an RRD. See *Table 4* for a comprehensive review of the AAO guidelines for treating retinal breaks.

The goal of treating a retinal break is to create a firm chorioretinal adhesion on the attached retina adjacent to and surrounding the retinal tear. To achieve this, the surgeon uses cryotherapy or argon laser photocoagulation to halt the progression of subretinal fluid from detaching uninvolved neurosensory retina.[25]

Cryotherapy is a method of freezing the retinal break area with the application of a cryo probe to the retinal breaks. The areas of concern are frozen until they are sealed by a chorioretinal adhesion.

Argon laser photocoagulation is most commonly employed and is considered a safe and effective treatment for symptomatic flap tears and other high-risk retinal breaks. Laser burns are applied in several rows around the retinal break to help achieve chorioretinal adhesion.

Regarding time for chorioretinal adhesion, cryotherapy produces a protective barrier in seven to 10 days, and laser photocoagulation achieves its barrier in two to three days. However, cryotherapy is often reserved for cases that cannot be treated with laser. Such cases may include those with difficulty viewing the retina due to opaque media or small pupils. Additionally, using argon laser photocoagulation may be limited by the size of the retinal break and the peripheral location.[27]

Treatment of peripheral horseshoe tears should be extended to the ora serrata. If the tear is not adequately treated, continued vitreous traction may extend the tear beyond the treated area and allow fluid to dissect through the subretinal space, causing a clinical RD.[28-30]

When reevaluating a patient following treatment of the tear, it is

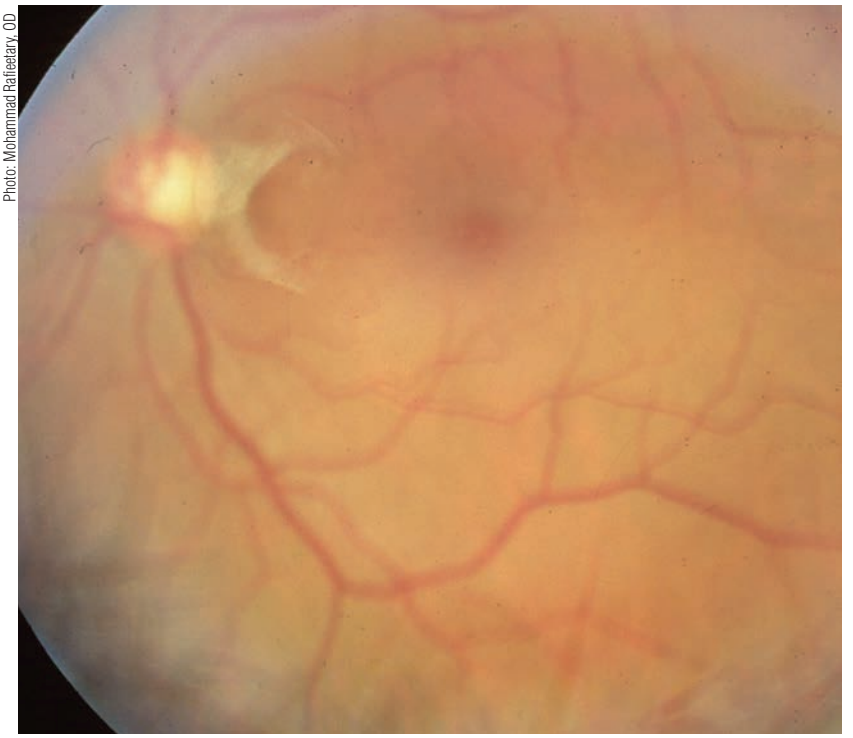


Photo: Mohammad Rafieetary, OD

A characteristic PVD such as this one is described as a ring shape overlying the optic nerve, referred to as a Weiss ring.

important to note the presence of a well-sealed barrier, in addition to the absence of new breaks anywhere in the retina. During follow-up visits, it is important for the clinician to evaluate for complications such as an epiretinal membrane (ERM) or macular pucker. Proliferation of the ERM or macular pucker has been observed following treatment for a retinal break. However, although the development of an ERM or macular pucker is possible, research has not shown a direct cause-and-effect relationship.[26] In one long-term follow-up study, the percentage of eyes that developed macular pucker after treatment of retinal breaks was no greater than that of eyes observed to have macular pucker before undergoing treatment.[28] ■

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Principles of PVD Management

As primary care providers, optometrists are usually the first to evaluate patients with acute posterior vitreous detachments, associated retinal breaks, or both. Since any acute PVD may lead to a rhegmatogenous retinal detachment or retinal break, all acute PVDs must be carefully evaluated and followed.

According to the American Academy of Ophthalmology guidelines published in 2014, follow up on an acute PVD should occur anywhere from one week to six weeks following the onset of symptoms, depending on risk factors, associated signs or symptoms, or both.[1] Similarly, according to guidelines published by the American Optometric Association, a patient with a symptomatic posterior vitreous detatchment should be followed at least every two to three weeks until the condition becomes asymptomatic and no concomitant retinal tears are found.[2] Patients with acute PVDs should be advised to return immediately if they experience an increase in signs or symptoms such as flashes, floaters or a curtain in their vision.

More than 80% of retinal detachments are associated with a retinal break that developed from a concomitant PVD.[3,4] Since 26% of patients with an acute posterior vitreous detachment will present with a concomitant retinal break at the time of the initial presentation, it may be best to follow all PVDs sooner rather than later.[5]

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1. Which one of the following is not a risk factor for early detachment of the posterior vitreous (PVD)?
a. Cataract surgery.
b. Posterior uveitis.
c. Myopia.
d. Choroidal melanoma.

2. Which of these is NOT considered a predisposed area of strong vitreoretinal adhesions?
a. Vitreous base.
b. Optic nerve.
c. Chorioretinal scar.
d. Retinal hemorrhage.

3. What percent of patients with an acute PVD will present with a concomitant retinal break at the initial presentation?
a. 15%.
b. 26%.
c. 2%.
d. 56%.

4. Which of the following is not a complication of an age-related posterior vitreous detachment?
a. Rhegmatogenous retinal detachment.
b. Operculated retinal break.
c. Flap tear.
d. Atrophic hole.

5. What are the chances of developing a retinal break after the initial presentation?
a. 10% to 20%.
b. 2% to 5%.
c. >50%.
d. About 15%.

6. Operculated breaks result from:
a. Vitreoretinal tuft.
b. Neovascularization.
c. An epiretinal membrane.
d. Diabetic retinopathy.

7. Horseshoe (flap) tears are the result of:
a. Vitreomacular traction.
b. Cystoid macular edema.
c. Incomplete vitreoretinal traction.
d. Vitreoretinal tuft.

8. Approximately what percent of symptomatic tears will progress to a rhegmatogenous retinal detachment?
a. 30%.
b. 10%.
c. 50%.
d. 75%.

9. What is the base of a horseshoe tear oriented towards?
a. Optic nerve.
b. Temporal arcade.
c. Posterior pole.
d. Anterior retina.

10. Atrophic holes result from:
a. Vitreoretinal traction.
b. Capillary perfusion.
c. Vascular insufficiency.
d. Complete operculated break.

11. Approximately what percent of subclinical RDs progress to rhegmatogeous retinal detachment?
a. 25%.
b. 33%.
c. 50%.
d. 75%.

12. As a result of retinal breaks, pigmented cells are liberated into the vitreous from what underlying structure?
a. Choroid.
b. Retinal pigment epithelium.
c. Photoreceptor integrity line.
d. Nerve fiber layer.

13. What percent of retinal detachments are associated with retinal breaks that developed from a concomitant posterior vitreous detachment?
a. 30%.
b. 65%.
c. More than 80%
d. 50%.

14. Which type of retinal break carries the highest risk towards developing a rhegmatogenous retinal detachment?
a. Atrophic hole.
b. Macular hole.
c. Symptomatic flap tear.
d. Lattice degeneration with atrophic hole.

15. In regards to treatment of a retinal break, chorioretinal adhesion to the

OSC QUIZ

attached retina is accomplished by which method?
a. Pars plana vitrectomy.
b. Cryotherapy.
c. Argon laser photocoagulation.
d. Both b and c.

16. What is the most common cause of failure when treating horseshoe tears?
a. Uncontrolled diabetes.
b. Choice of surgical method.
c. Failure to adequately treat the tear.
d. Uncontrolled hypertension.

17. In regards to retinal breaks, which of the following is not a risk factor for increasing the propensity of developing an RRD?
a. Persistent vitreoretinal traction.
b. Posterior capsule rupture during cataract surgery.
c. High myopia.
d. Astigmatism.

18. Which of the following is a risk factor for developing an RRD?
a. Trauma.
b. YAG capsulotomy.
c. Inflammation.
d. All of the above.

19. Traumatic retinal breaks:
a. Are usually treated.
b. Are not treated.
c. Have no sufficient evidence regarding treating.
d. Absolutely require no treatment.

20. Symptomatic flap tears:
a. Should be treated promptly.
b. Can usually be followed without treatment.
c. Have no sufficient evidence regarding treating.
d. Absolutely require no treatment.

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Limbal rings are subtle but powerful signals of youth and health.

Next, they tested the impact of the limbal ring on perceptions of attractiveness. Photos of male and female faces with neutral expressions were edited to produce a pair of otherwise identical images for each face – one with no limbal ring and the other with a dark limbal ring. Both male and female observers rated the faces with dark limbal rings as more attractive.

