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jmi

January 15, 2013

REVIEW® OF OPTOMETRY

Part 1 of 2

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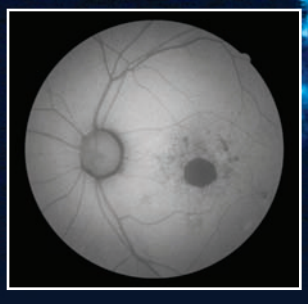
14th ANNUAL Dry Eye Report



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VOL. 150 NO. 1 ■ JANUARY 15, 2013

IN THE NEWS

Retinal arteriolar narrowing might be an **early warning sign** of the development of **open-angle glaucoma**, according to an analysis from the **Blue Mountains Eye Study** published in the January issue of *Ophthalmology*. The researchers analyzed retinal photos of nearly 2,500 participants and found that the risk for OAG at 10 years was about four times higher in patients whose retinal arteries had been narrowest when the study began, compared with those who had had the widest arteries.

It's not clear if the changes are part of the cause of the disease or part of its normal progression. Still, "Our results suggest that a computer-based imaging tool designed to detect narrowing of the retinal artery caliber, or diameter, could effectively identify those who are most at risk for open-angle glaucoma," says lead author **Paul Mitchell, MD, PhD**, of the Centre for Vision Research, University of Sydney. "Such a tool would also need to account for blood pressure and other factors that can contribute to blood vessel changes." Early detection would allow eye doctors to treat patients before optic nerve damage occurs and protect their vision, Dr. Mitchell says.

David Damari, OD, has been appointed **dean of the Michigan College of Optometry at Ferris State University**, effective March 28. Dr. Damari was most recently chair for the Department of Assessment and professor at Southern College of Optometry in Memphis, Tenn. He has served as president for the College of Optometrists in Vision Development and as co-chair at the Summer Institute for Faculty Development for the Association of Schools and Colleges of Optometry.

Multiple Sclerosis Can Be Measured by OCT

Patients whose MS is advancing have faster thinning of the retina, as seen on OCT. **By John Murphy, Executive Editor**

New research suggests that retinal thinning, as measured by OCT, can indicate how fast multiple sclerosis (MS) progresses—especially in the early course of the disease. Also, any eye doctor can measure it using commercially-available OCT equipment with retinal layer segmentation software.

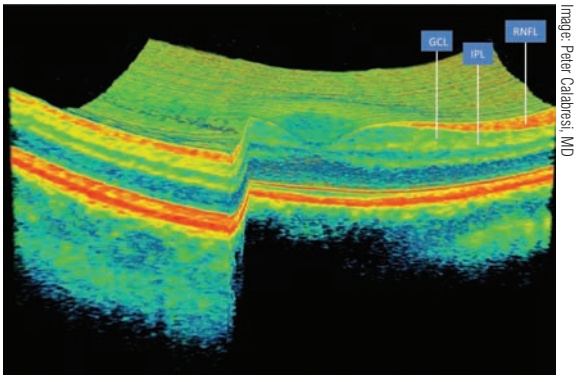

Image: Peter Calabresi, MD

OCT reveals thinning of the ganglion cell/inner plexiform layer, which indicates progression of multiple sclerosis.

In this study, conducted at the Johns Hopkins MS Center in Baltimore, 164 patients with MS and 59 healthy controls underwent spectral-domain OCT scans every six months, for an average of 21 months. Participants were also given MRI brain scans at baseline and at the yearly follow-up.

The researchers found that people with MS relapses had 42% faster thinning of the ganglion cell/inner plexiform (GCIP) layer than people with MS who had no relapses. Also, people with MS who had inflammatory gadolinium-enhancing lesions experienced 54% faster thinning, and those with new T2 lesions had 36% faster thinning than MS patients without these conditions.

People whose level of disability worsened during the study experienced 37% more thinning than those who had no changes in their level of disability. Further, those who had the disease less than five years showed 43% faster thinning than individuals who had the disease more than five years.

Looking to the future, the researchers concluded that OCT-derived GCIP thickness assessment could be used as an outcome measure for evaluating neuroprotective agents, particularly in early, active MS. "As more therapies are developed to slow the progression of MS, testing retinal thinning in the eyes may be helpful in evaluating how effective those therapies are," says lead author Peter Calabresi, MD, director of the Johns Hopkins MS Center.

Ratchford JN, Saidha S, Sotirchos ES, et al. Active MS is associated with accelerated retinal ganglion cell/inner plexiform layer thinning. Neurology. 2013 Jan 1;80(1):47-54.

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Vision Impairment Appears to Be Worsening in US and Worldwide

Around the world and here in the United States, visual impairment appears to be on the rise, despite improved treatment. But, as always, the devil is in the details.

• ***Worldwide***. The long-awaited results of the largest review on global vision impairment and blindness ever undertaken appeared in the December 13 issue of *The Lancet*.[1] Rupert Bourne, FRCOphth, MD, of Anglia Ruskin University's Vision and Eye Research Unit collaborated with 79 ophthalmologists and optometrists to complete the systematic review of all published—and several unpublished—sources of global data on vision impairment and blindness, from 1980 to January 2012.

The bottom line: Treatment for cataracts and other forms of blindness and infectious disease, such as trachoma, has been successful in curtailing vision loss and blindness.

However, the report's statistics indicate that blindness and vision loss has actually increased globally. Why? "The overall increase in the number of people suffering from blindness and vision loss is due to the huge population explosion that has occurred during the last couple of decades," Dr. Bourne says. "However, the Global Burden of Disease findings actually show that this increase is not as large as one would expect given the increasing life expectancy in the world's population over this time."

When age is taken into account, blindness and visual impairment decreased on a worldwide level. "This points to the successful intervention in treating cataracts and other forms of blindness and infectious diseases, such as trachoma," Dr. Bourne says.

The overall increase in the number of people suffering from blindness and vision loss is due to the huge population explosion that has occurred during the last couple of decades.

The largest global cause of vision impairment, at 29.5% of the total, is "other vision loss," which is due primarily to trauma as well as occupational and idiopathic conditions. Second is uncorrected refractive error, which accounts for 26.5% of vision impairment. Cataracts are the third largest contributor at 22.4%. Glaucoma and macular degeneration together account for 10.7%.

• ***United States***. Findings from another large-scale study looking at visual impairment came out around the same time—however, this one looked at non-refractive visual impairment in the US. The prevalence of non-refractive visual impairment increased 21% overall among US adults ages 20-plus, and 40% among non-Hispanic whites ages 20-39. During that same time period, the prevalence of diabetes with 10 or more years since diagnosis also grew, according to the study, published in the December 12 issue of the *Journal of the American Medical Association*.[2]

Researchers at Johns Hopkins University School of Medicine analyzed these changes in prevalence and their relationship to demographic and systemic risks factors in a sample of 10,480 subjects, with data collected through the NHANES study from 1999 to 2002 and from 2005 to 2008. The participants also answered questionnaires and participated in laboratory tests and physical examinations.

The authors suggest that the rise in serious eye conditions—such as cataracts and glaucoma—in the US may be linked, to some degree, with the higher prevalence of diabetes. "If the current finding becomes a persisting trend, it could result in increasing rates of disability in the US population, including greater numbers of patients with end-organ diabetic damage who would require ophthalmic care," the authors wrote. "Continued monitoring of visual disability and diabetes, as well as additional research addressing causes, prevention and treatment, is warranted."

1. Global Burden of Disease Study 2010. Lancet. 2013 Dec 15;380(9859). Available at: www.thelancet.com/themed/global-burden-of-disease. Accessed January 6, 2013.
2. Ko F, Vitale S, Chou CF, et al. Prevalence of nonrefractive visual impairment in US adults and associated risk factors, 1999-2002 and 2005-2008. JAMA. 2012 Dec 12;308(22):2361-8.

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Glaucoma Patients Show Significant Delay in Saccadic Eye Movement

A number of recent studies have highlighted the increased risk of falls and car accidents among glaucoma patients. Now, new research published online in the journal *Eye and Brain* may help to explain why.

Neeru Gupta, MD, PhD, MBA, and a team of researchers at St. Michael's Hospital in Toronto found that saccadic eye movements are significantly delayed in patients with glaucoma, even in those in the early stages of disease. These movements are key in our everyday lives, especially for scanning and navigating the surrounding environment.

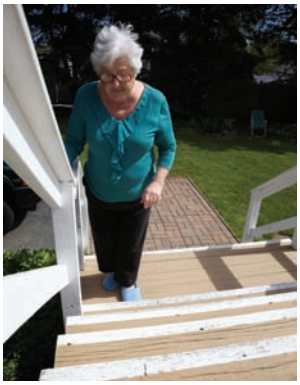

Photo: Mark Lorenz

Glaucoma slows saccadic eye movements, necessary for activities of daily living.

"Now that we know that eye movement reaction times are delayed in people with glaucoma, there is an opportunity to understand the effects of glaucoma on daily activities of living that most of us take for granted, such as walking up and down stairs, driving, navigating and reading," says Dr. Gupta, chief of glaucoma at the University of Toronto. "Just as alcohol causes a delay in hitting the brakes, glaucoma slows the time it takes to move the eyes quickly in response to a visual cue."

His team found that eye movement reaction times in glaucoma patients were delayed by about 15% compared to subjects without glaucoma. Also of interest, saccade parameters showed no significant correlation with visual field loss in glaucoma patients.

Dr. Gupta foresees that measuring these reaction times could provide a useful way to quantify visual loss in glaucoma patients, beyond eye charts or visual field tests.

Kanjee R, Yücel YH, Steinbach MJ, et al. Delayed saccadic eye movements in glaucoma. Eye and Brain. 2012 Nov;(4):63-8.

Nighttime Isn't the Right Time To Replace Extended Wear Lenses

Patients with 30-day extended-wear/continuous-wear (EW/CW) contacts should replace their lenses in the morning rather than at bedtime to reduce their risk for ocular adverse events, according to a study in the December issue of *Optometry and Vision Science*.

The study evaluated 215 patients who wore silicone hydrogel EW/CW lenses. Each day, the patients inserted fresh lenses either at night before going to bed or in the morning after waking. The researchers compared the rate of ocular adverse events between patients who replaced their lenses at night or in the morning vs. a previously studied group of patients who wore the lenses continuously for a month.

Results showed that just 4% of patients who replaced their lenses each morning experienced ocular adverse events, such as infiltrative keratitis or corneal erosion.

By contrast, 8% of patients who replaced their lenses each night and 9% of those who wore their lenses continuously for a month experienced ocular adverse events.

These results indicated that regular nighttime lens replacement did not appear to have any beneficial effect compared to continuous monthly wear—possibly due to handling the lenses, and thus contaminating them, just prior to overnight eye closure.

Further, the study researchers suggested that when users replace EW/CW lenses, they should do so in the morning to limit the risk or ocular adverse events.

Ozkan J, Willcox MD, de la Jara PL, et al. The effect of daily lens replacement during overnight wear on ocular adverse events. Optom Vis Sci. 2012 Dec;89(12):1674-81.

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Aspirin Increases Risk of Wet AMD?

Long-term aspirin use may increase patients' risk for the development of wet age-related macular degeneration, according to a study in the December 19 issue of the *Journal of the American Medical Association*.

The report garnered national attention. However, the study had several limitations—not the least of which is that there was less than 1% difference in the incidence of wet AMD between patients on regular aspirin and those who didn't take aspirin.

In this study, the researchers evaluated 4,926 patients between 43 and 86 years of age who were enrolled in the Beaver Dam Eye Study. During the examination process, the patients were asked whether they had used aspirin at least twice per week for more than three consecutive months.

After a mean follow-up period of nearly 15 years, 512 patients developed early AMD and 117 developed wet AMD. The researchers determined that 1.76% of patients who took aspirin regularly 10 years before undergoing retinal evaluation developed wet AMD vs. 1.03% of patients who had no history of routine aspirin use.

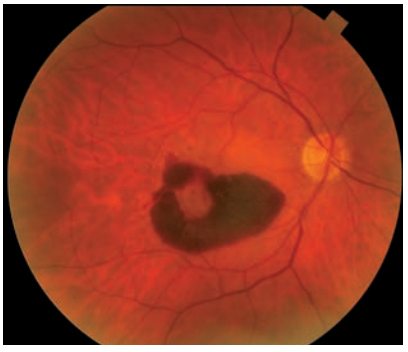

Photo: Steven Ferrucci, OD

A new study has found a slim link between daily aspirin intake and wet AMD.

Further, they found no significant relationship between the amount of aspirin taken and the overall incidence of AMD. Interestingly, researchers found no association between regular aspirin use and the onset of dry AMD.

The researchers acknowledge multiple study limitations, including insufficient data on total aspirin exposure as well as undocumented leukocyte counts and C-reactive protein levels at certain follow-up visits.

"One item we must consider is whether the higher incidence of neovascular AMD was caused by long-term aspirin use itself or by an underlying condition that aspirin therapy was being used to treat, such as carotid artery disease," says Steven Ferrucci, OD, chief of optometry at the Sepulveda VA Ambulatory Care Center and Nursing Home in North Hills, Calif. "Overall, however, such results from one study likely will not change the way I practice when managing wet AMD patients who are on long-term aspirin therapy."

Klein BE, Howard KP, Gangnon RE, et al. Long-term use of aspirin and age-related macular degeneration. JAMA. 2012 Dec 19;308(23):2469-24.

Study: Include Vision Insurance in All Health Plans

It's no surprise that Americans with vision insurance have better vision than those without it. But a new study published in the online edition of *Archives of Ophthalmology* also concluded, "Vision insurance for preventive eye care should cease to be a separate insurance benefit and should be mandatory in all health plans."

Researchers at the University of South Carolina School of Public Health compared the rates of eye care visits and vision impairment among working-age adults with and without vision insurance. The study included 27,152 respondents (between the ages of 40 and 64 years) to the Behavioral Risk Factor Surveillance Survey 2008. Included were 3,158 respondents (11.6%) with glaucoma, AMD and/or cataract. About 40% of the study population had no vision insurance.

According to the study results, individuals with vision insurance were more likely than those without insurance to have attended eye care visits. They also reported that they have no difficulty recognizing friends across the street or reading printed material.

"Lack of vision insurance impedes eye care utilization, which, in turn, may irrevocably affect vision," the authors concluded. "Because our study empirically establishes the consequential link between lack of vision insurance and vision damage mediated by its impact on eye care visits, it provides the needed evidence for policy interventions to mandate vision coverage in all standard health plans."