It turns out there may be an evolutionary reason for this. "Limbal rings are subtle but powerful signals of youth and health," says Dr. Peshek. The visual system is playing matchmaker for our genes, using information from the face and eyes to decide who you should get to know better. "We pay greater attention to the eyes than any other feature of the face. So if you want to do something to look more attractive," he says, "the eyes may be the best place to start."

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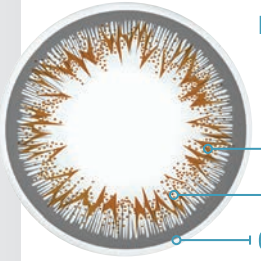

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¶ Some patients may notice a small difference in subjective vision performance, particularly in low light conditions.
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- Giving the lenses to staff members to wear is a smart strategy to inspire conversations about the lenses with patients
- To generate referrals, encourage patients to post a selfie to social media with #BeautyDEFINED and your practice name when they try the lens.

The Future of Retinal Imaging

Better technology improves care, but coding and reimbursement are slow to catch up.

By John Rumpakis, OD, MBA, Clinical Coding Editor

I am astounded by the new technologies that help us deliver higher levels of patient care, whether it's a retinal analyzer that allows me to look at numerous layers of the retina and choroid with a single scan, a fundus image where facial recognition software identifies typical retinal pathologies instead of faces, or the ability to have an autofluorescent image of the retina without using IV sodium fluorescein.

Future Reimbursement

These technological advancements have changed the way we describe the testing that is performed on a daily basis within ophthalmological and optometric practices, and in many respects the CPT coding definitions and rules can be somewhat behind the technology. We now have a single instrument that can provide a high-resolution fundus photograph (92250), an OCT of the optic nerve (92133) and retina (92134) and an autofluorescent image of the retina (92250).

It wouldn't surprise me if, in the future, all retinal imaging is grouped together and a new reimbursement paradigm is applied, allowing practitioners to perform any test at the frequency they believe would deliver the best outcome for the patient at a fixed cost per patient per year.

It's possible a carrier could propose a fixed global reimbursement per patient for all retinal images. It would most likely reduce the amount of testing performed—and it would certainly contain costs from a carrier's perspective.

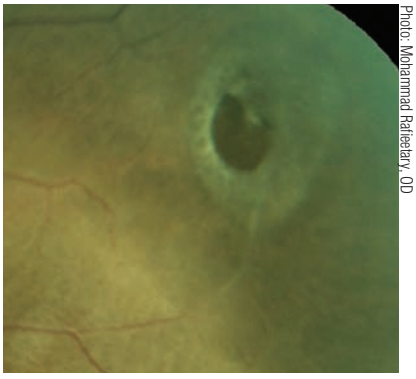

Photo: Mohammad Rafieetary, OD

Fundus imaging reveals a horseshoe tear.

Whether this would be a good policy or not is immaterial. The powers driving health care reform have a mandate to deliver high quality care in an efficient manner and at the lowest cost. Reducing the use of health care is also a mandate, and creating a coding and reimbursement paradigm that reflects the current technological advances and anticipates future ones is a priority of the American Medical Association and the Centers for Medicare & Medicaid Services.

Current Coding

So how does this affect us today? It is important to know the current definitions of the CPT for retinal imaging. In December 2014, the CPT refined its definition of autofluorescent imaging of the retina performed without sodium fluorescein. Prior to this ruling, CPT code 92499, "unlisted ophthalmic procedure," was the typical code used to describe autofluorescent imaging of the retina. But after the December ruling, the CPT now stipulates that the appropriate code is 92250, "fundus photography."

The implications of this decision affect your practice, considering that today, in many jurisdictions, a practitioner is allowed a limited number of fundus photographs (92250) in a given year on a per patient basis. So if you've already performed one and want to take another, you may not be allowed to and have a carrier pay for it. Special ophthalmic tests cannot be done prognostically—you must establish medical necessity prior to ordering the test. Yet, much of the technology on the market today allows practitioners to make better diagnoses earlier in the disease life cycle. It's a conundrum for sure, but one that a global annual allowance for retinal imaging would solve.

How we provide care and are reimbursed for it is under intense scrutiny with the mandate to do it better, faster and cheaper. Advancing technologies that offer multiple retinal imaging methods contained within a single instrument can certainly help us care for our patients better, faster and, in the future, most likely at a lower reimbursement. This same paradigm has been applied to almost every sector of the business world, and it's now being applied to health care. It's all about "big data" and data analysis, and we are about to get our numbers crunched. ■

Send questions and comments to ROcodingconnection@gmail.com.

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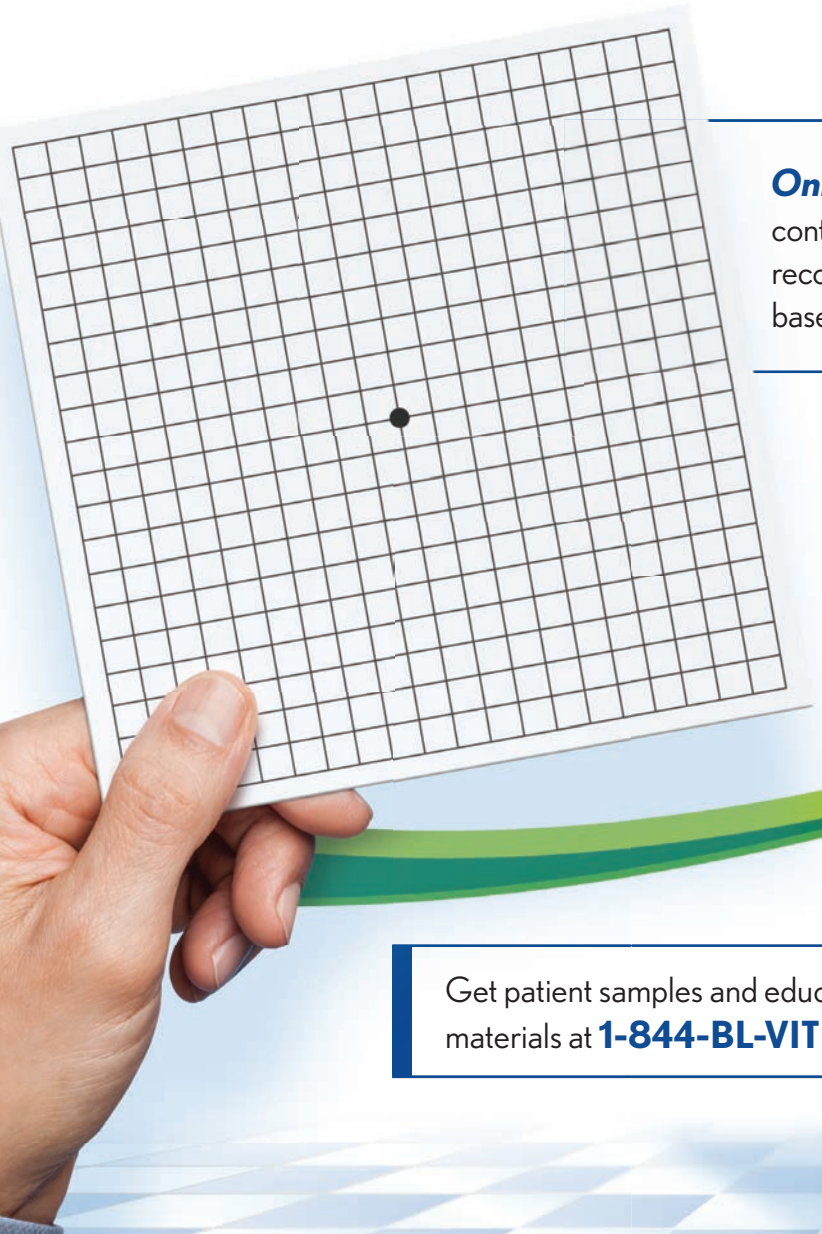

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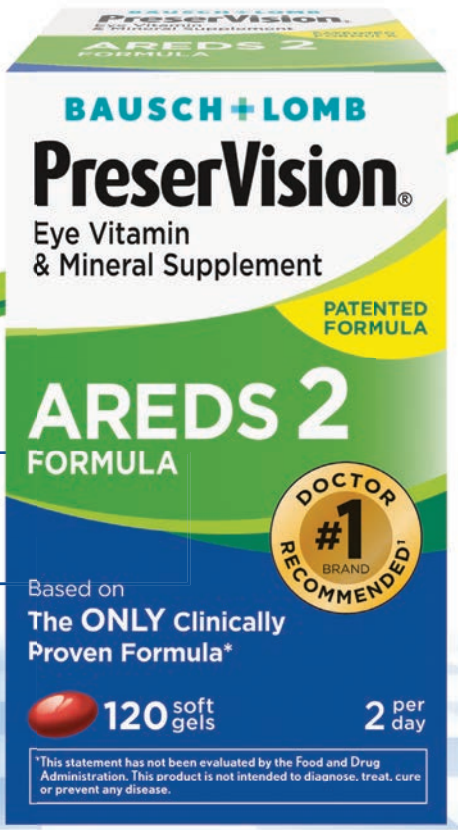

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Battle of the Bands

Knowing the possible reasons behind the resurgence of this common corneal disease is half the battle. **Edited by Joseph P. Shovlin, OD**

Q A recent 76-year-old patient of mine has developed a rapid recurrence of her band keratopathy following removal by chelation. What's the likely cause of her rapid accumulation of calcium or phosphorous?

A Band keratopathy is a common corneal disease characterized by the appearance of a band across the central cornea formed by the deposition of calcium from aqueous tears onto the subepithelial corneal surface. Patients with this condition may complain of reduced visual acuity (as the calcium deposition affects the visual axis), foreign body sensation and irritation.

A chelation is one method of treating band keratopathy; it involves "the scraping of the epithelium, administration of disodium EDTA and multiple cornea scrapings," says Eric Donnenfeld, MD, who performs refractive and cataract surgery on Long Island.

"The healing can be uncomfortable and visual rehabilitation prolonged. Promoting healing and reducing inflammation is imperative to reduce the risk of recurrent band keratopathy—once the epithelium is intact, the risk of recurrence decreases significantly."

Some patients are more at risk for recurrence, however, says Rishi K. Parikh, MD, an ophthalmologist in Atlanta. Patients with Paget's disease, sarcoidosis, hyperparathyroidism or kidney issues are more likely to have excess calcium in their systems, which can present in the tears and lead to deposition on the cornea. Calcium deposition can also occur as a result of low phosphorous levels in patients with renal failure, and as a result of pH changes in patients with uveitis, Dr. Parikh adds.

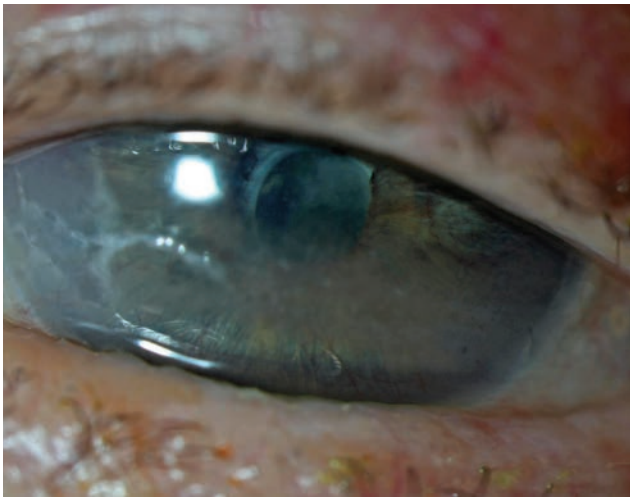

Photo: Aaron Bronner, OD

Band keratopathy as a result of calcium deposition.

Excess supplementation can also lead to calcium buildup, Dr. Donnenfeld notes, "so make certain the patient is not consuming an inordinate amount of antacids or other causes of milk alkali syndrome." Band keratopathy has also been linked to the presence of certain levels of drugs and supplements in the body other than calcium, such as excess vitamin D.[1] Patients who frequently use eye drops containing phosphate salts, especially phosphate topical steroids, are also more prone to band deposition.[2]

Two less common factors that may contribute to the development of band keratopathy are silicone oil and mercury, Dr. Parikh says. Silicone oil is used as a retinal tamponade during vitreous surgery for retinal detachments and is typically left in the eye for several months; this practice has been shown to correlate with band keratopathy.[3] Patients who have worked with mercury in the past may also be at greater risk for the corneal disease.[4] Pilocarpine contains trace levels of mercury, so consider its potential involvement in glaucoma patients on long-term therapy with cholinergic agents.

Regardless of the presentation's cause, however, when a chelation is required for the second time consider more aggressive healing measures. "I promote healing with an amniotic membrane, use copious corticosteroids and, if the stromal bed is irregular, will perform a lamellar keratectomy to smooth the corneal surface," Dr. Donnenfeld says. "In most cases, the second time is the charm." ■

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The Shoemaker's Plea

An acquaintance asks your opinion on a patient you've never seen. You're in a social setting, not in the office. How do you handle the situation? **By James L. Fanelli, OD**

While traveling recently in Italy, I received an email from an acquaintance who lives in Florence. I've known him for several years and have been a patron of his shoe store countless times. But we've never communicated by email, so I was curious why he was contacting me.

As you can see, it was an urgent issue for him and his family:

I am writing you on behalf of my nephew. He is 22 years old and in his last year of pharmacy school. His doctors recently told him he has problems with his eyes and probably has only a year left of sight. The doctors say he has a lot of pressure behind his eye and they can operate, but it's a temporary fix. My sister and I are both distraught and we are wondering if you would be able to just take a quick glance at it and see what your opinion is. I am so sorry to bother you but I don't entirely trust the doctors there. Below are his results from his tests.—Leonardo

Several documents were attached to this email, including a visual field report, optical coherence tomography, retinal nerve fiber layer scans and optic nerve images, along with concise data—including IOPs, medications and follow-up visits—from the nephew about his visits to a local ophthalmologist.

While the circumstances of this particular "social consult" are unique, we've all been asked for our opinion on a particular patient's glaucoma (or corneal, retinal or other) condition. How should we respond? Or, should we decline to respond? Let's look at this patient's problem as a case example of how to handle such encounters.

Diagnostic Data

Fortunately, the information from the nephew was substantial.

In February 2015, the patient, a 22-year-old white male, was seen by a local ophthalmologist and diagnosed with glaucoma. Initial IOPs measured 34mm Hg OD and 32mm Hg OS.

Looking at the optic nerve photos that accompanied the email, I estimated the cup-to-disc ratios to be approximately 0.90 x 0.90 OD and 0.80 x 0.80 OS.

OCTs of both the right and left retinal nerve fiber layer TSNIT scans showed some flattening of the superotemporal and inferotemporal nerve fiber layers. Macular scans demonstrated some thinning of the ganglion cell complex superiorly in the right eye, but were normal in the left eye. Threshold visual fields demonstrated only a slight enlargement of the blind spot OD and a normal visual field in the left eye.

There was no mention of related medical conditions, if any, or of the remainder of the ophthalmic exam. But I had enough information to see how and why the initial practitioner made the diagnosis of glaucoma.

Management

The patient was initially medicated with Xolamol (dorzolamide 2%/timolol 0.5%, Jamjoom Pharma) BID OU for two weeks. On return visit, IOPs were 20mm Hg OD and 18mm Hg OS. The provider then added Lumigan (bimatoprost, Allergan) HS OU.

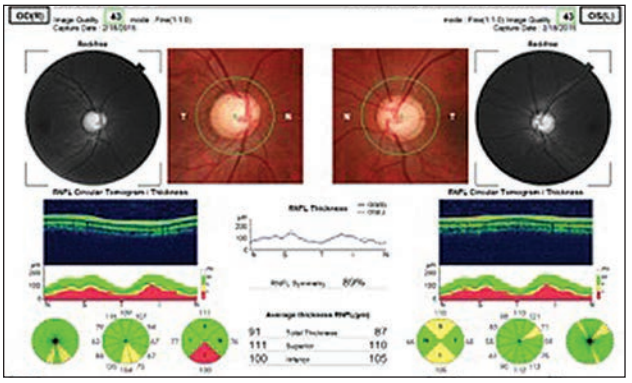
On follow-up two weeks later, IOPs were 32mm Hg OD and 30mm Hg OS. The patient was instructed to discontinue Xolamol and Lumigan and was started on Alphagan (brimonidine 0.2%, Allergan) TID OU, Ganfort (bimatoprost 0.03%/timolol 0.5%, Allergan) QD OU, and 500mg acetazolamide PO BID initially, which was reduced to QD after one week.

Two weeks after that visit, the patient's IOP was 22mm Hg OD and 20mm Hg OS. He was maintained on this dosage until mid-May, at which time his IOPs were "greater than 30mm Hg OU." At this point, the treating physician discussed surgical options with the patient. Apparently, the discussion centered on the severity of the disease, the inability to adequately maintain acceptable IOP and the suggestion that surgical intervention may offer some temporary relief. But, the provider told the patient the overall visual prognosis was not good and visual impairment was likely to occur in the near future.

Needless to say, such a prognosis was upsetting for this 22-year-old and his family. Ultimately, they reached out to me to provide an independent opinion on the matter.

When looking over the information provided, one thing jumped out at me immediately: both optic discs were anatomically large, with

the right larger than the left. Closer examination of the photos showed relatively symmetrical neuroretinal rims, devoid of any notching or damage consistent with advanced glaucoma. In short, the patient had big optic nerves with accompanying big cups. In fact, given the data available, the nerves looked rather normal. So, perhaps the treating physician may have labeled these particular optic nerves as glaucomatous, rather than large physiological nerves?



Clinical data for this patient was limited, but images did show large optic nerves in anatomically large optic cups.

However, IOP was elevated above 30mm Hg on initial presentation. Even more importantly, post-treatment IOPs were not stable and varied considerably. What was lacking in the initial information provided to me were pachymetry readings. It's quite possible that the elevated IOPs were simply a result of thick corneas; in the presence of a large cup, maybe this was just a case of an anatomical situation mimicking glaucoma, rather than frank glaucoma.

But even if his central corneas were thick, which would account for the IOP readings above 30mm Hg, that would not explain the variability in the post-treatment IOP readings. With fluctuations as wide as they have been recorded, it made me wonder what the anatomy of the anterior segment looked like—the angle anatomy in particular. While his optic nerves did appear to be normal though large in appearance, it's possible that the patient may in fact have a mixed mechanism or narrow angle component to his condition.