The researchers add, "Alternatively, federal and state governments may find it beneficial for their own budgets to initiate publicly sponsored eye-screening programs for the uninsured that are similar to those provided under the Best Chance Network for breast and cervical cancer screening."

However, the National Association of Vision Care Plans (NAVCP) took issue with the study. NAVCP says its own study determined that Americans with stand-alone vision plans are twice as likely to get annual eye exams as those with vision coverage bundled into major medical plans.

Li YJ, Xirasagar S, Pumkam C, et al. Vision insurance, eye care visits, and vision impairment among working-age adults in the United States. Arch Ophthalmol. 2012 Dec 10:1-8. [Epub ahead of print]

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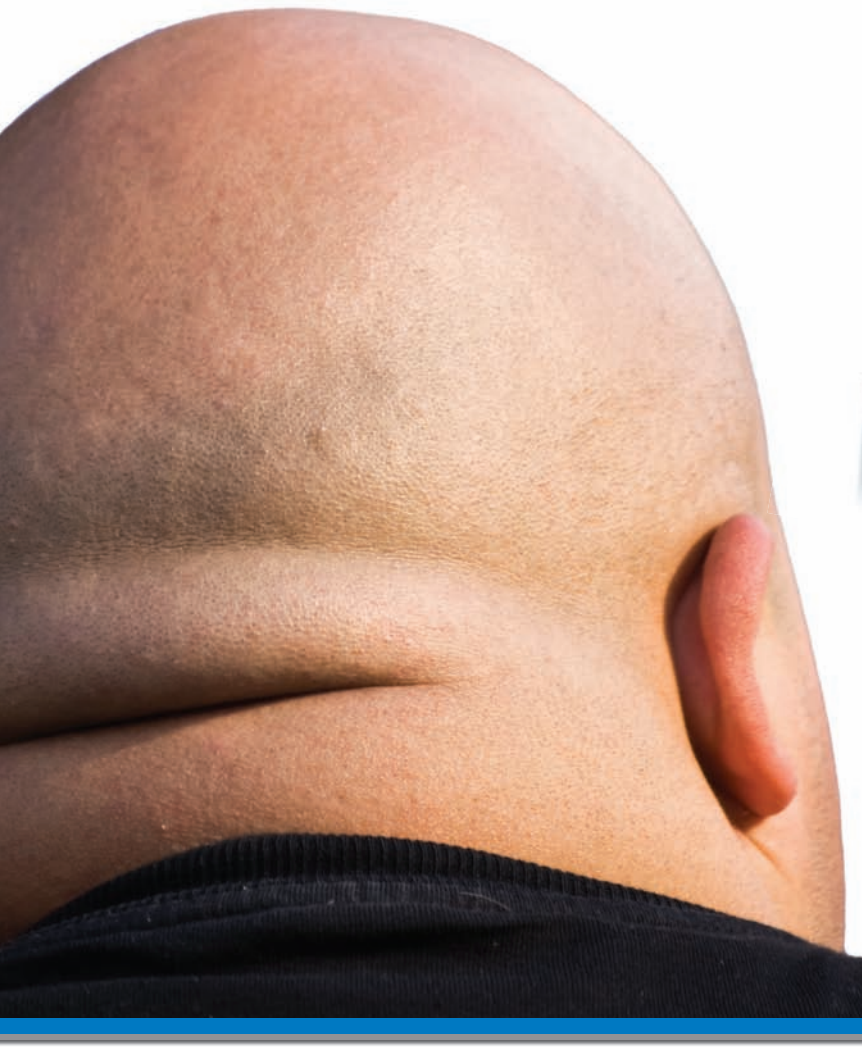

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Blind Mice See Again After Cell Replacement

Blind mice can see again, thanks to a stem cell rebuilding process developed by researchers at the University of Oxford in the UK.

Using a mouse model of severe human retinitis pigmentosa at a stage when no host rod cells remain, the researchers transplanted rod precursor stem cells to recreate a brand new outer nuclear layer. After just two weeks, the photoreceptor cells had been restored and the mice could see.

"We have shown the transplanted cells survive, they become light-sensitive, and they connect and reform the wiring to the rest of the retina to restore vision," says lead author Robert MacLaren, DPhil, FRCS, FRCOphth. ■

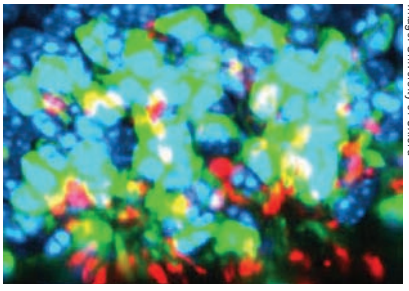

Image: University of Oxford

Re-formed outer retinal photoreceptor cells are shown in green.

Singh MS, Charbel Issa P, Butler R, et al. Reversal of end-stage retinal degeneration and restoration of visual function by photoreceptor transplantation. Proc Natl Acad Sci USA. 2013 Jan 3. [Epub ahead of print]

Biodegradable Disc Grows Stem Cells on Damaged Corneas

Researchers have developed a new kind of biodegradable disc that can grow stem cells on the eye to repair a damaged cornea.

Using a combination of techniques known as microstereolithography and electrospinning, engineers at the University of Sheffield in the UK were able to create a membrane of biodegradable material that is placed over the damaged cornea. Shaped as a disc, the membrane is then loaded with the patient's stem cells from the healthy eye, which then multiply and grow to form ocular tissue that will not be rejected.

Image: University of Sheffield

A biodegradable disc, loaded with a patient's own stem cells, can be used to repair damaged corneas.

The technique is ideal for use in developing countries, where corneal injuries are more common yet corneal grafting is not readily available. But it will be useful in developed countries, as well. "The current treatments for corneal blindness use donor tissue to deliver the cultured cells, which means that you need a tissue bank. But not everyone has access to banked tissues, and it is impossible to completely eliminate all risks of disease transmission with living human tissue," says study coauthor Frederik Claeyssens, PhD, of the Department of Materials and Engineering at the University of Sheffield. "By using a synthetic material [as a base], it will eliminate some of the risk to patients and be readily available for all surgeons. We also believe that the overall treatment using these discs will not only be better than current treatments, it will be cheaper as well."

Ortega I, Ryan AJ, Deshpande P, et al. Combined microfabrication and electrospinning to produce 3-D architectures for corneal repair. Acta Biomater. 2012 Nov 3. [Epub ahead of print]

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References: 1. Based on third-party industry report, Alcon data on file, Jan 2010-Sep 2011. **2.** Woods J, Woods C, Fonn D. Early symptomatic presbyopes—What correction modality works best? *Eye Contact Lens.* 2009;35(5):221-226. **3.** Rappon J. Center-near multifocal innovation: optical and material enhancements lead to more satisfied presbyopic patients. *Optom Vis Science.* 2009;86:E-abstract 095557. **4.** In a randomized, subject-masked clinical study at 20 sites with 252 patients; significance demonstrated at the 0.05 level; Alcon data on file, 2009. **5.** Rappon J, Bergenske P. AIR OPTIX® AQUA Multifocal contact lenses in practice. *Contact Lens Spectrum.* 2010;25(3):S7-S9.

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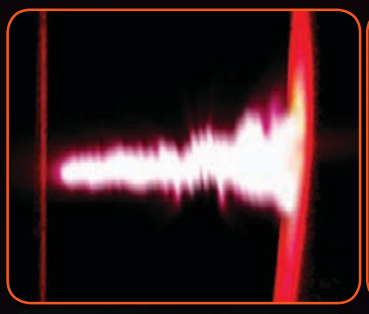
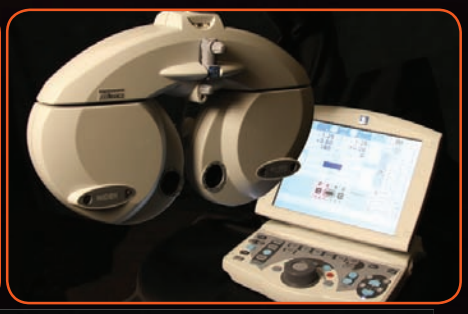


Graphic depiction of OPD-Scan III light transmission of 2520 real data points across a 9.5mm pupil



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	T. Sph	T. Coma	T. Tre	HO
Total:	0.020	0.040	0.025	0.059
Cornea:	0.061	0.108	0.073	0.155
Internal:	0.041	0.085	0.091	0.156

Refraction: VD = 13.75mm

	Sph	Cyl	Axis	RMS
WF@4.00	+1.00	-0.50	105	0.07D
WF@5.42	+0.75	-0.50	111	0.19D
Diff	-0.25	0.00	6	

When the OPD-Scan III report indicates 'WF', the patient will require only a basic refraction– saving 5-7 minutes per patient.

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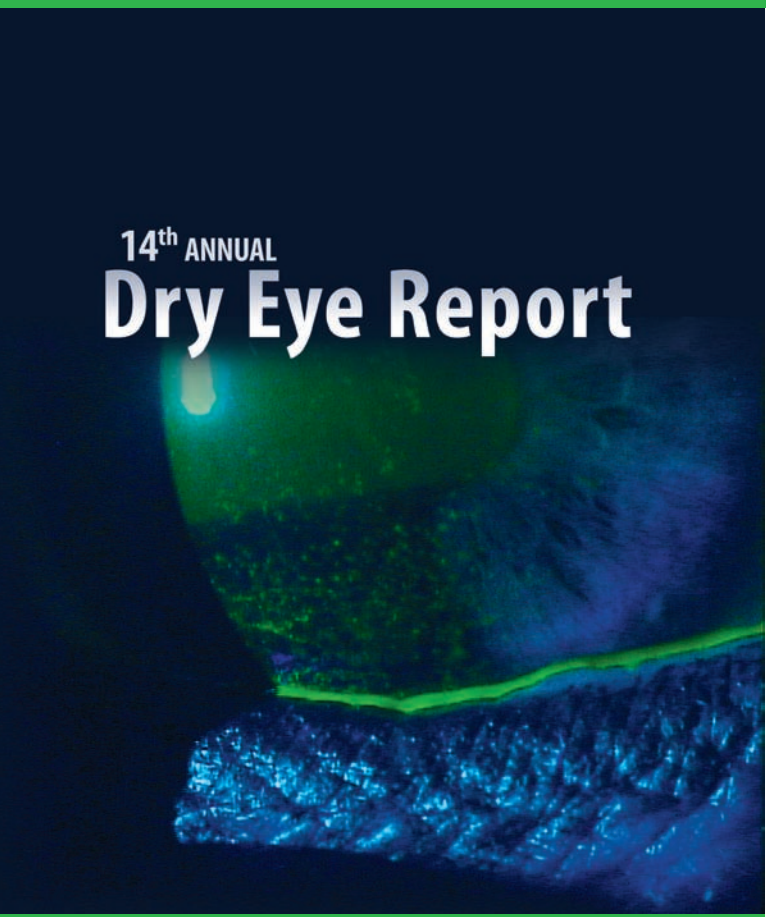

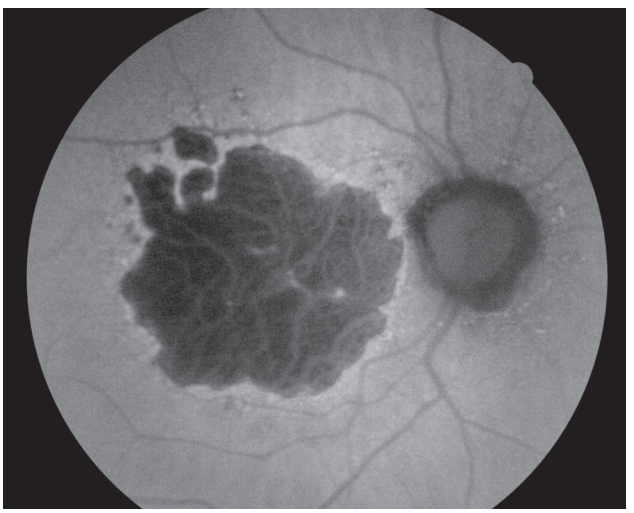

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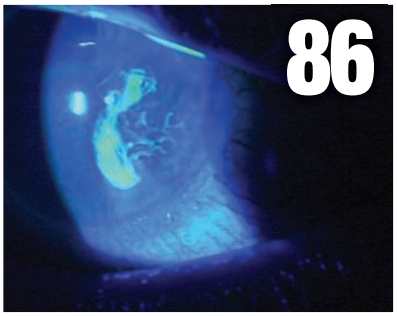

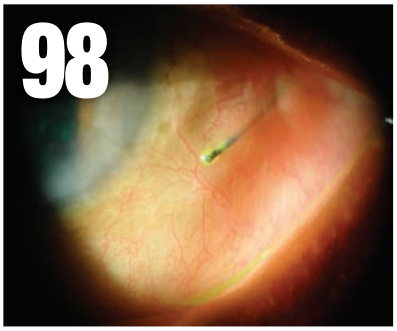

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All the World's a Stage

With privacy rights eroding, should optometrists worry that the Sunshine Act puts their business dealings in front of an audience? **By Jack Persico, Editor-in-Chief**

It seems oddly fitting that the day before Robert Bork died, so did a privacy law that he inspired. The controversial judge maintained that privacy rights only exist if they are expressly written into law. In a bit of two-can-play-at-that-game shenanigans, a reporter once accessed and published Bork's video rental history. The incident became part of his failed supreme court confirmation hearings, and inspired the Video Privacy Protection Act of 1988. That act was quietly killed in December at the request of Netflix, which wants the ability to share its users' viewing habits on Facebook. The next day, Bork passed away.

Wherever they come from, privacy rights ain't what they used to be. Also in December, photo-sharing website Instagram shot itself in the foot by changing the wording of its terms of service to assert its right to use the site's photos in conjunction with advertising, causing a hue and cry about the commercialization of our private lives. Even intellectual luminaries such as Kim Kardashian weighed in on that one. The irony is that Instagram's original terms of service already gave it that right; the company was merely trying to be more forthright about its intentions. Tech analysts think the backlash will cause Internet privacy contracts to become more, not less, opaque.

Google has also caused ill will with its callous disregard for the privacy of personal data—in 2009 its CEO said, "If you have something that you don't want anyone to know, maybe you shouldn't be doing it in the first place"—and Facebook practically brags about the company's life-in-a-fishbowl philosophy.

In this environment of ever-greater public access to our lives, it's not surprising that health care providers, including optometrists, will now find their business dealings with industry exposed to scrutiny. As Contributing Editor Jane Cole details this month (*see page 32*), a provision of the Affordable Care Act mandates public disclosure of any payment of $10 or more to any doctor from a medical drug or device company. The goal of the so-called Sunshine Act is to bring to light financial relationships between doctors and industry so that bias can be exposed.