Ultimately, two important items quickly rose to the surface:

- First, while the actual diagnosis of glaucoma is not firm, the patient does not seem to be in imminent danger of losing vision as his neuroretinal rims appear healthy.
- Second, as in most cases of "social consults," more information is needed—CCT readings and gonioscopic examination, in this particular case. But, when asked to render an opinion about a patient you've never seen, you'll invariably be handicapped by some lack of information, and what information you do have will be secondhand.

So, what did I tell my shoemaker friend? What was I comfortable saying, and what was I comfortable in declining to say?

Discussion

Given that I know the acquaintance well, I felt I could speak frankly. I reassured him that his nephew did not seem to be in danger of "going blind" in the next few months. This was a major relief to him, his family and the patient, as this was their main concern at this time.

But I did explain to him that some information was missing, so making a firm diagnosis was impossible. I also warned him that his nephew was at risk and needed further evaluation. Although medications used in Europe may differ from those in the United States, that doesn't sufficiently explain why this patient's IOPs are so variable, given the broad coverage of medications he is on—a question I could not answer with the information I had.

Here are my suggestions on how to handle the situation when you're asked to render an opinion on a patient you haven't seen:

1. Remember, any information you are given is secondhand at best and may be incorrect.
2. You will probably not be given all the information you need.
3. Extenuating circumstances will likely affect what you are comfortable divulging, such as your relationship with the individual asking the question, the patient and the provider rendering the care.
4. Try to put a positive spin on the case and be encouraging, although that's sometimes hard to do.
5. Try not to disparage another provider. You can work around a situation when the rendered care appears substandard simply by suggesting that in "difficult cases such as this, a second opinion is always a good thing to consider."
6. If you're not comfortable with the line of questioning for whatever reason, just tell the other person that it's not good practice to proffer advice without being able to examine all the facts.
7. Lastly, always treat the questioner as you would want to be treated—the "golden rule" of patient care and life.

In short, it all boils down to being comfortable—for both you and the person asking for your opinion. ■

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A Yellow Polka-Dotted Retina

Can this patient's fundus exam reveal the cause of his visual distortion?

By Mark T. Dunbar, OD

A 16-year-old white male presented complaining of "shiny gray spots" in his peripheral vision, more in the right eye than the left. He also reported a mild decrease in vision in both eyes, which started about a month earlier. Finally, he noted an increase in floaters in both eyes with no complaints of flashes.

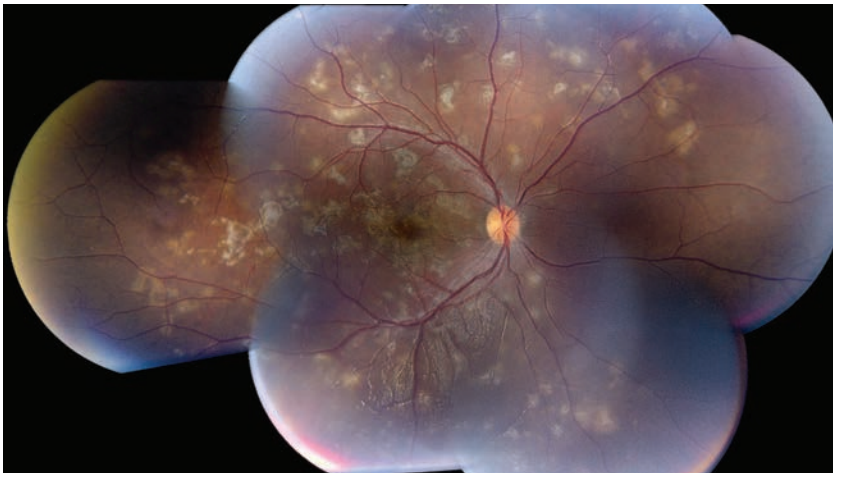
His medical history was unremarkable and he reported good general health. He was not on any medications and denied any previous history of vision problems.

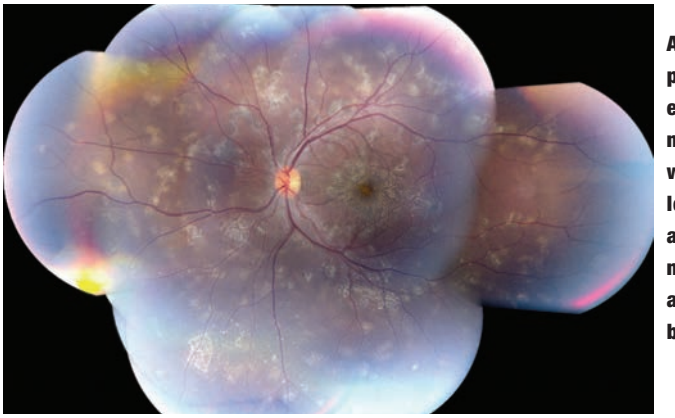
Upon examination, his best-corrected vision was 20/40 in the right eye and 20/30 in the left eye. His pupils, confrontation visual fields and motility were all within normal limits in both eyes.

Anterior segments were also unremarkable with no inflammation noted in the anterior chambers of either eye.

Upon dilation, a mild vitritis was noted in both eyes. The fundus examination revealed multiple creamy white "placoid" lesions along the arcades, in the mid-periphery and periphery of both eyes.

Lesions that were similar in appearance, but more pigmented, were scattered throughout the peripheral retina close to the cream-colored lesions, as seen on fundus autofluorescence images.





A 16-year-old patient's fundus exam revealed multiple creamy white "placoid" lesions along the arcades, in the mid-periphery and periphery of both eyes.

Take the Retina Quiz

1. Where are the fundus lesions located?
   a. RPE and choroid.
   b. Vitreous.
   c. Outer retinal layers.
   d. Photoreceptor layer.

2. How should this patient be treated?
   a. Start patient on steroid.
   b. Start patient on antivirals.
   c. Perform diagnostic vitrectomy.
   d. Monitor closely.

3. What is the likely etiology?
   a. Infectious.
   b. Viral.
   c. Autoimmune.
   d. Unknown.

4. What's the likely diagnosis?
   a. Acute posterior multifocal placoid pigment epitheliopathy. (AMPPE)
   b. Serpiginous chorioretinopathy.
   c. Ampiginous chorioretinopathy.
   d. Infectious retinitis.

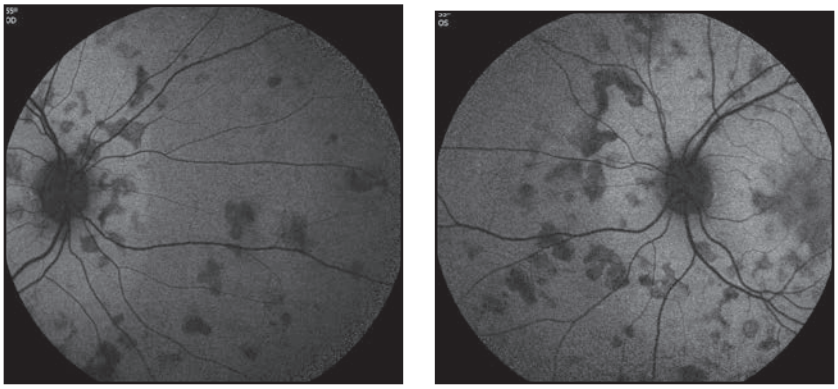
For answers see page 130.

Diagnosis

We ordered a systemic medical workup for our patient, which revealed normal complete blood count (CBC), rapid plasma reagin (RPR), and erythrocyte sedimentation rate (ESR). Tests for Lyme disease, *Bartonella* species and HIV came back negative. His thyroid panel was within normal limits and a chest X-ray was unremarkable.

Our patient returned in two days with a further decrease in vision to 20/60 OD and 20/40 OS. More creamy-white placoid lesions had developed. We started him on oral prednisone 80mg a day. Over three months, the patient had a gradual improvement of vision to 20/30 OD and 20/25 OS and stabilization of retinal lesions. The prednisone was tapered and then discontinued six months after the initial presentation; however, over the next six months, the symptoms returned and new active placoid lesions were noted. The patient was restarted on 40mg a day of prednisone.

Based on the clinical presentation and disease course, our patient has a rare condition called ampiginous chorioretinopathy.



Fundus autofluorescence shows more lesions scattered througout the peripheral retinas in both eyes.

Ampiginous Chorioretinopathy

Ampiginous chorioretinopathy is an inflammatory condition of the retinal pigmented epithelium and the inner choroid. It is similar to acute posterior multifocal placoid pigment epitheliopathy (APMPPE) and serpiginous choroiditis but has distinct "phenotypical" features and clinical course that sets it apart from the other white-dot syndromes.

Ampiginous chorioretinopathy causes bilateral, yellowish-white placoid lesions with geographic borders in the mid-peripheral and peripheral fundus.[1] The location of these lesions is unlike serpiginous choroiditis and APMPPE, in which lesions are located at the posterior pole. The posterior pole may become involved, but this rarely occurs during the initial presentation. The placoid lesions are much smaller (approximately a half disc diameter) compared with APMPPE and serpiginous lesions.[2] Ampiginous lesions are recurrent and relentless, like serpiginous, but unlike APMPPE.