Hold on a minute, detractors say. Since when does payment for services rendered constitute bias—especially among doctors who pledge to put the patient's interest before their own? Other professions more prone to influence peddling have no such requirement, so why single out doctors? To many, the Sunshine Act looks like a solution in search of a problem, and just one more regulatory albatross around the necks of industry (think of the reporting requirements and the tedious paper trail it will require).

Regardless, start preparing for life on stage. If you have concerns about misinterpration, disclose any financial relationships now and explain to patients or colleagues their origins and intent. As many doctors interviewed for our story mentioned, patients would likely see it as a *good* thing to be treated by a doctor who is courted by industry for product development and education. When your expertise is valued, don't be modest. If you've got it, flaunt it!

Hey, it seems to work for Kim Kardashian. If she can withstand public scrutiny, surely you can too. ■

Review Welcomes Additions to its Clinical Editorial Staff

To keep *Review of Optometry* in tune with the procedures and protocols that define cutting-edge optometric care, we rely on the guidance of many thought leaders in the field. I'm pleased to announce the addition of three prominent ODs whose voices will help to shape the publication's editorial content going forward.

- **Paul Karpecki, OD**, an expert in anterior segment care and a fixture at live education events, will expand his role by becoming Co-Chief Clinical Editor. Paul brings a wealth of practical expertise gleaned from his work at an MD/OD office in Kentucky, home of some of the most forward-thinking scope of practice laws.
- **Alan Kabat, OD**, comes on board as Associate Clinical Editor. Al's 20 years of experience in optometric education at Nova Southeastern will add a vital perspective—the essential scientific underpinnings of real-world clinical techniques.
- **Andrew Gurwood, OD**, of Salus University takes on the role of Case Reports Coordinator. Andy's intellectual rigor ensures that these contributions meet the exacting standards of case-based education and augment our scientific literature.

Monthly Multifocal Pearl

Multifocal Contact Lenses: Keeping Your Focus on This Profit Center

By John Rumpakis, OD, MBA

Today's ophthalmic practices face many challenges: a stagnant economy, diminishing reimbursements from refractive insurance carriers, reduced access to patients and a high level of reticence from consumers willing to spend money where the benefit of the purchase isn't readily identifiable. To many, it sounds pretty bleak; to others, it represents a huge opportunity to recognize population trends, capitalize on new technology and create a market for clinical excellence within their practices.

The opportunity at hand here is in multifocal contact lenses—the latest frontier in contact lens technology and performance. It's important for practitioners to be comfortable with different presbyopic contact lens designs, but they must also realize that not all multifocal lenses are created equal.

THE PRESBYOPIC MODEL

The global population of contact lens wearers is significant, as is the number of presbyopes who require vision correction (see table).[1] Presbyopes, who are prevalent in most optometric practices, have increased near tasks such as computers and cell phones. They want a high-performing, well-designed lens.

When a successful contact lens patient begins to experience the effects of presbyopia, they may think they no longer have any choices to continue to be "spectacle free." Those who discontinue contact lens wear can have a considerable economic impact on the bottom line of an eye care practice. **A single patient who drops out of lens wear represents up to $24,000 of lost revenue over their lifetime.**[2] One study showed that AIR OPTIX® AQUA Multifocal contact lenses ranked as "highest performer" in subjective real-world situations when compared to monovision.[3] Therefore, establishing a multifocal center of excellence within your practice can set you apart from others in the optometric community, and instill greater loyalty to your practice.

PRESBYOPIC ANALYSIS[1]

Total U.S. Population: 310,000,000

Population Requiring Vision Correction
- At ages 45–49: 15,510,604
- At ages 50–54: 17,822,556
- At ages 55–59: 16,609,549

Population Wearing Contact Lenses
- At ages 45–49: 3,323,701
- At ages 50–54: 2,707,224
- At ages 55–59: 1,822,999

Population of Opportunity
- At ages 45–49: 12,186,903
- At ages 50–54: 15,115,333
- At ages 55–59: 14,786,550

SEIZE THE OPPORTUNITY

Presbyopes are rarely targeted and cultivated for contact lenses. Yet, they are a ripe target segment for emerging technology such as AIR OPTIX® AQUA Multifocal contact lenses, which were preferred by 90% of practitioners participating in a clinical evaluation.[4] **Providing a patient with an annual supply of contact lenses reduces the chance of them seeking out alternative suppliers while at the same time maximizing your revenue.** Take it one step further and offer online ordering through your practice's website.

Multifocal spectacles are also important in the care of the patient; however they are not mutually exclusive with multifocal contact lenses. While high-end glasses are profitable in the short term, patients may be hesitant to upgrade again for a few years because of the high initial out-of-pocket cost. Now consider the fact that multifocal lenses such as AIR OPTIX® AQUA Multifocal contact lenses can provide a steady yearly income from the same patient.

So, how can you program success into your practice with multifocal lenses? Communication with patients is essential to achieving success. Present a balanced view to each individual and set realistic expectations for him or her. The goal is to reduce dependency on—not eliminate the need for—glasses, which keeps the door open for other products you prescribe. When discussing multifocal contact lenses with your presbyopic patients, make sure they're willing to accept the occasional need to use reading glasses for fine visual tasks. If your staff is educated on the auxiliary visual needs of these patients, this is an excellent opportunity for them—or you—to suggest a second/back-up pair of spectacles and/or a pair of non-prescription sunglasses. It's also helpful to emphasize to patients what they can see vs. what they can't. Finally, keep explanations simple and pertinent to each patient's needs in terms of benefits that they can expect.

PRINCIPLES BY WHICH TO PRACTICE

When you see contact lens patients—particularly those who are multifocal candidates—prescribe with confidence. Make product-specific recommendations based on their lifestyle needs, recommend the appropriate lens care (such as OPTI-FREE® PureMoist® MPDS) with the lenses prescribed. Try to make things convenient for patients by providing an annual supply of lenses. If you create an integrated solution for your patients, you will truly have made not only an impact on their lives, but on the bottom line of your practice as well.

Dr. Rumpakis is the president and CEO of PRMI, a management and consulting firm serving the medical industry. He lectures nationally and internationally on the economics of clinical standards of care, medical coding and compliance, practice appraisal and other practice management topics.

1. U.S. Census Bureau, Population Division. Table 1. Annual estimates of the resident population by sex and five-year age group for the United States: April 1, 2010 to July 1, 2011 (NC-EST2011-01). Available at: http://www.census.gov/popest/data/national/asrh/2011/tables/NC-EST2011-01.xls. Accessed August 2012.
2. Rumpakis J. New data on contact lens dropouts: an international perspective. Rev Optom. 2010;37–42.
3. Woods J, Woods CA, Fonn D. Early symptomatic presbyopes – what correction modality works best?" Eye Contact Lens. 2009;5: 221–226.
4. Rappon J, Bergenske P. AIR OPTIX AQUA Multifocal contact lenses in practice. Contact Lens Spectrum. 2010;25(3):S7-9.

My Old School

Are you an Old School OD or a New School OD? If you recognize the name of the Steely Dan song in the headline, you're Old School, pal. **By Montgomery Vickers, OD**

My mirror reminds me often that there is a changing of the guard in optometry. This is an inevitable reality in all of life's journeys—but, for some reason, it still manages to surprise especially the newest members of the "Old School," e.g., me.

The "New School" members don't really have time to ponder such things. They barely have time to take care of many critically important issues, such as tee times and which cell phone plan is more important than putting food on the table.

But is there really a difference between Old School and New School in the world of eye care? After all, are we not held to the same standard ethically, medically and legally? We should all be sort of the same, right? Well, yes, except for the obvious, which includes the number of colonoscopies and AARP memberships.

My crack team of investigators (I have to spend my huge *Review of Optometry* budget on something) has turned up some very interesting differences between Old School and New School that you need to know:

Old School	New School
• Direct ophthalmoscopy	• OCT
• Fundus photography	• OCT
• Pay for wife's blepharoplasty	• Pay for OCT
• PD ruler	• "Won't OCT do that?"
• Calls receptionist "Honey"	• Watches "Honey Boo-Boo"
• White lab coat	• White iPhone
• Facial lesion on lid	• Facetime on iPad
• Cutting carbs	• Cutting cards
• Seasoned	• Half baked
• Adjusting nosepads	• Adjusting iPads
• Funny looking disc	• ONH cupping

New School	Old School
• "I see cataracts."	• "I have cataracts."
• Accepts vision plans for patients	• Creates vision plans for patients
• "We'll have to run some tests."	• "I have to have some tests."
• Antibiotics	• Hot compresses
• PALs needed	• Pal needs knee replacement
• Casual Fridays	• Why work Fridays?
• Strollers	• Walkers
• Need to use YAG laser	• Need to have YAG laser
• Need great website	• Hope wife doesn't find out I looked at a great website.
• "Your child should have an eye exam."	• "Your child's child's child should have an eye exam."
• Gangnam style	• Resected bowel

So, New School doctors, here's a little advice:

1. Hang around Old School doctors. You'll be amazed what you didn't learn in school.

2. Always remember that anyone who has shoes older than you are probably deserves some respect.

And, Old Schoolers?

1. Yes, the New Schoolers are smarter than you. Don't hate them... Refer to them.

2. And, yes, the New Schoolers can be really dumb sometimes, but not as dumb as you were 30 years ago, I promise.

Just remember: Old or New, we're all together in the same School. ■

MYTHS, METHODS AND MEANS FOR SOOTHING END-OF-DAY CONTACT LENS DISCOMFORT

Fig. 1: Headstand in an ice bucket.

Fig. 2: Switch to Avaira®.

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*U.S. Study. Data on file.

T Minus 12 and Counting…

The Affordable Care Act goes into full effect in 12 months. But, CMS is not waiting to implement significant changes! **By John Rumpakis, OD, MBA, Clinical Coding Editor**

It's January 2013 already. Twelve months and counting until major provisions of the Affordable Care Act (ACA) go into place. To many, January 2014 seems like it's far away.

Well, guess what? It's virtually here. There are so many initiatives that are critical to the average practitioner, I can't even list them all here. Suffice it to say that it is the purview of this column and opinion of this author that the average OD's knowledge and preparation for this event is woefully inadequate.

Be aware that many aspects of the ACA will be implemented over time *before* the first day of 2014. So, let's discuss an example that hits us this month, January 2013—the MPPR, or the Multiple Procedure Payment Reduction.[1]

What is the MPPR? It's a new Medicare payment reduction that applies when multiple services are furnished to the same patient on the same day.

The Affordable Care Act specifies that Health and Human Services shall identify potentially misvalued codes. To do so, HHS will look at multiple codes that are frequently billed in conjunction with furnishing a single service. As a further step in implementing this provision, Medicare is expanding the MPPR policy by applying MPPRs to the Technical Component (TC) of diagnostic ophthalmology procedures.

The MPPRs on diagnostic ophthalmology procedures apply when multiple services are furnished to the same patient on the same day. The MPPRs apply to TC-only services and to the TC of global services. (The MPPRs do not apply to professional component services.)

In short, CMS will make a full payment for the TC of the highest priced procedure, but will pay 80% of the TC for subsequent services provided by the same physician (or by multiple physicians in the same group practice) to the same patient on the same day.

Here's an example: Let's say that a patient came in for a glaucoma work-up and the tests that you want to do on this date of service are visual fields, fundus photography and pachymetry. Below is what your reimbursement would look like both before and after the ACA MPPR is put in place. (Note: the Reimbursement Values are based on 2012 CMS National Averages.[2])

As you can see, the reduction affects all procedures performed after the one with the highest payment. (A list of all procedures subject to the MPPR is at www.cms.gov/Regulations-and-Guidance/Guidance/Transmittals/Downloads/R1149OTN.pdf.) When these payments are reduced, they will be reflected on your Explanation of Benefits with a Claim Adjustment Reason Code of 59.

Furthermore, the 2013 Physician Fee Schedule Final Rule indicated that CMS will monitor these tests to identify inappropriate changes in timing of the delivery of these diagnostic tests. In other words, if physicians start changing their practice and billing patterns to avoid the reductions, they will most likely be identified as an outlier—which could result in an audit.

It may be T minus 12 and counting until full implementation of the Affordable Care Act; however, it's clear that no one at the government level is waiting until 2014 to put these changes into place. Certainly, we're just beginning to see the far-reaching impact of this law. It will impact our practices now and will continue to do so in the future. So, it is important for all to be prepared and aware how it will affect the delivery of care to our patients. ■

Reimbursement Before and After MPPR

	CPT code 92083	CPT code 92250	CPT code 76514	Before MPPR	After MPPR
Professional Component (-26)	$27.57	$23.15	$9.53	$60.25	$60.25
Technical Component (-TC)	$61.95	$53.44*	$5.11*	$120.50	$108.79
Total	$89.52	$76.59	$14.64	$180.75	$169.04
			Total reduction in this example		6%

** Red indicates the two technical components subject to reduction.*

1. Centers for Medicare & Medicaid Services. Multiple Procedure Payment Reduction (MPPR) on the Technical Component (TC) of Diagnostic Cardiovascular and Ophthalmology Procedures. Available at: www.cms.gov/Regulations-and-Guidance/Guidance/Transmittals/Downloads/R1149OTN.pdf. Accessed December 22, 2012.
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Optimizing Visual Performance with Wavefront Refractions

A study of the use of modern technology to improve patient satisfaction.

By Kevin Reeder, OD, Earl Sandler, OD, Joel Cook, OD, and Lynette Potgieter, B. Optom (RSA)

The clinical methods for measurement of the manifest refraction of patients have remained unchanged for more than 100 years. At the same time, we have learned that the measurements vary between practitioners and, more importantly, the variation is higher for individual patients.[1] Zadnik concluded that the repeatability of subjective refractions was worse than that of autorefractions, and found to be + 0.63 diopter.[2]

The coefficient of variation between measurements may be based on the subjective nature of the test and the common use of 0.25-diopter steps in sphere and cylinder measurements. The use of the Jackson Cross Cylinder has been questioned for its variable effectiveness as a function of the axis being measured.[3] In addition, we have concerns about the clinical limitation of conducting a subjective refraction under a single light level and pupil size.

Dr. Cook graduated from the University of Houston's College of Optometry and has practiced in San Diego since 1977. While a student, he was awarded a Bausch + Lomb research fellowship in contact lenses. He served as an optometrist with the Navy in San Diego and is a Past President of the San Diego County Optometric Society.

Dr. Reeder is a 1988 graduate of University of California, Berkeley, and is a partner in the Carmel Mountain Vision Care Center, a four doctor multi-specialty practice in San Diego. He has been a clinical investigator for numerous companies in the vision care industry. He specializes in contact lenses, refractive therapy and low vision.

Dr. Sandler commenced his optometric training in South Africa, where he received a Bachelor Degree in Optometry and continued his training at the New England College of Optometry in Boston where he received a Doctorate of Optometry in 1996. His areas of interest include specialty contact lenses and laser vision correction.

Dr. Potgieter graduated with a Bachelor Degree in Optometry from the University of Johannesburg, South Africa in 1996. After practicing in South Africa, she worked in various roles for spectacle and contact lens companies in England, Australia and the United States.

We have experienced patients reporting that they see well with our prescription lenses during the day and less well when driving at night or attempting tasks in reduced illumination. The understanding that the spherical and cylindrical refractive error varies with pupil size has been expanded by the ability to measure the refractive error objectively with autorefractors and wavefront aberrometers using a range of aperture sizes.[4]

As clinicians having a practice philosophy of providing the highest quality of care possible and striving for the highest level of patient satisfaction and enthusiasm, we wondered if we could improve our standard method for determining a final lens prescription. The principles of evidence-based health care and our desire to be ahead of the curve presented a need for discovery. To close this gap, we decided to investigate a new technology that presented the potential to provide a prescription that would optimize visual performance under the full continuum of lighting conditions.

The instrument we investigated is the i.ProfilerPlus® (Carl Zeiss Vision) and the resultant i.Scription® lenses (Figures 1 and 2). The technology uses an algorithm that blends our manifest refraction with the wavefront data. The manifest refraction is entered into the i.Scription Software, which then calculates the i.Scription prescription up to a pupil size of 5.5mm.[5] The resultant i.Scription is ordered to the one hundredth of a diopter (0.01D) as opposed to the traditional increments of one quarter of a diopter (0.25D). i.Scription uses sphere and cylindrical equivalents for the pupil range of the respective eye and the higher-order aberrations.

We wanted to concentrate on pre-presbyopic patients who were expected to have the largest range of pupil reactivity when measured under mesopic and photopic illumination. The decision to study pre-presbyopic patients also allowed for testing single vision lenses rather than multifocal lenses. This eliminated the variables of style of progressive addition lenses, precision fitting of the PALs and individual adaptation variability.

Sponsored by Carl Zeiss Vision

OUR STUDY DESIGN

We have served as clinical investigators on scores of contact lens and pharmaceutical studies and knew we could execute a study to determine if new technology was right for our practice. We wished to avoid the pitfall of very small experiences, and we wanted to see if there was an indication that we could do a better job. We used a design similar to contact lens studies we had conducted. A randomized, single-masked, cross-over clinical study comparing the efficacy of customized ZEISS Individual® Single Vision (SV) lenses with i.Scription to customized ZEISS Individual® SV lenses without i.Scription was selected for our practice, Carmel Mountain Vision Care in San Diego. A direct comparison of the visual quality of 37 subjects was made between the two single vision lenses, under mesopic and photopic conditions.

Each subject was measured with the i.ProfilerPlus® and received a comprehensive eye examination, including our customary subjective manifest refraction. Objective measurements were taken of the wavefront aberrations of the eye with the i.ProfilerPlus and combined with our subjective manifest refractions by use of the proprietary Zeiss VoluMetric merit function algorithm to create customized ZEISS Individual® SV lenses with i.Scription (Figure 3). A second pair of customized ZEISS Individual SV lenses without i.Scription, using only the conventional subjective refraction, were manufactured as the control lenses.

The control and test lenses were fitted into two identical frames, with the same position of wear, measured with the i.Terminal® by ZEISS. A refractive index of 1.6 was used, as the lens material and lenses were manufactured according to the standard customization parameters of ZEISS Individual SV lenses. The study spectacles were randomized for wearing order and labeled to mask the type. The subjects were compensated at their final visit with the study pair that they felt gave them the best overall visual quality and comfort.

SUBJECT SELECTION

Our eligibility criteria for subjects required normal, healthy eyes; patient age between 18-40 years; best corrected monocular distance visual acuity (logMAR) of 0.10 (20/25) or better; and subjects who were full-time eyeglass wearers for all distances.

Subjects were classified according to three different prescription power categories. The classification was made according to the eye with the highest Manifest Refraction Spherical Equivalent (MRSE). Subjects with an MRSE greater than or equal to -4.00D were classified as *Subjects with High Myopia*; between -3.75D and -0.25D inclusive, as *Subjects with Mid-Myopia* and more plus or equal to plano, as *Subjects with Hyperopia*. The study concluded with 11 subjects with high myopia; 21 subjects with mid-myopia and five subjects with hyperopia.

PATIENTS SHARE THEIR IMPRESSIONS

"Both pairs were very close, but the 1st pair (i.Scription) was a lot better at night."

"Slightly better vision: on computer; viewing screen during presentations; sharpness of lights at night."

"Better night time conditions, overall slightly better."

"1st pair (i.Scription) seemed clearer and overall more comfortable for my eyes."

"It (i.Scription) is more comfortable than the other, less strain. They also gave me sharper view of things and brightened my surroundings."

"I prefer the 2nd pair (i.Scription) because my eyes didn't feel any straining, everything seemed clearer."

"The 1st pair (i.Scription) is sharper and clearer and I feel more confident with them."

CLINICAL MEASUREMENTS

Clinical measurements were conducted under mesopic (30 lux) and photopic (300 lux) conditions. The i.ProfilerPlus measurement, Subjective Refraction and Point-Spread-Function (PSF) test were performed under mesopic conditions. The PSF test was designed with a single green LED light source, the same size as a -0.1 LogMAR letter, that was mounted against a matte black background and placed at the same distance from the patient as the visual acuity chart.

Monocular and binocular high (100%) and low (10%) contrast distance visual acuities were measured in a straight-ahead gaze position with the M&S Smart System II 20/20™ 2010. Monocular and binocular high-contrast near visual acuities were measured in a straight ahead gaze position, with ZEISS near VA chart. Independent mesopic and photopic pupil sizes were measured with a Colvard Pupilometer.

Control and Test eyeglasses were worn independently for a 10-day period each, followed by a one-week direct comparison period. Clinical measurement evaluations and patient-reported outcomes were performed on days 10, 20 and 27, respectively. Results were analysed using the following methods: Percentage Analysis; Descriptive Statistical Analysis; P-Test Analysis; Bland-Altman Plots and ANOVA calculations. A Visual Analog Scale response valuation was used in all questionnaires (Figure 4).

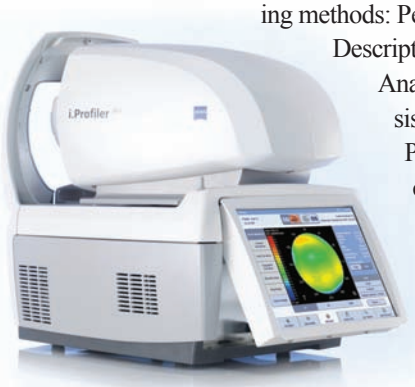

Figure 1. The compact, space-saving i.ProfilerPlus.

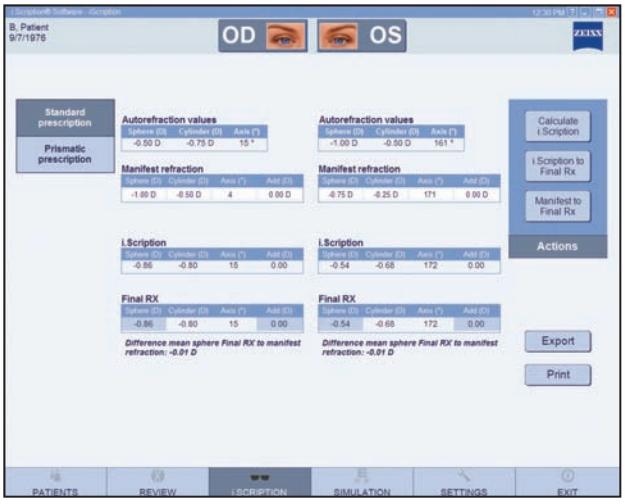

Figure 2. The i.Scription software user interface entry screen.

Subjects were required to fill out a daily study journal commenting on their visual experiences under specific visual conditions that were outlined in the journal.

RESULTS

• ***Comparison to Habitual Rx.*** We asked the subjects to compare the two pairs of study eyewear to eyewear with their habitual prescription prior to the study. Subjects were asked to rate the i.Scription Rx and manifest refraction Rx to their habitual Rx after they wore each pair independently for a minimum of 10 days. The overall preference for the i.Scription Rx vs. the Habitual Rx was 95%. The overall preference for the Manifest Rx vs. the Habitual Rx was 92%. This outcome indicates that both study prescriptions were preferred over the habitual Rx and there was an apparent need for prescription change.

• ***Overall Preference Final Choice.*** Subjects were asked which study spectacles they preferred overall as their final choice for visual quality and visual comfort, and to rate that choice. Answers were recorded on a 1 to 6 numerical scale and analyzed. A mean difference of 0.514 higher for the Test lenses resulted. Percentage analysis revealed that 59.5% of subjects preferred the Test lenses as their final choice for visual quality and visual comfort, compared to the Control. This difference was driven mainly by the subjects with high myopia and mid-myopia: 67% of subjects with high myopia and 59% of subjects with mid-myopia preferred the Test lenses and subjects with Hyperopia had an equal preference for the Test and Control lenses.

• ***Visual Acuity.*** The Test lenses provided better mean visual acuity in mesopic conditions when compared to the Control lenses. All visual acuity measurement conditions resulted in no statistically significant mean logMAR acuity differences with the Test and Control lenses. In each case, the difference was less than one line of vision improvement. A difference of one line or more is required to conclude that the difference is clinically significant.[5]

• ***Preference Under Lighting Conditions.*** Our subjects wore each pair solely for 10 days, and then were allowed to make a direct comparison with both study Test and Control spectacles for one week prior to their final visit where clinical measurements were also conducted with direct comparison. The comparison is reported in Figure 4.

Overall, the Test lenses were preferred for five of the seven visual conditions; for one visual condition the Test lenses were preferred equally to the Control and for the other visual condition the Control lenses were preferred over the Test.

• ***Adaptation Time.*** Our subjects were asked how quickly they adapted to the study spectacles after having worn each pair independently for a minimum of 10 days. Answers were recorded on a 1 to 5 numerical scale and analyzed. A mean difference of 0.027 resulted, indicating no significant difference and that the Test rated slightly higher than the Control. There were no cases of non-adaptation for either the Test or Control.

• ***Visual Conditions Questionnaire.*** Our subjects were asked which study spectacles they preferred overall for 17 different visual conditions (Figure 5). The Test lenses rated higher than the Control lenses for all 17 different visual conditions: distance vision; mid-range vision; near vision; active vision; brightness; brightness of environment; colors more vivid; edges sharper; less glare; peripheral vision; depth perception; adaptation; visual comfort for distance vision; visual comfort for near vision; quicker to change focus; night vision; natural vision.

They were asked which statement(s), out of eight, best described their study spectacles. They were allowed to select either or both study spectacles for each statement when applicable. The Test lenses rated higher than the Control for all eight statements as follows: Provides more comfortable vision; provides fewer headaches; provides good near vision; provides good intermediate vision; provides good distance vision; provides a feeling of more relaxed vision; provides less tiredness; provides less strain.

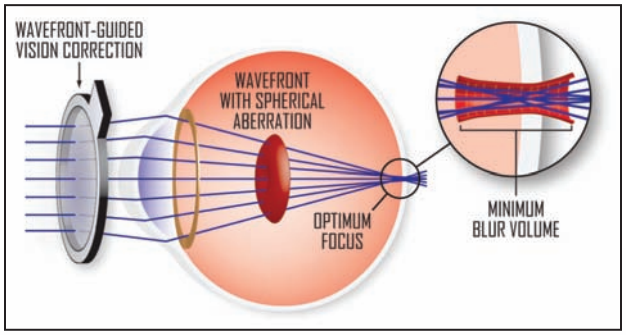

Figure 3. Diagrammatic explanation of the Volumetric Merit Function.

FIGURE 4

Preference Category Description	Test: iProfiler	Control: Manifest	No Difference
Distance visual acuity OU, under mesopic high contrast conditions	51%	41%	8%
Distance visual acuity OU, under mesopic low contrast conditions	38%	24%	38%
Near visual acuity OU, under mesopic conditions	22%	19%	59%
Point Spread Function Test under mesopic conditions OU	35%	27%	38%
Distance visual acuity OU, under photopic high contrast conditions	41%	35%	24%
Distance visual acuity OU, under photopic low contrast conditions	22%	22%	57%
Near visual acuity OU, under photopic conditions	16%	24%	59%

• ***Likelihood to recommend lenses.*** Our subjects were asked to rate on a scale from 1 (very unlikely) to 10 (very likely) how likely they would recommend the study spectacles to family or friends. A mean difference of 0.135 resulted, which indicates that the Test lenses rated higher than the Control. Percentage analysis of the number subjects who gave the highest rating (10) revealed that 41% of subjects would "very likely" recommend the Test lenses and 27% of subjects would "very likely" recommend the Control.

OUR CONCLUSIONS

As clinicians and practice managers, we try to make quantitative decisions for constant product and service improvements to meet our mission of providing the highest quality of care in our region. While we appreciate that our in-office studies may not have statistical power for 95% confidence, we endeavour to execute our studies to allow us to have more than anecdotal evidence.

In this study, we found that the blended or optimized prescription of the iScription lenses prevailed in every category over the same Zeiss Individual lenses made according to our subjective refractions only. The overall preference rating was higher with the Test spectacles than with the Control spectacles. This difference was driven mainly by the subjects with high myopia and mid-myopia.

While not statistically or clinically significant, the preponderance of the evidence supports a trend for enhanced performance and patient satisfaction with the Test lenses. The finding that the visual acuity was better for the Test lenses under mesopic lighting conditions was consistent with a clear area where we wanted to improve our patients' visual performance. The preference for the Test lenses for the Point Spread Function test also supported our desire to improve visual comfort in the presence of point sources of light under dim light conditions.

Adaptation time was faster with the Test spectacles than with the Control spectacles. There were no cases of non-adaptation for either the Test or Control spectacles.

We were impressed that the Subjective ratings were higher with the Test spectacles than with the Control spectacles for all 17 visual conditions. This complements the discovery that the Test spectacles were more likely to be recommended than the Control spectacles.