Ampiginous chorioretinitis has a male predominance and usually presents in the third or fourth decade of life.[2] Common presenting symptoms are sudden, painless loss of vision, new onset floaters, metamorphopsia, or any combination.

Disease Stages

In the acute stage of the disease, fundus exams reveal multiple bilateral yellow to gray colored lesions involving the mid-peripheral and peripheral retina. The macula and posterior pole are spared during the first presentation, but can become involved during recurrent episodes.

There is minimal to 2+ vitritis during the active stage of the disease with occasional subretinal fluid.[1] In some cases, subretinal fibrosis and an epiretinal membrane are present.[2,3] Anterior segment inflammation may be present with occasional keratic precipitates and episcleritis.[1-3]

On fluorescein angiography, central hypofluorescence of the active lesion exists with hyperfluorescent margin during the early phase.

The late phase shows hyperfluorescence of the entire lesion. APMPPE lesions, unlike serpiginous and ampiginous chorioretinitis, block fluorescence completely during the early phase of fluorescein angiograpy.[1]

Active Phase

On fundus autofluorescence, during the active phase of the disease, there is a subtle, diffuse increased autofluorescence of the lesion. A few weeks into the active phase, the lesions reveal a more intense, discrete and coalesced autofluorescence, which decreases over several weeks and eventually shows decreased autofluorescence as the lesions become quiescent.

With our patient, we noted increased autofluorescence of the creamy placoid lesions in both eyes and decreased autofluorescence of the pigmented lesions.[4]

Relentlessness

Another diagnostic characteristic of ampiginous chorioretinitis is its relentless nature. Various case series

show a relapse rate of 35% to 67%.[2] Some studies show a relapse rate as high as 81%.[2]

Relapse can occur as early as six months to as late as five years. During a recurrent phase, new active white placoid lesions often accompany older, healed lesions with chorioretinal atrophy. In severe recurrent cases, more than 50 lesions can be found scattered throughout the fundus.[1,2]

Treatment

Ampiginous chorioretinopathy is treated with a steroid combined with an immunosuppressive agent. Most reported cases required prolonged treatment with the steroid, or immunosuppressants, or both, to prevent recurrences.[1] Subtenon steroid injections have also been used to treat more aggressive and persistent disease.

No reports show improvement of viral prodrome with antiviral therapy.[1]

The prognosis of ampiginous chorioretinopathy is variable. Permanent vision loss is usually rare unless the fovea is involved.[1]

Our patient's final visual acuity was 20/30 OD and 20/25 OS. He has had several recurrences over a period of two years upon discontinuation of the steroids. He was put on a maintenance dose of 15mg of methotraxate weekly and has been quiescent since. He is being followed every six months and follows up with his rheumatologist on a regular basis. ■

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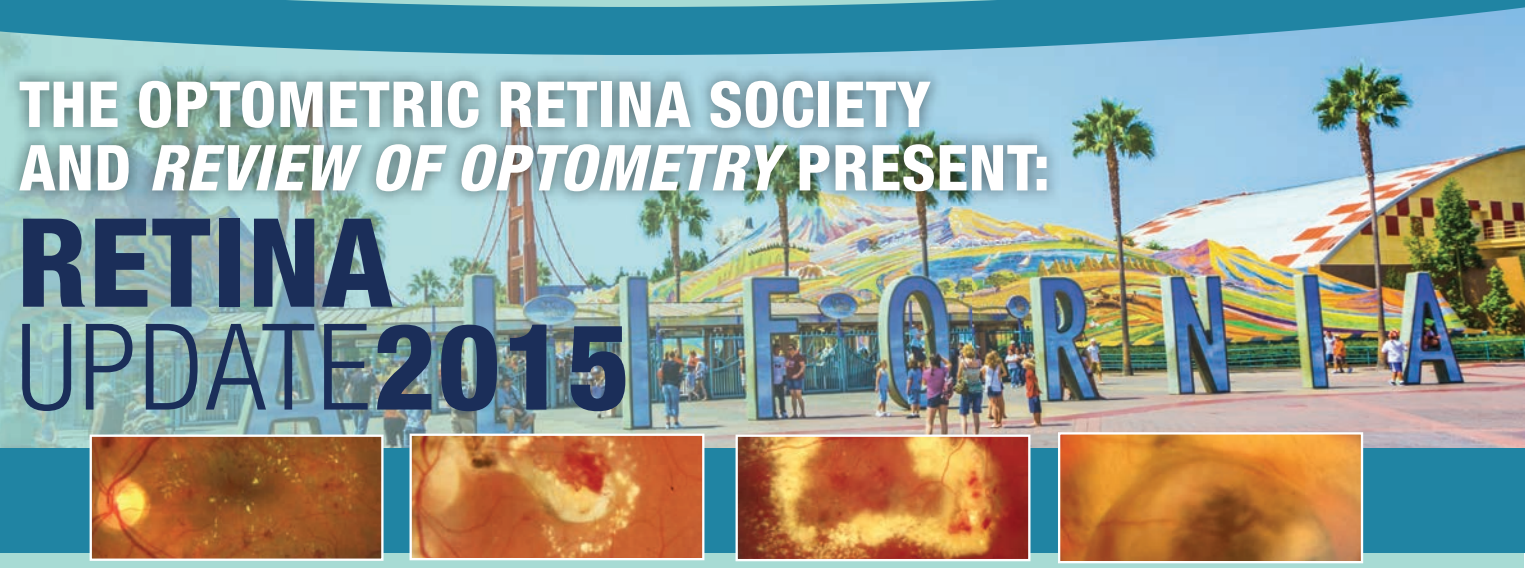

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- Macular Holes and Epiretinal Membranes
- What's New in Retinal Imaging

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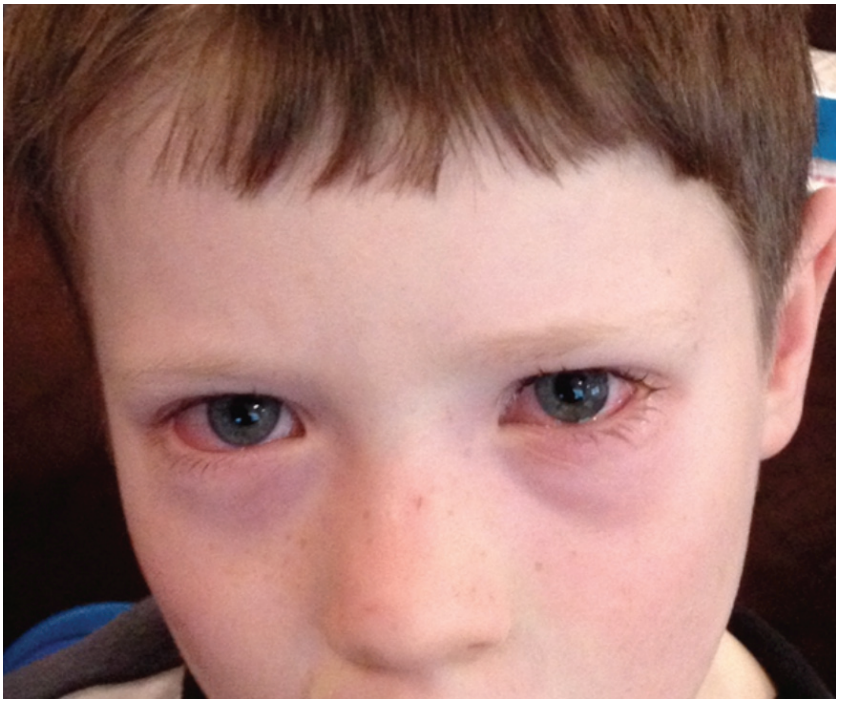
Undercover Ophthalmic Agents

Industry outreach may be slowing down, but you can still keep up on the latest up-and-coming drugs. **By Alan G. Kabat, OD, and Joseph W. Sowka, OD**

Large pharmaceutical companies used to introduce new drugs to the market with a great deal of fanfare. Remember the days of listening to podium talks, reading sponsored supplements or participating in webinars where landmark drugs such as Restasis (cyclosporine, Allergan) Travatan (travoprost, Alcon) or Zylet (loteprednol/tobramycin, Bausch + Lomb) were first launched? Check the back of your desk drawer—you may still have a pen or notepad bearing these logos. Well, that's all changed now. Industry has diminished its promotional outreach in an effort to adhere to Pharmaceutical Research and Manufacturers of America (PhRMA) guidelines.

Indeed, those days of passive learning are behind us. The result of this diminished exposure is reduced physician awareness of new products. We're here to make sure you're not left in the dark. In this month's column, we hope to shine a spotlight on some recent introductions to the ophthalmic "medicine cabinet."



Several new pharmaceutical options are now available to help patients, like this little guy, with allergic rhinoconjunctivitis.

Allergy

It's likely you've at least heard of Pazeo (olopatadine 0.7%, Alcon Laboratories), which received FDA approval in January. At 0.7%, Pazeo contains seven times the olopatadine concentration in the company's original olopatadine formulation, Patanol, and 3.5 times the concentration in Pataday. Pazeo is approved for the relief of ocular itching associated with allergic conjunctivitis, with a recommended dosing of once daily. Although Pataday also enjoys once daily dosing, Pazeo demonstrated slightly greater relief of itching at 24 hours post-treatment in a head-to-head comparison with Pataday.[1] While Pazeo banners were visible at the SECO International meeting in March, little information has trickled down to practitioners. Samples have yet to be received at either of our academic institutions.

Allergen-specific immunotherapy has traditionally been employed in younger patients whose symptoms are not adequately controlled with pharmacotherapy, or who cannot tolerate anti-allergy medications.[2,3] In years past, this consisted of "allergy shots"—subcutaneous injections of the actual allergen, given in an effort to progressively desensitize the patient's immune system.[4] The recently developed sublingual immunotherapy (SLIT) technology incorporates an allergen extract in dissolvable tablet form placed under the tongue for a defined amount of time and then swallowed. Three such products received FDA approval in 2014.

These include Oralair (sweet vernal, orchard, perennial rye, timothy and Kentucky blue grass mixed pollens allergen extract, Greer Labs), Grastek (timothy grass pollen allergen extract, Merck) and Ragwitek (short ragweed pollen allergen extract, Merck).[5-7]

All of these products are indicated "as immunotherapy for the treatment of…pollen-induced allergic rhinitis, with or without conjunctivitis." Hence, these agents may have implications for many of our patients.

The FDA further requires that all individuals receiving SLIT for allergy management must have laboratory confirmation by either a positive skin test or in vitro testing for pollen-specific IgE antibodies.[5] We support recommendations that such testing and initiation of therapy be performed only by physicians certified in the treatment of allergy by the American Board of Allergy and Immunology (ABAI).

Glaucoma

A recent study published in the *American Journal of Ophthalmology* concluded that Izba (travoprost 0.003%, Alcon), a glaucoma medication approved last year, provided equivalent IOP-lowering efficacy to benzalkonium chloride-preserved travoprost 0.004% in patients with open-angle glaucoma and ocular hypertension.[8] Although Izba is currently listed on the FDA's website of approved drug products (www.accessdata.fda.gov/scripts/cder/drugsatfda/), we are not aware of any marketing efforts in the United States to date, and the product is not currently available for purchase in pharmacies. A generic version of travoprost 0.004% (Par Pharmaceutical) received FDA approval in 2013,

Generic gatifloxacin and travoprost are now available in the United States.

and just recently came to market.

Other Generics

While we're on the subject of generics, three generic versions of bimatoprost 0.03% will soon hit the market from manufacturers Lupin Pharmaceuticals, Apotex and Alcon, the last of whom, in addition to making numerous branded pharmaceuticals, has a thriving business in generics (Falcon). While generic bimatoprost is not yet available in the United States, we anticipate seeing pharmacies carry this product within the next year or two.

A generic version of Zymaxid (0.5% gatifloxacin, Allergan) also recently hit US pharmacies. Both Lupin Pharmaceuticals and Hi-Tech Pharmacal have gained FDA approval to manufacture 0.5% gatifloxacin solution, although the latter company's product is being marketed through Akorn. The price of generic gatifloxacin appears to be substantially less than branded Zymaxid and, to our knowledge, this is the first topical fourth-generation fluoroquinolone to attain generic status.

Other Ophthalmic Agents

Two additional drugs with ophthalmic indications received FDA approval in 2014, although it is unlikely optometrists will directly use either. Omidria (intracameral phenylephrine 1%/ketorolac 0.3%, Omeros) is an intraocular injection indicated for maintenance of pupil size by preventing intraoperative miosis, as well as the reduction of postoperative pain associated with cataract surgery or intraocular lens replacement.[9]

Adequate dilation is crucial to a positive outcome in the aforementioned procedures, and numerous factors can contribute to poor or ill-sustained pharmacologic mydriasis; these can include long-term use of topical miotic agents (e.g. pilocarpine), diabetes, pseudoexfoliation syndrome, prior trauma or uveitis, and intraoperative floppy iris syndrome associated with chronic use of oral alpha-adrenergic antagonists such as Flomax (tamsulosin, Boehringer Ingelheim).[10] Omidria is added preoperatively to the surgical irrigating solution and is infused into the eye throughout the procedure. This way, rather than diluting the preoperative mydriatics, a relatively constant concentration of phenylephrine and ketorolac is delivered to the anterior chamber. The efficacy and safety of Omidria has been well demonstrated in a large, multicenter international clinical trial.[11]

Finally, Hetlioz (tasimelteon 20mg capsules, Vanda Pharmaceuticals) is indicated for an unusual condition known among sufferers as "non-24." Non-24-hour sleep-wake disorder is a serious, chronic malady that disrupts an individual's circadian rhythms due to an inability to differentiate day from night. Although it may rarely affect sighted or partially-sighted individuals, non-24 is most common in those with no light perception, affecting between 55% and 70% of totally blind patients.[12] Researchers believe

it results from a dysregulation of endogenous melatonin secretion in the brain, with resultant somnolence during daytime hours as well as insomnia at night. Hetlioz is a melatonin-receptor agonist, helping to both stimulate the sleep centers in the brain while restoring a more synchronous, regular circadian rhythm.[13] Although Hetlioz is not the first melatonin-receptor agonist to be approved by the FDA, it is the first drug to be specifically indicated for the treatment of non-24.[13]

While this agent is not classified by the FDA as a scheduled drug, only a sleep specialist is qualified to confirm a diagnosis of non-24, and therefore the prescription use of Hetlioz is most appropriately limited to these physicians. ■

Dr. Kabat is clinical care consultant at TearWell Advanced Dry Eye Treatment Center in Memphis, Tenn. Neither he nor Dr. Sowka have any direct financial interest in the products mentioned above.

Two new drugs with implications in eye care: Hetlioz is indicated for management of non-24, while Omidria is used to maintain pupillary dilation during intraocular surgery.

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RGN-0237

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

Crystal Clear Results

Keep these post-op concerns in mind to ensure Crystalens patients have good outcomes.

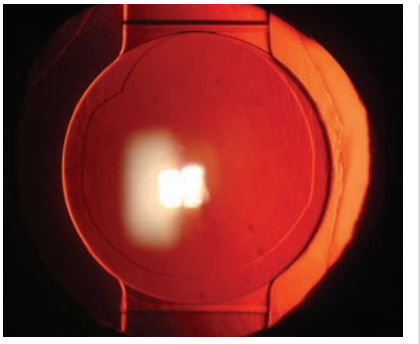
Correcting near vision with an intraocular lens is one of the toughest challenges in refractive cataract surgery. While multifocal IOLs are often an excellent solution, they come with limitations such as reduced contrast sensitivity and night vision problems. For many cataract patients, an accommodating IOL is a promising alternative. As a monofocal lens, it focuses all available light to a single point, thereby addressing the issues seen with multifocal IOLs, while still providing improved near power. This is especially advantageous when any ocular pathology that affects vision is present, such as significant ocular surface disease, corneal dystrophies such as EBMD, macular pathologies or glaucoma.

And these lenses provide at least some accommodation, unlike standard IOLs. Due to anatomical differences, patients will experience varying amounts of accommodation: some may experience over two diopters of accommodation, but most will experience around 1.00D to 1.50D. In rare cases, a patient will experience little to none.

Crystalens (Bausch + Lomb)—the only accommodating IOL marketed in the US—has been available since 2003 and has seen several design updates. It's a silicone elastomer-based plate haptic lens. Two hinged haptics allow the optic to vault with accommodation of the ciliary muscle, creating an accommodative effect on the visual system.

Implantation & Post-op Care

Surgical implantation of Crystalens is slightly different than a traditional IOL because of its design and larger size. A different injector is used to implant the lens through a slightly larger corneal incision. Although the rotational orientation of the Crystalens inside the eye does not matter, there is a toric version of the lens, Trulign (Bausch + Lomb), that requires on-axis rotation for astigmatism correction. In addition, the surgeon will typically spend extra time polishing the posterior capsular surface to prevent early postoperative capsular opacification. The lens will end up sitting in the capsular back, with the optic posterior to the two hinged plate haptics.



A Crystalens in place. Its hinged plate haptics bend in response to ciliary contraction, mimicking accommodation.

The accommodative effects of the lens are often not realized for several weeks to months after implantation, and patients should be counseled accordingly. The targeted refraction should be realized immediately, and postoperative management for the first several weeks is no different than a standard IOL.

There are some unique concerns with Crystalens that become significant several weeks post-op. These involve capsular contraction and opacification and can affect vision in several different ways. Any amount of capsular contraction can limit the movement of the lens and push it slightly anteriorly. This can result in limited accommodation and a slight myopic refractive shift. Both of these issues will resolve with capsular tension release provided by a YAG capsulotomy.

If the contraction is excessive or asymmetric, one of the bendable plate haptics can fold forward, leaving the lens tilted in the capsular bag. With one haptic folded anteriorly and one posteriorly, the lens will look like the letter Z in the bag, known as Z syndrome. Your first sign of this will be a refractive change and decreased uncorrected visual acuity several weeks after surgery. Refractive errors will often mimic crossed cylinder effects and give you hyperopic refractions with roughly double the amount of cylinder. This complication should be addressed in a timely manner and, again, is often alleviated with a simple YAG capsulotomy. Z syndromes are rare, but do need to be ruled out for any Crystalens patient who has a refractive shift.