Under direct comparison, the Test spectacles were preferred for visual quality over the Control spectacles under five of seven lighting and contrast acuity conditions. Further, the Test spectacles provided more comfortable vision; fewer headaches; good near vision; good intermediate vision; good distance vision; a feeling of more relaxed vision; less tiredness and less strain.

Overall, our consideration of all the investigational categories supports a trend that i.Scription by ZEISS provides better visual quality and comfort for our patients, and our patients are more likely to recommend these lenses; thereby adding to the growth of our practice. We expect the use of this technology, combined with our other study-based decisions, will continue to support our valuable final product for the practice: *enthusiastic, satisfied patients*.

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FIGURE 5

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The study of lens performance under varying light conditions

EXIT QUESTIONNAIRE – VISIT FIVE

[1] Which pair of eyeglasses did you prefer for distance vision (e.g. straight-ahead viewing; day-time driving)?

1st Pair Much Better	1st Pair Somewhat Better	1st Pair Slightly Better	2nd Pair Slightly Better	2nd Pair Somewhat Better	2nd Pair Much Better

[2] Which pair of eyeglasses did you prefer for mid-range vision (e.g., using a computer)?

1st Pair Much Better	1st Pair Somewhat Better	1st Pair Slightly Better	2nd Pair Slightly Better	2nd Pair Somewhat Better	2nd Pair Much Better

[3] Which pair of eyeglasses did you prefer for near vision (e.g. reading a book)?

1st Pair Much Better	1st Pair Somewhat Better	1st Pair Slightly Better	2nd Pair Slightly Better	2nd Pair Somewhat Better	2nd Pair Much Better

[4] Which pair of eyeglasses did you prefer for active vision (e.g. walking; going up and down stairs)?

1st Pair Much Better	1st Pair Somewhat Better	1st Pair Slightly Better	2nd Pair Slightly Better	2nd Pair Somewhat Better	2nd Pair Much Better

[5] With which pair of eyeglasses does your environment look brighter?

1st Pair Much Brighter	1st Pair Somewhat Brighter	1st Pair Slightly Brighter	2nd Pair Slightly Brighter	2nd Pair Somewhat Brighter	2nd Pair Much Brighter

[6] Which pair of eyeglasses did you prefer in terms of the brightness of your environment?

1st Pair Much Better	1st Pair Somewhat Better	1st Pair Slightly Better	2nd Pair Slightly Better	2nd Pair Somewhat Better	2nd Pair Much Better

[7] With which pair of eyeglasses do colors appear more vivid?

1st Pair Much More Vivid	1st Pair Somewhat More Vivid	1st Pair Slightly More Vivid	2nd Pair Slightly More Vivid	2nd Pair Somewhat More Vivid	2nd Pair Much More Vivid

[8] With which pair of eyeglasses do edges of objects look sharper?

1st Pair Much Sharper	1st Pair Somewhat Sharper	1st Pair Slightly Sharper	2nd Pair Slightly Sharper	2nd Pair Somewhat Sharper	2nd Pair Much Sharper

[9] With which pair of eyeglasses do you experience less glare (e.g. driving at night with oncoming headlights)

1st Pair Much Less Glare	1st Pair Somewhat Less Glare	1st Pair Slightly Less Glare	2nd Pair Slightly Less Glare	2nd Pair Somewhat Less Glare	2nd Pair Much Less Glare

[10] With which pair of eyeglasses do you have Brighter peripheral vision (e.g. moving your eyes to look at an object off to the side)?

1st Pair Much Brighter	1st Pair Somewhat Brighter	1st Pair Slightly Brighter	2nd Pair Slightly Brighter	2nd Pair Somewhat Brighter	2nd Pair Much Brighter

[11] With which pair of eyeglasses do you have Brighter depth perception (e.g. judging distances)?

1st Pair Much Brighter	1st Pair Somewhat Brighter	1st Pair Slightly Brighter	2nd Pair Slightly Brighter	2nd Pair Somewhat Brighter	2nd Pair Much Brighter

[12] Which pair of eyeglasses did you find it easier to adapt to wearing?

1st Pair Much Easier	1st Pair Somewhat Easier	1st Pair Slightly Easier	2nd Pair Slightly Easier	2nd Pair Somewhat Easier	2nd Pair Much Easier

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[13] With which pair of eyeglasses did you have less eye strain, fatigue, dizziness or other feelings of visual discomfort during general, straight-ahead day-time viewing?

1st Pair Much Less Discomfort	1st Pair Somewhat Less Discomfort	1st Pair Slightly Less Discomfort	2nd Pair Slightly Less Discomfort	2nd Pair Somewhat Less Discomfort	2nd Pair Much Less Discomfort

[14] With which pair of eyeglasses did you have less eye strain, fatigue, dizziness or other feelings of visual discomfort during near work?

1st Pair Much Less Discomfort	1st Pair Somewhat Less Discomfort	1st Pair Slightly Less Discomfort	2nd Pair Slightly Less Discomfort	2nd Pair Somewhat Less Discomfort	2nd Pair Much Less Discomfort

[15] With which pair of eyeglasses do you find it quicker to change your focus to a nearby object

1st Pair Much Much Quicker	1st Pair Somewhat Much Quicker	1st Pair Slightly Much Quicker	2nd Pair Slightly Much Quicker	2nd Pair Somewhat Much Quicker	2nd Pair Much Much Quicker

[16] Which pair of eyeglasses did you prefer for night vision or viewing in dim light?

1st Pair Much Better	1st Pair Somewhat Better	1st Pair Slightly Better	2nd Pair Slightly Better	2nd Pair Somewhat Better	2nd Pair Much Better

[17] With which pair of eyeglasses does the view of your surroundings feel more natural?

1st Pair Much Better	1st Pair Somewhat Better	1st Pair Slightly Better	2nd Pair Slightly Better	2nd Pair Somewhat Better	2nd Pair Much Better

[18] With your FIRST pair of eyeglasses, did you notice any unnatural magnification, where objects appear too big or too small, or appear closer or farther away than normal?

Yes, I noticed this very strongly | Yes, I noticed this a little bit | No, I didn't notice this

[19] With your SECOND pair of eyeglasses, did you notice any unnatural magnification, where objects appear too big or too small, or appear closer or farther away than normal?

Yes, I noticed this very strongly | Yes, I noticed this a little bit | No, I didn't notice this

[20] Which pair of eyeglasses did you prefer overall?

1st Pair Much Better	1st Pair Somewhat Better	1st Pair Slightly Better	2nd Pair Slightly Better	2nd Pair Somewhat Better	2nd Pair Much Better

What are your reasons for this choice? What are your reasons for disliking the other pair?

[21] On a scale of 1 to 10, how likely would you be to recommend your FIRST pair of eyeglasses to family or friends?

1 (Very UNLIKELY) 2 3 4 5 6 7 8 9 10 (Very LIKELY)

[22] On a scale of 1 to 10, how likely would you be to recommend your SECOND pair of eyeglasses to family or friends?

1 (Very UNLIKELY) 2 3 4 5 6 7 8 9 10 (Very LIKELY)

[23] Select which of the following best describe your FIRST and SECOND pair of eyeglasses. You are allowed to select more than one option per pair of eyeglasses.

	1st Pair	2nd Pair
Provides more COMFORTABLE vision overall		
Makes my eyes feel LESS TIRED overall		
Makes my eyes feel LESS STRAINED overall		
Experience LESS HEADACHES overall		
Provides good NEAR vision e.g. when reading		
Provides good INTERMEDIATE vision e.g. on the computer		
Provides good DISTANCE vision e.g. when driving		
Makes my eyes feel more RELAXED overall		

Will the Sunshine Act Shine a Bright Light or Cast a Dim Shadow?

From invasion-of-privacy issues to the benefits of greater transparency, sweeping changes may result from this new federal regulation. **By Jane Cole, Contributing Editor**

These days, financial disclosures are a given. If you've sat in enough lecture halls or read a doctor-penned—but industry-sponsored—CE course, you are well aware if the presenting doctor has an affiliation with a specific company, or more typically, companies. But now, with the so-called "Sunshine Act" ready to take effect, transparency between physicians and industry is about to be raised to a whole new level—welcome news in some ways, but a boondoggle for industry. Under this new federal mandate, manufacturers are required to annually report any gifts or compensation to doctors, including honoraria, food, travel and research dollars, to the Centers for Medicare & Medicaid Services. CMS will then post this information publicly on the web.

What does this mean for optometry? If you're a doctor who has received compensation (monetarily or otherwise) from industry, the nature and the specific dollar value of that transaction—with your name attached to it—will be readily available to the public if they choose to look.

"This new regulation is yet another assault on the esteemed place health care providers once enjoyed within society," says optometrist Art Epstein of Phoenix. "I don't know any other profession where people have to disclose income at this level. This is especially odd for health care professionals, where trust between patient and practitioner is a fundamental principle as well as a sworn oath."

"It is somewhat of an insult that they would think that I would prescribe a certain drug or medical device based on a gift. That just smacks of their lack of understanding of what being a doctor is all about."
—Kirk Smick, OD

Slated to begin this month but likely held up by the typical bureaucratic grind, the Sunshine Act has many people talking—about whether it's an invasion of privacy, a welcome addition to the medical community, or ultimately a detriment to the future of industry support of continuing education. Here, several prominent ODs weigh in.

Sunshine Act: Good, Bad or Indifferent?

"I think as a whole, disclosure and transparency are positive things, and I'm a big believer it is important to have that," says Lexington, Ky., optometrist Paul Karpecki. "All the education boards, including ARBO, already have disclosure requirements, and this new rule is simply adding to it."

One potential downside: Companies may decide to cut back on industry-supported efforts if the reporting requirements become too onerous, Dr. Karpecki adds. For example, under the proposed rule, companies would be required to report to CMS any gift to a doctor that is over $10 (see *Pens to Honoraria to Steak Dinners: What Companies Need to Divulge*, page 36). So, a billion-dollar drug company will now have to track each gift worth over $10, whether it's a lunch, consulting fees, pens or research dollars,

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Take a Systematic Approach to Eye Care

given to an individual doctor over a one-year period and report all that information to CMS. Large pharma companies will potentially have to track what they gave to thousands of doctors; smaller companies may not have the manpower to track and report this deluge of data.

"This is going to be a cost for companies," says Dr. Karpecki, both financially and operationally. "Unfortunately, this may not benefit the profession, as money that may have been allocated to professional efforts will now be allocated to tracking and monitoring," Dr. Karpecki says.

Though the final rule of the Sunshine Act has not yet been issued as of press time, companies are already starting to prepare, ODs say.

Q&A With the AOA

The Sunshine Act is intended to shine a spotlight on relationships between drug or medical device manufacturers and doctors, optometrists included. At a time when regulations of CE and other industry support have been ratcheted up in recent years, the Sunshine Act takes scrutiny to another level, as industry will need to publicize the majority of compensation and/or gifts they provide to doctors.

We asked the AOA to weigh in on this new law and what it may mean to you and your colleagues.

What are the proposed components of this new regulation and how might they impact optometry?

AOA: Congress passed the law to make transparent the benefits that physicians, including optometrists, receive from manufacturers whose products (drugs and devices) are covered by Medicare. The obligation will be on the companies to track and report what goes to the doctors, and the amounts will be posted on a website that the public can access.

The impact on optometry will depend on what the public does, if anything, with the information. Final regulations from the federal government will spell out exactly which types of manufacturers will be subject to reporting, and the process for doctors to review and correct the information. AOA carefully reviewed the proposed requirements and made suggestions to the government on ways to make the reporting more fair and accurate for optometrists and the public. The law and regulations do not prohibit any activity by manufacturers or optometrists, but sheds light on their transactions.

Optometrists will probably want to carefully review their individual reports, and use the available time and procedures described in the final regulations to make corrections before the information is posted online. Optometrists will also want to be prepared to answer questions from patients and potential patients about the transparency.

Is the proposed regulation a positive or negative addition to the medical community and optometry, and what problem, if any, does it resolve?

AOA: The law was enacted because some physicians receive large benefits from manufacturers, and Congress wanted to make sure that patients, regulators and others would know about those transactions in case the rewards were valuable enough to potentially impact decisions, findings or recommendations made by a doctor. Many physicians receive small or token benefits from manufacturers, which will also be publicly reported.

Optometry is not particularly helped or harmed, since the law does not restrict any relationships with manufacturers. Individual optometrists who appear in the reports might not like the public to know the value of benefits they received from manufacturers. All physicians are subject to the reports, so optometrists will likely not stand out in comparison to medical colleagues. The law does not resolve any problems unless the public learns, as a result of this transparency, there are physicians whose judgments were potentially clouded by the value of their relationships with manufacturers.

Although the final regulation has not come out yet, what is the AOA's position on the Sunshine Act?

AOA: AOA did not support the law because it does not benefit optometry. But it also does not hinder optometry much, particularly in comparison to other medical specialties. AOA continues to advocate for changes in the law and regulations to make the Sunshine Act less burdensome for manufacturers and less intrusive for optometrists, and to avoid misleading the public about the legal and appropriate relationships between doctors and manufacturers. This will also require more time for the individual OD to monitor what is reported and if the information is correct. We want to make sure that our members have the necessary steps to view or monitor their activity and potentially identify any incorrect activity.

AOA also asked that the next statement be very visible for the public to read: "We recognize that disclosure alone is not sufficient to differentiate beneficial, legitimate financial relationships from those that create conflict of interests or are otherwise improper. Moreover, financial ties alone do not signify an inappropriate relationship." This is a statement made by CMS.

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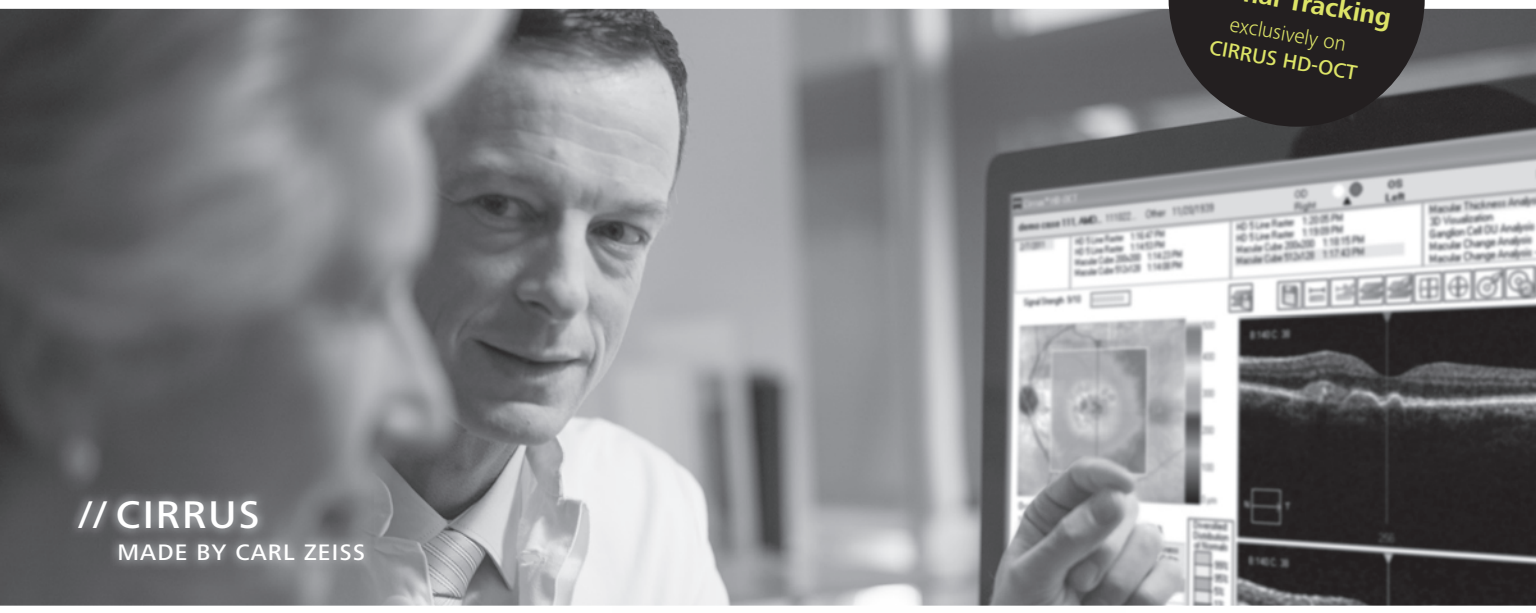

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Kirk Smick, OD, of Morrow, Ga., cites an example of a company that abruptly stopped using a particular doctor for the rest of 2012. Although there is no mandatory reporting yet, the company didn't want the financial compensation to this particular doctor to appear inordinately high.

Just what the federal government hopes to achieve with the Sunshine Act is unclear, according to Dr. Smick. "It is somewhat of an insult that they would think that I would prescribe a certain drug or medical device based on a gift," he says. "That just smacks of their lack of understanding of what being a doctor is about."

"I can't imagine very many patients are going to see this information," Dr. Smick adds. "Secondly, I don't know that they would care even if they do see it; finally, I think there is a way to put a positive spin on it. If anyone asks me if I am getting money from a company, I will say, 'You know, I'm pretty well known, and companies invite me to leave my practice to do educational presentations, and of course they pay me for that time out of the office where I normally earn my living.'"

If a patient goes online and discovers their doctor has earned money from a company, Dr. Karpecki doesn't believe this is going to be a significant negative. "I don't think patients are going to decide they don't want to go to a doctor who works with a lot of companies—a lot of times they do just the opposite," he says.

Optometrist Ben Gaddie of Louisville, Ky., feels that the Sunshine Act will have very little impact on optometry or medicine. "If I were a consumer of eye care, I would *want* to find the doctor who is helping shape the industry and is a leader amongst his or her peers." Dr. Gaddie thinks the regulation will, if anything, allow a concerned party to see the company affiliations a particular doctor has so that they can decide if that relationship somehow affects the care that they receive.

"I don't think it solves any problems," says Dr. Gaddie, "and I'm not convinced there is a problem to begin with." Simply put, he says, without doctors acting as consultants or performing research, there would be no innovation in science or patient care. "It's not a crime to help move your profession or industry forward and ultimately better serve the patients who seek our care."

The Sunshine Act is just the latest addition to an already toxic health care environment, Dr. Epstein says. "Overregulation creates a typically senseless bureaucratic cluster that ultimately stifles research and innovation. Worse yet, it insidiously impacts patient care. We are regulating ourselves to death," he says.

Tighter Regulations, Transparency and CEs

New pharma guidelines have become stricter every year, and as a result, relationships between industry and doctors are already under tight scrutiny, which is a good thing, says Jack Schaeffer, OD, of Birmingham, Ala. Long gone are the days of pharma-sponsored CE dinners; the new norm is transparency between doctors and their audience. For example, if a doctor is giving a lecture, it is extremely important for the audience to know the doctor has done research or

Pens to Honoraria to Steak Dinners: What Companies Need to Divulge

Under the proposed rule of the Sunshine Act, manufacturers would need to report any financial gifts to doctors that are valued over $10 or an aggregated sum of $100 over a one-year period.

For example, if a company takes you out for an $8 lunch twice in a year, they don't need to report this to CMS, since the one-time gifts are less than $10 and the total yearly amount is under $100. However, if in the course of one year a company provides you five meals each worth $9, a speaker fee of $150 and pens worth $5, the aggregate amount is greater than $100; the company would have to report each item to CMS.

Based on this financial formula under the current proposed rule, companies would need to report the following:

- Consulting fees
- Compensation for services other than consulting
- Honoraria
- Gift
- Entertainment
- Food
- Travel
- Education
- Research
- Charitable contribution
- Royalty or license
- Current or prospective ownership or investment interest
- Direct compensation for serving as faculty or as a speaker for a medical education program
- Grant

Companies would also have to track and report the type of payment they gave doctors. The types of payment include:

- Cash or a cash equivalent.
- In-kind items or services.
- Stock, a stock option, or any other ownership interest, dividend, profit or other return on investment.

CMS has included a few exceptions to this mandatory reporting requirement. For a full list and to view the proposed rule in its entirety, go to http://federalregister.gov/a/2011-32244

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is on an advisory board, so that the lecture becomes fair and balanced, Dr. Schaeffer says.

"As a medical community, we adopted evidence-based medicine many years ago," Dr. Schaeffer says, and those principles are evident in every lecture. "Keeping that in mind, there are many anecdotal experiences that add value but may not be supported by research" and these should be disclosed as anecdotal in nature when lecturing. If the clinician's experience is limited to one drug in a class due to a consulting arrangement, it would be valuable for that limitation to be disclosed.

Dr. Epstein believes the Sunshine Act will have little impact on optometry because the profession was never the recipient of the corporate largesse other medical specialties received in the past. "I have heard stories of a few key optometrists being treated to incredible perks, including international trips, but that was before my time, and I have been around for a while."

All health care professions "share a symbiotic relationship with industry, since we depend on the drugs and devices they produce," Dr. Epstein says. "There are a number of optometrists who work very closely with industry, and some earn significant income from that, but I suspect most people are already aware of those relationships."

But will the Sunshine Act change the landscape of CE, with doctors opting to skip the lecture circuit due to a sudden spotlight shone on financial compensation they receive? "I don't think it will have that effect at all because I don't think we care," Dr. Smick says. However, he suggests that it may impact some companies if they prefer to shield their consulting relationships from scrutiny. "Maybe I'm a lecturer for the company and that company may not want me to know they are supporting another lecturer more than me," Dr. Smick says. "They may try to even the playing field a little as a result."

Since disclosures are in place already, Dr. Schaeffer doesn't believe the new Sunshine Act will prompt ODs to shy away from industry affiliations. "Almost all doctors who lecture receive money from different companies. That is how CE is paid for," Dr. Schaeffer says, and there are buffers already in place. At most meetings, pharma companies support the conference, and the organization that plans the meeting pays the doctors for the independent lectures, Dr. Schaeffer adds.

For those ODs just starting out on the lecture circuit, the increased scrutiny of the Sunshine Act may make speaking engagements seem less appealing. "In the future, I do think that people who do research or go on the podium may be less inclined to do so," says Dr. Epstein. "Becoming a sought-after speaker requires an incredible amount of study and preparation. It's not as easy or as glamorous as some might think," he says, to be in the public eye. "You are away from family, and if you have young children, you risk missing moments you can never recapture. Even for the most experienced road warrior, travel wears you out." Financial scrutiny and the potential for misinterpretation of consulting relationships only adds yet another reason to stay home, he says.

Speaking is also not a get-rich-quick scheme. Dr. Epstein explains

Shedding Light on the Sunshine Act

The Sunshine Act, a part of the Affordable Care Act, was created to increase transparency in the health care system. It would require manufacturers of drugs, devices, biologicals and medical supplies covered by Medicare, Medicaid or the Children's Health Insurance Program to report to CMS payments or other transfers of value they make to physicians and teaching hospitals.

This would include gifts, consulting fees, research activities, speaking fees, meals and travel arrangements. The proposed rule would also require manufacturers and group purchasing organizations (GPOs) to disclose to CMS ownership or investment interests held by physicians (or the immediate family members of physicians).

CMS's position is that disclosure of these relationships will discourage the inappropriate influence on clinical decision-making that sometimes occurs, while still allowing legitimate partnerships between physicians and industry.

Companies will not be required to begin collecting data for CMS until after a final rule is published. As of press time, CMS had not issued the final rule and would not comment on a timeline when the rule would be finalized. Once the rule is issued, companies—along with doctors and teaching hospitals—will be allowed to review and correct information prior to its publication. Depending on the timing of the final rule, CMS is proposing that manufacturers and GPOs will be required to submit a partial year on March 31, 2013.

For those who violate the reporting requirements, the penalties are steep. Violators would be face monetary penalties capped at $150,000 annually for failing to report, and $1 million for knowingly failing to report.

CMS is proposing to leave it up to the company and physician to resolve any potential disputes about the information reported. CMS is also proposing that if the dispute cannot be resolved, the transaction will be noted as disputed, and both amounts will be published.

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that most on the lecture circuit earn the same or less than they would generate if they stayed home and saw patients in their practice.

"People shouldn't misunderstand," Dr. Epstein says. "If someone earns a significant amount of money speaking or consulting, they really earned it. No company gives money away. People earning that kind of money are working hard for it, and they have developed skills and abilities that are in demand. There is no skullduggery here. There are no behind the scenes pay-offs."

Will Top Lecturers Retreat?

Doctors interviewed for this article are mostly familiar faces on the teaching and lecture circuit. When asked if they will refrain or cut back on industry-affiliated educational efforts as a result of the Sunshine Act, each OD said most likely no.

"I really don't see the need for the regulation, but at the same time, I don't have anything to hide," Dr. Gaddie says. "I am proud of the work I do to promote the profession, and I hope that my educational efforts help advance the level of care that other optometrists provide to their patients. It is a mutually beneficial relationship; industry couldn't survive without the doctors and the doctors would be hard pressed to maintain the level of education and product innovation without industry partners."

Dr. Epstein says that, although he resents the Sunshine Act's invasion of his privacy, he doesn't think it will change the way he conducts his professional pursuits. "I never hid industry relationships. I work with a number of different companies, and I believe that what I offer them has substantial value." He points out that he has also used his industry relationships to help the profession.

"At this point in my career, people know who I am," says Dr. Epstein. "I have always been a straight shooter. I say what I think even when it is clearly not in my personal best interest. I believe my colleagues will see me the same way regardless of how I chose to earn my living."

For those who love to teach, public reporting of earnings from industry won't stop them. "This isn't going to affect my attitude for wanting to do these things because I enjoy it and I enjoy teaching," Dr. Smick says. "It probably won't have any direct effect on me."

Company Loyalty

For those doctors who are aligned with a specific company, what will this new regulation mean to them?

"It certainly makes me think that a company now will be able to see that not only am I getting income from them, but also maybe from their competitor," Dr. Smick says. "What does that mean? It depends on the company, but most of my friends and colleagues don't want to get bundled into Company XYZ's camp and viewed as its mouthpiece." Still, he says some manufacturers do cultivate a cadre of speakers who don't really lecture for other companies. "That is really going to be borne out. I think companies are going to have to 'spread it around' a little more."

Some speakers give the impression to their audiences that they work with a variety of companies and therefore are not biased, Dr. Gaddie says. "The regulation will allow everyone to see if the speaker is indeed working with everyone or just one entity. It should be interesting," he says.

With the Sunshine Act, the audience will know if a certain speaker is aligned with a certain company and its products, Dr. Schaeffer says. "But even those individuals are going to still give a fair and balanced lecture. They may just favor one drug over another as long as the efficacy is the same. I can't think of anyone I've seen lecture favor one drug or product over another that is less effective when teaching other optometrists about clinical care."

Dr. Epstein says some doctors do have issues with bias, but "you can generally tell, and the audience picks that up."

So in the end, how much of an impact will the Sunshine Act have on optometry?

"The main thing is that there will never be any laws or guidelines that are stricter than the guidelines that we place on each other," Dr. Schaeffer says. As a participant and planner on the lecture circuit, Dr. Schaeffer says if he or a colleague hears a lecturer or reads an article that they thought was not in the best interest of patient care, that individual would have a very difficult time pursuing a career lecturing or writing.

Adds Andy Gurwood, OD, of Salus University in Philadelphia: "My general thoughts are that unless an egregious bias is exposed and put forth in a place where it can be easily discovered by the public, the act does no harm. I generally believe that while doctors may work with industry, they continue to do what is right, forgetting about relationships in favor of correct management." That, he says, is what this legislation is about—revealing the appearance of impropriety.

"I have faith in my colleagues," Dr. Gurwood says, "and unless it becomes clear that decisions are being made contrary to the standards of care on favor of bias for gain, I consider the issue moot." ■

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Peeling Back the Layers of RCE

Diagnosing a recurrent corneal erosion is relatively easy. Treating it, however, is a different story. Here's a look at the best available options. **By Aaron Bronner, OD**

In terms of diagnostic mystery, isolated recurrent corneal erosions (RCE) don't usually offer much—their symptomology and timing are consistent to the point of being pathognomonic. Symptomatically, they present with a shout rather than a whisper. Patients report substantial—in many cases—debilitating pain that occurs acutely upon waking or in the middle of the night, as well as a dramatically watering eye and photophobia.