Because capsular contraction can occur in the peripheral part of the capsular bag, it is important to dilate any Crystalens patient who is not achieving expected results. Dilation can be considered as soon as several weeks post-op, but is usually not necessary for at least a month. Luckily, most Crystalens complications are temporary and easily alleviated with a YAG capsulotomy. Think YAG early and often to keep these patients happy. ■

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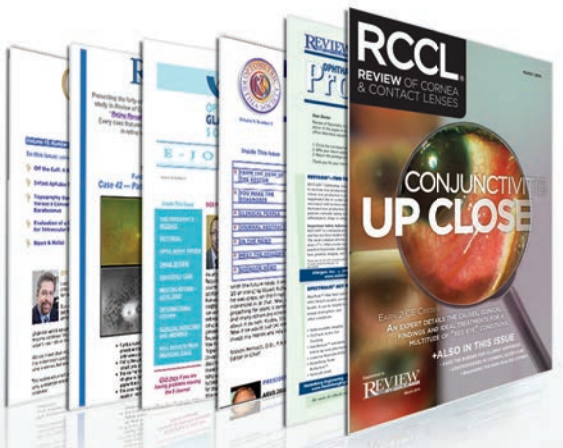

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Product Review

Contact Lenses

New Daily Disposable for Presbyopia

Patients with presbyopia can now consider switching to 1-day Acuvue Moist Multifocal contact lenses. The new lenses address the natural variation in pupil size and are designed according to age and refractive error ranging across 61 distance powers and three add powers, according to Johnson & Johnson Vision Care.

The lenses come in powers from -9.00D to +6.00D in 0.25D steps, with low add powers from +0.75D to +1.25D, mid add powers from +1.50D to +1.75D and high add powers from +2.00D to +2.50D.

Visit www.acuvueprofessional.com.

Extended Depth of Focus Contact Lenses

Presbyopia patients now have a new contact lens option with Brien Holden Vision's (BHV) extended depth of focus (EDOF) contact lenses, which recently received FDA clearance. BHV's new generation of EDOF contact lenses uses higher-order aberrations to improve retinal image quality over a wide range of distances from far to near while minimizing ghosting and haloes, the company says.

The lenses, available at the end of 2015, perform relatively independent of a patient's natural aberrations and variation in pupil size and are designed to meet the vision needs of emerging presbyopes, middle aged and older people, the company says.

Visit www.brienholdenvision.org.

New Plus Powers and Packaging

More patients now have the option to use Miru 1day Menicon Flat Pack. Its expanded powers range from +0.50D to +4.00D in 0.25D increments. They are available in trial six-packs, 30-packs and 90-packs.

The lenses also come in new packaging made from recycled molds with parameter information laser etched on the case, ensuring legible text that cannot be damaged or worn down over time, the company says.

A rebate of up to $110 on an annual supply of lenses is available to qualifying patients, the company says.

Visit www.meniconamerica.com.

Contact Lens Technology

Practitioner Advice for CL Success

A new series of interviews may help you better understand the ins and outs of Duette contact lenses, according to SynergEyes. In 48 new videos, eight optometrists answer questions on topics such as: advice for new fitters, patient education, practice implications, product comparisons, and insertion and removal tips.

Through these short videos, ODs explain how Duette has had a positive impact on both patient satisfaction and their businesses, SynergEyes says.

The lens features an 84Dk silicone hydrogel skirt around the 130Dk center. A patient's initial pair of Duette lenses can be designed empirically based on refraction and corneal curvature measurements.

Visit www.synergeyes.com/professional.com.

Lid Hygiene

Lid Scrub

Patients with blepharitis have a new lid scrub that may help increase moisturizing throughout the day, according to OcuSoft. The company's OcuSoft Lid Scrub Plus Platinum contains 0.2% phytosphingosine, a water-binding agent that mimics the natural lipid layer of the outer epidermis for increased moisturizing, it says.

OcuSoft Lid Scrub Plus Platinum is an extra strength leave-on eyelid cleanser containing surfactants plus a moisturizer and preservative blend. According to OcuSoft, the lid scrub eradicates seven different strains of bacteria commonly found on the eyelids, including MRSA and *Staph. epidermidis*.

Visit www.ocusoft.com.

Practice Management

Guaranteed ROI Program

Practices can now evaluate the ROI they can generate with an OCT before purchasing. Optovue's Guaranteed ROI Program lets practices try an iVue, iFusion or iScan SD-OCT for up to 60 days. The company installs the instrument at no charge to the practice and provides an in-practice training session as well as ongoing support from a practice implementation consultant.

At the end of the evaluation period, the company will provide an ROI report detailing the practice's ability to generate revenue with the OCT. The practice may then purchase or return the instrument.

Visit www.optovue.com. ■

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Expert Panel on Glaucoma Diagnosis and Treatment
What the Primary Care Optometrist Should Never Miss
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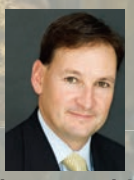

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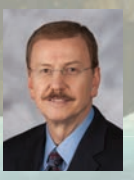

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Optional Catamaran/Snorkeling Activity, Saturday July 25 - $125 (includes lunch) This 3.5-hour trip includes round trip transportation from Wailea Marriott to Maalaea Harbor, all snorkel gear, expert instruction, plus a barbecue lunch and open bar! Not that into the snorkeling scene? No worries, take your pick of flotation devices—including an oversized boogie board with built-in viewing port—or use the onboard waterslide to take a dip in the Pacific Ocean. But that's not all—the Four Winds II features a glass bottom for 180° of underwater viewing!	$125	x	_____	=	$_____
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- Full refund on registration fee until June 23, 2015
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Meetings + Conferences

June 2015

■ **19-21.** *2015 VOA Annual Conference.* Hilton, McLean, VA. Hosted by: Virginia Optometric Association. To register, call Bo Keeney at (804) 643-0309.

■ **24-28.** *Optometry's Meeting 2015.* Washington State Convention Center, Seattle, WA. Hosted by: American Optometric Association and American Optometric Student Association. To register, go to http://optometrysmeeting.org.

■ **26-July 5.** *A Comprehensive Update on Contemporary Eye Care.* Northern European Capitals Cruise, departs Copenhagen, Denmark. Hosted by: Dr. Travel Seminars/The New Jersey Society of Optometric Physicians. Key faculty: Randall Thomas. CE hours: 12. To register, email Robert Pascal at info@DrTravel.com or go to DrTravel.com.

July 2015

■ **4-11.** *Tropical CE Puerto Rico.* El Conquistador-Waldorf Astoria, Puerto Rico. Hosted by: Tropical CE. Key faculty: Jimmy Bartlett, Kim Reed. CE hours: 20. To register, call Stuart Autry at (281) 808-5763, email sautry@tropicalce.com or go to www.tropicalce.com.

■ **10-12.** *21st Conference on Clinical Vision Care.* Southern College of Optometry, Memphis, TN. Hosted by: OEP Foundation. CE hours: 17. To register, email Theresa Krejci at theresakrejciOEP@verizon.net or go to www.oepf.org.

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

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

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

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

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

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

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

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

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

August 2015

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

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

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

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

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

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

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

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

■ **28-30.** *Alumni Weekend*. UAB School of Optometry, Birmingham, AL. Hosted by: UAB School of Optometry. Key faculty: Ian Gaddie, Marie Bodack, Diana Shechtman, Scot Morris, Sunny Sanders. CE hours: 18. To register, email Katherine Clore at kclore@uab.edu, call (205) 934-5700 or go to www.uab.edu/optometry.

September 2015

■ **2-13.** *Adventure CE Italy*. Siena/Sorrento/Rome, Italy. Hosted by: Tropical CE. Key faculty: Jill Autry, Ian Ben Gaddie. CE Hours: 20. To register, email Stuart Autry at sautry@tropicalce.com, call (281) 808-5763 or go to www.tropicalce.com.

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

■ **10-13.** *GWCO Congress 2015*. Oregon Convention Center, Portland, OR. Hosted by: Great Western Council on Optometry. Key faculty: Paul Karpecki, April Jasper, Mile Brujic. CE hours: Total: 71; 26 per OD. To register, email Tracy Oman at gwco@gwco.org, call (503) 654-1062 or go to www.gwco.org. ■

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2 Hr CE/TQ

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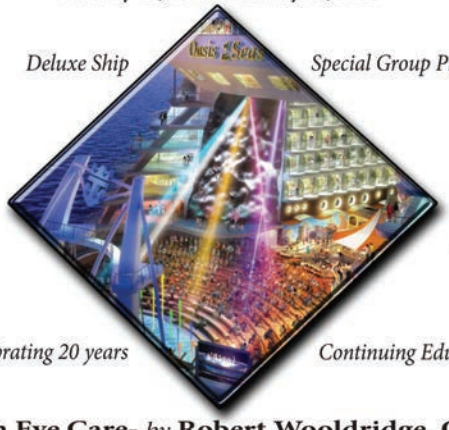

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TRAVATAN Z®

(travoprost ophthalmic solution) 0.004%

BRIEF SUMMARY OF PRESCRIBING INFORMATION

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

DOSAGE AND ADMINISTRATION
The recommended dosage is one drop in the affected eye(s) once daily in the evening.
TRAVATAN Z® (travoprost ophthalmic solution) should not be administered more than once daily since it has been shown that more frequent administration of prostaglandin analogs may decrease the intraocular pressure lowering effect.