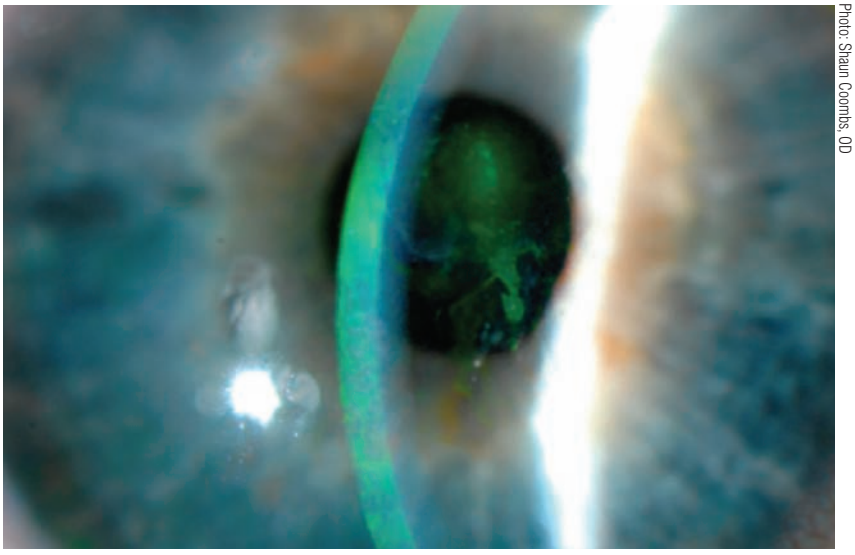
When paired with concomitant ocular surface disease—specifically, substantial dry eye syndrome, floppy lid syndrome or nocturnal lagophthalmos—arriving at the diagnosis can be more challenging. However, in most cases, carefully listening to patient symptoms paired with timely examination of the cornea can almost always lead to the correct diagnosis.

Despite relatively little difficulty identifying RCE and our fairly good understanding of its pathogenesis, our most frequently employed treatments for it—bland or hypertonic ointment—haven't evolved much since RCE was first recognized 140 years ago. In this article, we'll examine the condition, discuss its pathogenesis and compare various treatment strategies.

Where It All Begins

RCEs have their foundation in abnormalities in the junction between the patient's corneal epithelium and Bowman's layer, a thin acellular layer located just below the corneal epithelial basement membrane. These abnormalities may be primary in nature (caused by a dystrophy) or secondary (caused by trauma). Traumatic RCEs are the most common type, accounting for 45% to 64% of cases.[1,2] Dystrophy-associated RCE, typically linked to epithelial basement membrane dystrophy (EBMD), accounts for 19% to 29% of cases.[1,2] It may also be encountered in stromal dystrophies, such as lattice and granular dystrophies.

With each type of RCE, the initial insult differs—but the anatomy



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involved in their formation is the same. Let's take a deeper look.

The corneal epithelium, five to seven cells in thickness, is composed of the mature superficial layer, the evolving wing cell layer and the miotic monolayer of basal cells. Like all of the corneal epithelial layers, the basal cells are joined to adjacent cells by desmosomes. On their basal surface, they are also joined to the basement membrane, Bowman's layer and the anterior stroma by an adhesion complex made up of hemidesmosomes and type VII collagen-anchoring fibrils.[3,4]

Abnormal deposition of the epithelial basement membrane—after trauma or secondary to dystrophic processes—disrupts this adhesion complex, which is thought to be causative in the genesis of RCE. When an isolated corneal abrasion occurs (leaving behind intact basement membrane), the lesion will generally heal in five to seven days with appropriate formation of the adhesion complex. However, when the basement membrane is also removed, mature adherence does not take place until six to eight weeks.[2]

During the interval when the causative epithelial abrasion has superficially healed, but an immature, absent or adherent complex is present, the patient is at risk for spontaneously sloughing the fragile epithelium. This sloughing nearly always takes place at night, when mild epithelial edema or ocular surface drying may weaken the epithelium's tectonic integrity or promote adhesion to the eyelid.

Onset occurs upon waking or during REM sleep, when the shearing force generated between eyelid and corneal epithelium results in a reopening of the abrasion. EBMD-related RCEs are caused by similar, though primary, abnormalities in the adhesion complex.

RCEs may be either microform or macroform in size. Macroform RCEs present with the classic history and an epithelial defect. Microform lesions typically epithelialize between the onset of symptoms and presentation to clinic.

Treatment

Treatment of RCE can be divided into the acute and chronic phases.

• In the acute phase, the goal of therapy is defect closure. This provides subsequent symptom relief to the patient. Acute-phase RCEs, like all corneal abrasions, often respond well to patching, ointment (bland or antibiotic) or bandage soft contact lenses (BSCLs). Palliative care in the form of topical NSAIDs can effectively limit pain, but does have the potential to slow healing.

• The goal of treatment in the chronic phase is to either passively allow—or therapeutically facilitate—effective formation of the anchoring complex. This is attempted either through protecting the corneal epithelium to allow time for appropriate adhesion complex formation, or through induction of scar-based adhesions from the epithelium to the anterior stroma.

The treatment of these two phases is not mutually exclusive—that is, modulation of the corneal healing response can and should be implemented while the acute episode is healing to reduce potential for future episodes. However, this idea doesn't seem applicable to the original offending abrasion; currently, there is no research to suggest that treating traumatic corneal abrasions as an RCE will reduce the likelihood of subsequent RCE development.[5]

Bland or Hypertonic Ointments

Historically, the mainstay treatment for RCE has been nocturnal use of bland or hypertonic ointments. The therapeutic goal of bland ointment is to limit nocturnal friction between the corneal epithelium and the lid. Theoretically, this minimizes shearing forces and, if applied over time, allows for the epithelial adhesion complex to develop appropriately. Hypertonics are designed to limit nocturnal epithelial edema, which is thought to reduce corneal epithelial adherence.

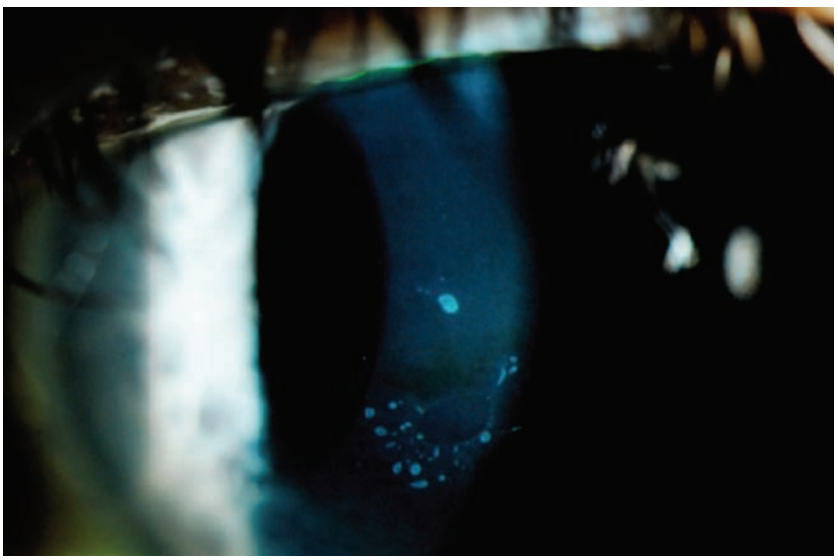

Photo: Reid Mamiya, OD

Typical EBMD with gray subepithelial deposits of basement membrane.

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INDICATIONS AND USAGE: PATADAY™ solution is a mast cell stabilizer indicated for the treatment of ocular itching associated with allergic conjunctivitis.

DOSAGE AND ADMINISTRATION: The recommended dose is one drop in each affected eye once a day.

DOSAGE FORMS AND STRENGTHS: Ophthalmic solution 0.2%: each ml contains 2.22 mg of olopatadine hydrochloride.

CONTRAINDICATIONS: None.

WARNINGS AND PRECAUTIONS: For topical ocular use only: not for injection or oral use. **Contamination of Tip and Solution:** As with any eye drop, to prevent contaminating the dropper tip and solution, care should be taken not to touch the eyelids or surrounding areas with the dropper tip of the bottle. Keep bottle tightly closed when not in use.

Contact Lens Use: Patients should be advised not to wear a contact lens if their eye is red. PATADAY™ (olopatadine hydrochloride ophthalmic solution) 0.2% should not be used to treat contact lens related irritation. The preservative in PATADAY™ solution, benzalkonium chloride, may be absorbed by soft contact lenses. Patients who wear soft contact lenses and whose eyes are not red, should be instructed to wait at least ten minutes after instilling PATADAY™ (olopatadine hydrochloride ophthalmic solution) 0.2% before they insert their contact lenses.

ADVERSE REACTIONS: Symptoms similar to cold syndrome and pharyngitis were reported at an incidence of approximately 10%. The following ocular adverse experiences were reported in 5% or less of patients: blurred vision, burning or stinging, conjunctivitis, dry eye, foreign body sensation, hyperemia, hypersensitivity, keratitis, lid edema, pain and ocular pruritus. The following non-ocular adverse experiences were reported in 5% or less of patients: asthenia, back pain, flu syndrome, headache, increased cough, infection, nausea, rhinitis, sinusitis and taste perversion. Some of these events were similar to the underlying disease being studied.

USE IN SPECIFIC POPULATIONS: Pregnancy: Teratogenic effects: Pregnancy Category C. Olopatadine was found not to be teratogenic in rats and rabbits. However, rats treated at 600 mg/kg/day, or 150,000 times the MROHD and rabbits treated at 400 mg/kg/day, or approximately 100,000 times the MROHD, during organogenesis showed a decrease in live fetuses. In addition, rats treated with 600 mg/ kg/day of olopatadine during organogenesis showed a decrease in fetal weight. Further, rats treated with 600 mg/kg/day of olopatadine during late gestation through the lactation period showed a decrease in neonatal survival and body weight. There are, however, no adequate and well-controlled studies in pregnant women. Because animal studies are not always predictive of human responses, this drug should be used in pregnant women only if the potential benefit to the mother justifies the potential risk to the embryo or fetus. **Nursing Mothers:** Olopatadine has been identified in the milk of nursing rats following oral administration. It is not known whether topical ocular administration could result in sufficient systemic absorption to produce detectable quantities in the human breast milk. Nevertheless, caution should be exercised when PATADAY™ (olopatadine hydrochloride ophthalmic solution) 0.2% is administered to a nursing mother. **Pediatric Use:** Safety and effectiveness in pediatric patients below the age of 2 years have not been established. **Geriatric Use:** No overall differences in safety and effectiveness have been observed between elderly and younger patients.

NONCLINICAL TOXICOLOGY: Olopatadine administered orally was not carcinogenic in mice and rats in doses up to 500 mg/kg/day and 200 mg/kg/day, respectively. Based on a 40 µL drop size and a 50 kg person, these doses were approximately 150,000 and 50,000 times higher than the maximum recommended ocular human dose (MROHD). No mutagenic potential was observed when olopatadine was tested in an in vitro bacterial reverse mutation (Ames) test, an in vitro mammalian chromosome aberration assay or an in vivo mouse micronucleus test. Olopatadine administered to male and female rats at oral doses of approximately 100,000 times MROHD level resulted in a slight decrease in the fertility index and reduced implantation rate; no effects on reproductive function were observed at doses of approximately 15,000 times the MROHD level.

U.S. Patents Nos. 5,641,805; 6,995,186; 7,402,609

References: 1. IMS Health, IMS National Prescription Audit™, August 2010 to September 2012, USC 61500 OPHTH ANTI-ALLERGY. **2.** Blaiss MS, Tort MJ. Zero itch in eyes treated with olopatadine hydrochloride ophthalmic solution, 0.2% in bilateral conjunctival allergen challenge studies. Poster presented at: World Allergy Conference; December 2011; Cancun, Mexico.

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Yet when compared directly in one study, the authors noted no difference between hypertonic ointment and bland ointment, leading them to conclude that lubrication was the sole therapeutic benefit to both approaches.[4] While most studies seem to suggest a 30% to 50% recurrence rate with conservative therapy, a randomized study with 72 patients who suffered traumatic corneal injury found an actual worsening of symptoms when treated with bland ointment—although no impact on the likelihood of future RCE was seen.[1,5,6]

Bandage Soft Contact Lenses

BSCLs, along with matrix metalloproteinase inhibitors and autologous serum use, occupy the middle ground between conservative and aggressive therapy. In principle, BSCLs effectively provide a buffer between the lid and the corneal epithelium. Therefore, long-term use of BSCLs could then be used to prevent RCE while the epithelial adhesion complex matures.

In the past, BSCLs were not viewed as an efficacious or even entirely safe therapy; however, since the advent of silicone hydrogel contact lenses, they seem to have taken on more of a prominent role.[7] In a 2011 study, subjects who previously had failed medical-only therapy were assigned to treatment with a plano power, 8.6mm base curve Ciba Night and Day BSCL (Alcon) and prophylactic topical ofloxacin ophthalmic drops (dosed BID).[8] The patients continued this treatment for three consecutive months, reporting back to clinic every two weeks for lens replacement and evaluation. Of the group, 75% had no recurrence over one year after discontinuing therapy.

Despite the small sample size of this study (12 patients), it is interesting to note that the success rate is as good or better than that reported with anterior stromal puncture (ASP) and equivalent or slightly worse than that reported with epithelial debridement with diamond burr polishing and phototherapeutic keratectomy (PTK), which are considerably more invasive and costly (especially PTK).[1,9-11]

MMP Inhibitors

Matrix metalloproteinases (MMPs) are a family of enzymes that play a role in the remodeling degradation of connective tissue, including epithelial basement membrane. While there are several important members of this family active in the corneal wound response, MMP-2 and MMP-9 appear to be of particular importance in RCE. Both MMP-2 and MMP-9 are produced by stromal fibroblasts and epithelial cells, respectively, and each is important in breaking down components of the epithelial adhesion complex.[12]

Just how deleterious are the effects of increased MMP expression on the tectonic structure of the cornea? *Pseudomonas aeruginosa*—a gram-negative bacterium with the potential to rapidly cause corneal perforation—secretes its own MMPs, allowing it to break down connective tissue, enabling deeper penetration.[12]

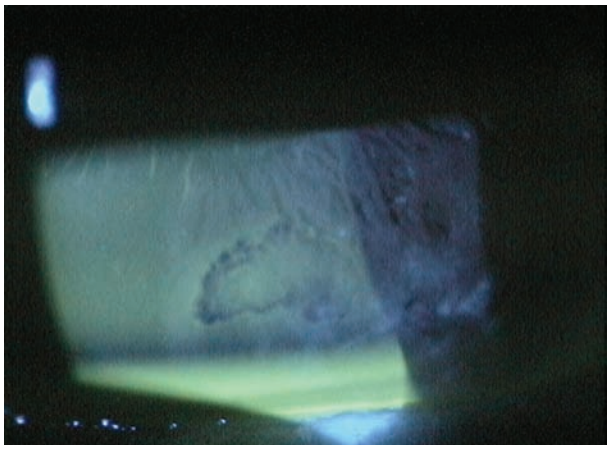
In regard to their role in RCE, MMP-2 and MMP-9 have been shown to increase concentration within the tear film among patients with RCE, theoretically leading to reduced stability of the epithelial basement membrane and increased potential for RCE.[13] Theoretically, then, the use of tetracyclines as inhibitors of MMP activity is supported in the treatment of RCE. In a group of seven patients with RCE recalcitrant to conservative therapy, oral doxycycline was used (50mg BID for two months) with no recurrences noted.[14] However, this study only evaluated RCE cases with traumatic etiologies; the sample did not include any dystrophic cases of RCE. This may have importance when selecting a treatment for RCE, as MMP activity conceptually would not be as likely to play a role in the genesis of dystrophy-associated RCE.