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

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

CONTRAINDICATIONS
None

WARNINGS AND PRECAUTIONS
Pigmentation
Travoprost ophthalmic solution has been reported to cause changes to pigmented tissues. The most frequently reported changes have been increased pigmentation of the iris, periorbital tissue (eyelid) and eyelashes. Pigmentation is expected to increase as long as travoprost is administered. The pigmentation change is due to increased melanin content in the melanocytes rather than to an increase in the number of melanocytes. After discontinuation of travoprost, pigmentation of the iris is likely to be permanent, while pigmentation of the periorbital tissue and eyelash changes have been reported to be reversible in some patients. Patients who receive treatment should be informed of the possibility of increased pigmentation. The long term effects of increased pigmentation are not known.

Iris color change may not be noticeable for several months to years. Typically, the brown pigmentation around the pupil spreads concentrically towards the periphery of the iris and the entire iris or parts of the iris become more brownish. Neither nevi nor freckles of the iris appear to be affected by treatment. While treatment with TRAVATAN Z® (travoprost ophthalmic solution) 0.004% can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly.

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

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

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

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

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

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

ADVERSE REACTIONS
Clinical Studies Experience
Because clinical studies are conducted under widely varying conditions, adverse reaction rates observed in the clinical studies of a drug cannot be directly compared to rates in the clinical studies of another drug and may not reflect the rates observed in practice. The most common adverse reaction observed in controlled clinical studies with TRAVATAN® (travoprost ophthalmic solution) 0.004% and TRAVATAN Z® (travoprost ophthalmic solution) 0.004% was ocular hyperemia which was reported in 30 to 50% of patients. Up to 3% of patients discontinued therapy due to conjunctival hyperemia. Ocular adverse reactions reported at an incidence of 5 to 10% in these clinical studies included decreased visual acuity, eye discomfort, foreign body sensation, pain and pruritus. Ocular adverse reactions reported at an incidence of 1 to 4% in clinical studies with TRAVATAN® or TRAVATAN Z® Solutions included abnormal vision, blepharitis, blurred vision, cataract, conjunctivitis, corneal staining, dry eye, iris discoloration, keratitis, lid margin crusting, ocular inflammation, photophobia, subconjunctival hemorrhage and tearing.

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

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

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

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

There are no adequate and well-controlled studies of TRAVATAN Z® (travoprost ophthalmic solution) 0.004% administration in pregnant women. Because animal reproductive studies are not always predictive of human response, TRAVATAN Z® Solution should be administered during pregnancy only if the potential benefit justifies the potential risk to the fetus.

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

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

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

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

NONCLINICAL TOXICOLOGY
Carcinogenesis, Mutagenesis, Impairment of Fertility
Two-year carcinogenicity studies in mice and rats at subcutaneous doses of 10, 30, or 100 mcg/kg/day did not show any evidence of carcinogenic potential. However, at 100 mcg/kg/day, male rats were only treated for 82 weeks, and the maximum tolerated dose (MTD) was not reached in the mouse study. The high dose (100 mcg/kg) corresponds to exposure levels over 400 times the human exposure at the maximum recommended human ocular dose (MRHOD) of 0.04 mcg/kg, based on plasma active drug levels. Travoprost was not mutagenic in the Ames test, mouse micronucleus test or rat chromosome aberration assay. A slight increase in the mutant frequency was observed in one of two mouse lymphoma assays in the presence of rat S-9 activation enzymes.

Travoprost did not affect mating or fertility indices in male or female rats at subcutaneous doses up to 10 mcg/kg/day [250 times the maximum recommended human ocular dose of 0.04 mcg/kg/day on a mcg/kg basis (MRHOD)]. At 10 mcg/kg/day, the mean number of corpora lutea was reduced, and the post-implantation losses were increased. These effects were not observed at 3 mcg/kg/day (75 times the MRHOD).

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

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

Handling the Container
Patients should be instructed to avoid allowing the tip of the dispensing container to contact the eye, surrounding structures, fingers, or any other surface in order to avoid contamination of the solution by common bacteria known to cause ocular infections. Serious damage to the eye and subsequent loss of vision may result from using contaminated solutions.

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

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

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

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Waiting is the Hardest Part

By Andrew S. Gurwood, OD

History

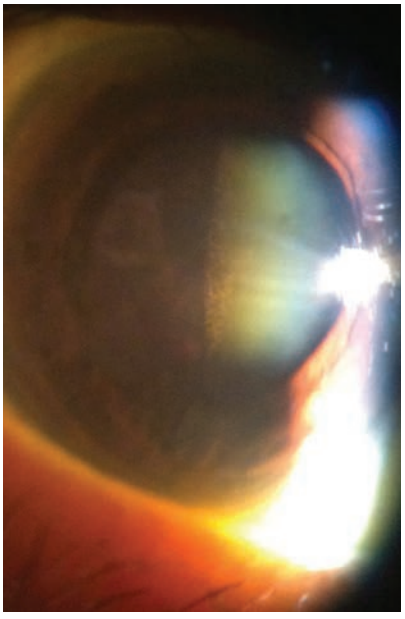
A 67-year-old black female reported to the office with a chief complaint of vision loss in her right eye for five months. She explained that she began to see spots three months ago but that she waited to make an appointment for almost four months. She added that since it didn't hurt, even though her vision was getting worse, she didn't think it was an emergency.

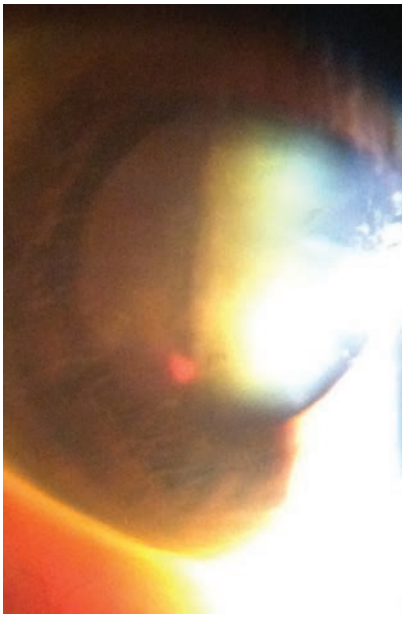
Her ocular history was non-contributory and her systemic history was positive for hypertension and high cholesterol, for which she was medicated with Prinivil (lisinopril, Merck) and Lipitor (atorvostatin, Pfizer). She denied trauma or exposure to chemicals or any allergies.

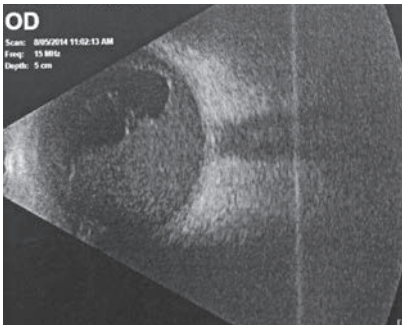
Diagnostic Data

Her best-corrected entering visual acuity was hand motion in her right eye and 20/20 OS at distance and near. Her external examination was normal with evidence of a grade II afferent pupil defect in her right eye.

The biomicroscopic examination of the anterior segment was normal, with Goldmann applanation tonometry measuring 15mm Hg OU. The pertinent posterior segment findings are demonstrated in figures 1 and 2 and ultrasonography in her right eye in figure 3.







Figs. 1 & 2. At top, relevant posterior segment findings.
Fig. 3. At right, the patient's ultrasound. Do these images hint at a diagnosis?

The dilated fundus examination of her left eye was normal.

Your Diagnosis

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

Retina Quiz Answers *(from page 109):* 1) a; 2) a; 3) d; 4) c.

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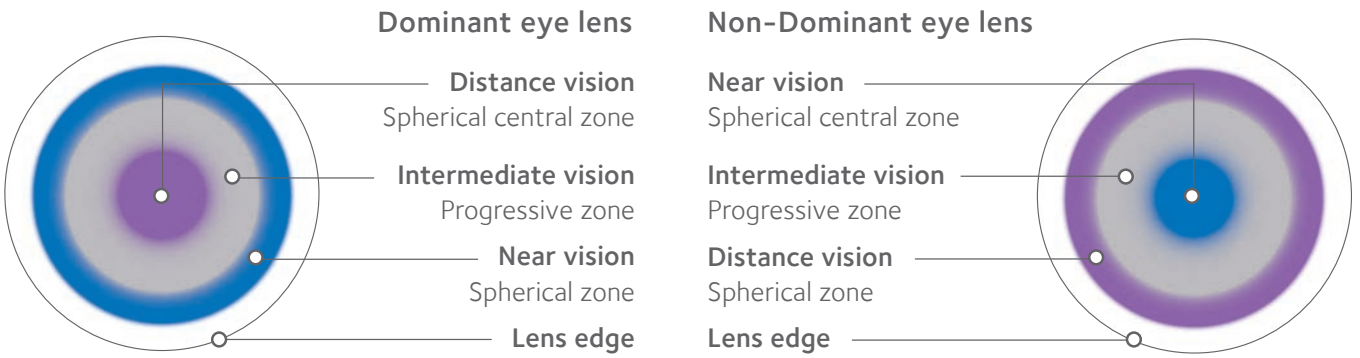

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INDICATIONS AND USAGE

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

Dosage and Administration

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

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

IMPORTANT SAFETY INFORMATION

Warnings and Precautions

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

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

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

Adverse Reactions

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

Use in Specific Populations

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

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

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

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

TRAVATAN Z®

(travoprost ophthalmic solution) 0.004%

Alcon
a Novartis company