Likewise, corticosteroids also can suppress MMP activity and expression. In the same study noted above, topical corticosteroids also were used during the acute stage with the goal of further suppressing MMP activity. These too were found to have a beneficial effect—although it was slightly weaker in limiting expression compared to doxycycline.[14] With any use of topical corticosteroids, prospective benefit needs to be balanced with potential risk, and appropriate follow-up is required. As the anchoring complex takes two to three months on average to stabilize, therapeutic suppression of MMPs should be continued over that timeframe.[2]

Autologous Serum

Autologous serum topical eye drops are made out of blood drawn from the patient. The blood is centrifuged; the serum is drawn off and, in some cases, diluted. The serum solution is then packaged and used as an eye drop. The benefit of this modality is that the biochemical properties of blood serum are very similar to that of the tear film.

Its use has been explored in the treatment of a variety of ocular surface diseases and has been found to be particularly effective in accelerating the closure of persistent epithelial defects.[15] In one study of eyes



Topical sodium fluorescein clearly delineating the RCE bed.

with RCE not amenable to standard therapy, autologous serum eye drops were used.[15] Over two years of follow-up, three recurrences were noted out of 11 eyes. Autologous serum generally is accepted as safe, but serum sterility is not guaranteed. So, concomitant use with prophylactic antibiotic drops is advisable. Once again, treatment duration should reflect the timing required for full healing. Dosing should be reflective of coexisting ocular surface disease and could vary from four times per day to every hour or more.

It's important to discuss this therapy's inherent limitations with patients—it can be costly and cumbersome. It takes several steps to obtain: finding a compounding pharmacy to prepare the sample, a lab to draw the sample and sending it to the compounding pharmacy. The patient must undergo a series of blood tests to ensure the sample is HIV negative and free of hepatitis prior to processing of the blood. Autologous serum constitutes experimental therapy, a point that should be covered with patients and reflected in the follow-up.

Superficial Keratectomy

For RCE that is unresponsive to moderate treatment, consider more aggressive approaches, such as delamination of the corneal epithelium and basement membrane with or without polishing of Bowman's layer, ASP and PTK.

The premise behind superficial keratectomy is that if irregularities in the epithelium and anchoring complex are removed and allowed to grow back in a controlled environment, the structures may normalize as they develop.

The effectiveness of superficial keratectomy alone, without polishing of Bowman's layer, seems to be in

line with less aggressive approaches. When combined with diamond burr polishing of Bowman's layer, the effect is enhanced.[16] A 2009 study reported a 6% recurrence rate in 25 eyes, compared to 18% in a patient-blinded group that received debridement alone.[16] Other groups have confirmed similar findings.[16]

ASP and PTK

So far, the strategies we discussed have focused on protecting the epithelium as it heals to allow for appropriate formation of the anchoring complex. The goal with ASP and PTK is slightly different—here, we want to create new anchoring junctions.

• ***ASP.*** In anterior stromal puncture, a 23- to 25-gauge needle is inserted shallowly into the cornea, penetrating the anterior stroma in a grid-like pattern throughout the bed of erosion. The goal is to create small pinpoint scars into the anterior stroma. The rationale for ASP is that RCE occurs much less frequently when traumatic abrasions penetrate the anterior stroma, compared to those that are simply epithelial in nature.[11]

Different reports have found that the effectiveness of the procedure varies from 60% to 80%.[1,9,11] ASP should be reserved for RCE cases that have their beds out of the visual axis, as there is some potential for reduction in best spectacle-corrected visual acuity if performed in or near the visual axis secondary to scarring.

• ***PTK.*** PTK surfaced as a treatment for RCE when it was realized that recurrence of RCE in eyes undergoing photorefractive keratectomy was reduced.[4] The exact mechanism is uncertain; however, it has been shown that type VII collagen fibers and hemidesmosomes (both major components of the epithelial-anchoring complex) increased in the laser-treated corneas of monkeys.[4]

In the treatment of RCE, PTK involves applying the excimer laser to the bed of the RCE, either transepithelially or after epithelial debridement. The success rate is variable but generally high (ranging from 74% to 100%); however, the cost associated with the device and the procedure keep it from being used as a frontline therapy.[11,17,18] It should be reserved for cases in which more affordable, less invasive therapies have failed.

In Practice

Rather than using a cookie-cutter treatment paradigm, I have found it reasonable to approach RCE in a scientific manner in my practice. In otherwise healthy eyes, I've had good success with a silicone hydrogel BSCL worn for 10 to 12 weeks and replaced every other week. I always pair this with a prophylactic antibiotic eye drop that has minimal epithelial toxicity, such as ciprofloxacin, while the epithelial defect persists. In the first month, I require the patient to return to the clinic so I can change the BSCL—I use jeweler's forceps to assist in atraumatic removal.

After the first month, I've allowed patients to exchange the BSCL themselves, if they feel comfortable with it. I instruct them to use artificial tears to assist in floating the lens prior to removal.

However, if the patient has significant lid margin disease or has failed with the BSCL approach in the past, I use oral doxycycline and a soft steroid-antibiotic combination drop, such as Zylet (loteprednol 0.5% and tobramycin 0.3%, Bausch + Lomb). This provides some antibacterial coverage, especially in the acute stage when an epithelial defect is present.

Do Amniotic Membrane Grafts Mesh Well with RCE Treatment?

Recently, amniotic membrane grafting has become an increasingly popular topic in the optometric community, possibly due to the ProKera ring (Bio-Tissue) becoming more widely available. This device is an amniotic membrane sheet supported on a 16mm plastic ring. It can be applied simply as a large-diameter contact lens, though the ring itself is much thicker than a standard contact lens.

The benefits of amniotic membrane tissue in the management of inflammatory and non-healing corneal wounds have been well documented during the last 20 years. Amniotic membrane works in these cases by reducing inflammatory mediators, reducing vascularization, providing an artificial basement membrane for re-epithelialization, reducing the scar response, providing antimicrobial effects and promoting appropriate innervation.[19,20]

The indications for amniotic membrane include: Stevens-Johnson syndrome, acid or alkali injury, pain relief for bullous keratopathy, re-epithelialization of neurotrophic ulcers and as a surgical adjunct in pterygium, glaucoma and limbal-grafting procedures.[20] Given its mechanism of action in enhancing epithelialization and reducing inflammation (i.e., MMP activity), amniotic membrane could have a place in the treatment of RCE.

While there are sporadic case reports of successful RCE treatment with ProKera, there are no well-designed studies on the modality. In addition, the life of the amniotic membrane is roughly two weeks on the eye—which does not fit with the six- to eight-week pathogenic timeframe of RCE. Coupled with the costs and regulation associated with biologic tissue, amniotic membrane grafting likely should be relatively far down the list of possible therapies for RCE.

(For more information on amniotic membrane grafts, read this month's Research Review, "The Miracle of Birth," page 86.)

The two strategies of BSCL and MMP suppression with doxycycline could be easily combined as well. But, further suppression using a topical corticosteroid should be avoided—especially prior to closure of the epithelial defect.

I reserve surgical referral for recalcitrant cases of RCE. It could be expected that patients with RCE caused by dystrophic processes will be less amenable to conservative therapy. In these cases, I refer for PTK or epithelial debridement with diamond burr polishing sooner in the disease process.

While the large number of therapeutic options we have at our disposal can seem overwhelming at first, we generally can achieve good success rates in minimizing the recurrence of this painful condition if we remember to treat each patient as an individual, while keeping the different risks and benefits of each therapy in mind. ■

Dr. Bronner is a staff optometrist at the Pacific Cataract and Laser Institute in Kennewick, Wash.

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14th Annual Dry Eye Report

Why Dry Eye Trials Often Fail

From disease variability to confounding underlying conditions, there are countless reasons why new dry eye drugs have come up short in FDA testing.

By Paul M. Karpecki, OD, Co-Chief Clinical Editor

During the last decade, 14 companies have unsuccessfully attempted to secure FDA approval for a dry eye drug. There are just two underlying explanations for this seemingly insurmountable hurdle: drug ineffectiveness or a flawed approval process.

It is interesting to note that many dry eye agents that failed FDA testing subsequently received approval in Asia and/or Europe, and have achieved tremendous commercial success. For example, since its debut in 1995, topical sodium hyaluronate (Hyalein, Santen) has been the most frequently prescribed dry eye agent in Japan.[1]

So, what can we learn from the last 10 years of unsuccessful clinical trials? To address the question accurately, we must possess a better understanding of the FDA approval system, including protocol development, the inclusion/exclusion of signs and symptoms, patient selection and, of course, dry eye disease itself. Only then will we know if future products have a reasonable chance of approval.

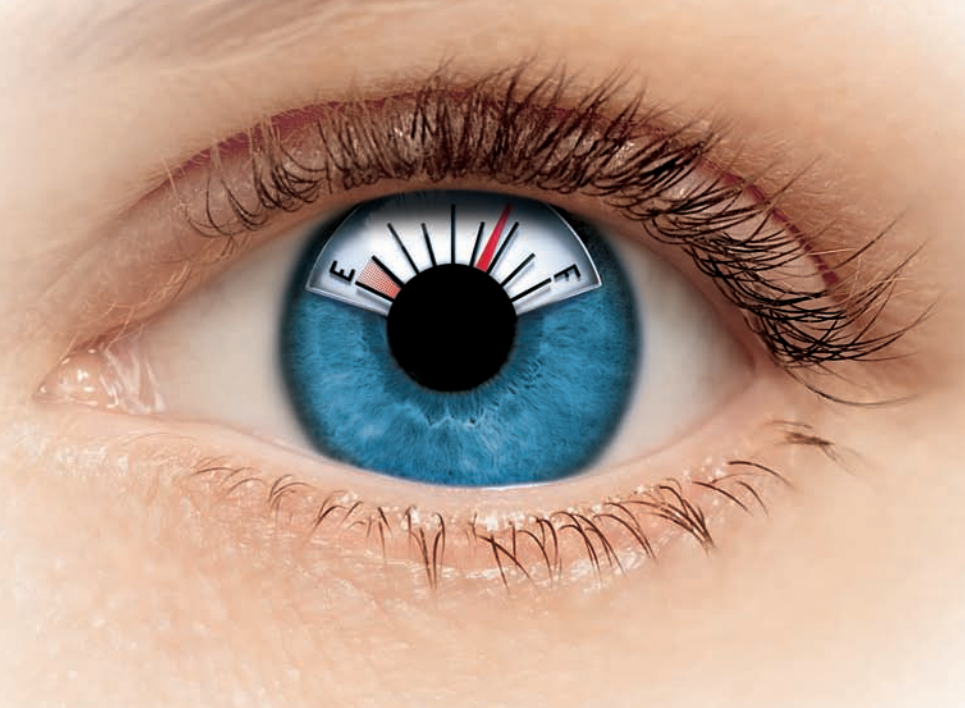
Here, we'll review the inherent difficulties associated with conducting successful FDA testing on potential dry eye drugs. Some of these include appropriate patient selection, the potential for masquerading conditions that can complicate the trial screening process, and the presence of underlying systemic disease that can further exacerbate dry eye signs and symptoms.

Patient Selection

The patient selection process for any FDA trial can be either fairly straightforward or extremely tedious. Certain investigators may have specific patient preferences in mind, depending upon their definition and understanding of the condition being treated. In general, however, the less disease variability that exists between patients, the more rapid and streamlined the selection process.

For example, post-cataract surgery inflammation is one of the most commonly uniform presentations in eye care. Because there is little variability between individual cases, it is relatively simple for researchers to select a population of postoperative cataract patients and evaluate the effectiveness of an anti-inflammatory agent vs. a placebo. Further, because of presentation uniformity, the agent's effects likely will be consistent as well.

Clearly, however, this is not the case with regard to dry eye disease. When investigators select dry eye patients for FDA trials, they must consider disease type; severity level; and varying contributory factors, such as age, gender, lifestyle and concomitant systemic disease. And, although pharmaceutical companies have well-defined inclusion and exclusion criteria, a wide range of patients often still qualify for the



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study. Frequently, these are individuals who have failed on other common therapies for dry eye, such as Restasis (cyclosporine, Allergan) or even topical corticosteroids.

So, what's the result? These patients ultimately are more difficult to treat during the clinical trial, because they have an established history of poor therapeutic response and/or exhibit an underlying systemic condition that requires treatment beforehand.

Types of Dry Eye

As alluded to previously, FDA investigators must consider that there are multiple types of dry eye disease, including evaporative and aqueous deficient. Each form not only exhibits variable severity, but also responds differently to intervention.[2] This means that trial researchers must successfully treat patients with widely differing disease patterns.

For example, a long-standing aqueous deficient dry eye patient with Sjögren's syndrome typically will present with filamentary keratitis and other advanced ocular surface disease findings, such as confluent corneal staining and even scarring of the lacrimal gland.

By contrast, a patient with evaporative dry eye and pronounced meibomian gland dysfunction (MGD) often will not manifest filamentary keratitis or a corneal presentation with severe central staining. Instead, he or she will exhibit obstructed meibomian glands, significant telangiectasia, eyelid inflammation, thickened secretions and notching of the lid margin. Further, this patient may or may not have rosacea.

You cannot realistically expect that both patients, who have markedly different presentations of dry eye disease, will respond similarly to one medication—even though there may be some overlap of clinical findings. And yet, there is a reasonable probability that both individuals would be enrolled in the same clinical trial.

Masquerading Conditions

Perhaps one of the most overlooked reasons for dry eye drug failure in FDA trials is that the patient may not, in fact, have pure dry eye disease. There are many masquerading conditions that either mimic dry eye or can present concomitantly. Either way, masquerading conditions may complicate the results or prohibit complete resolution of the signs and symptoms—even if the dry eye component is treated successfully with the tested agent.

• *Conjunctivochalasis*. Patients with conjunctivochalasis often report foreign body sensation,

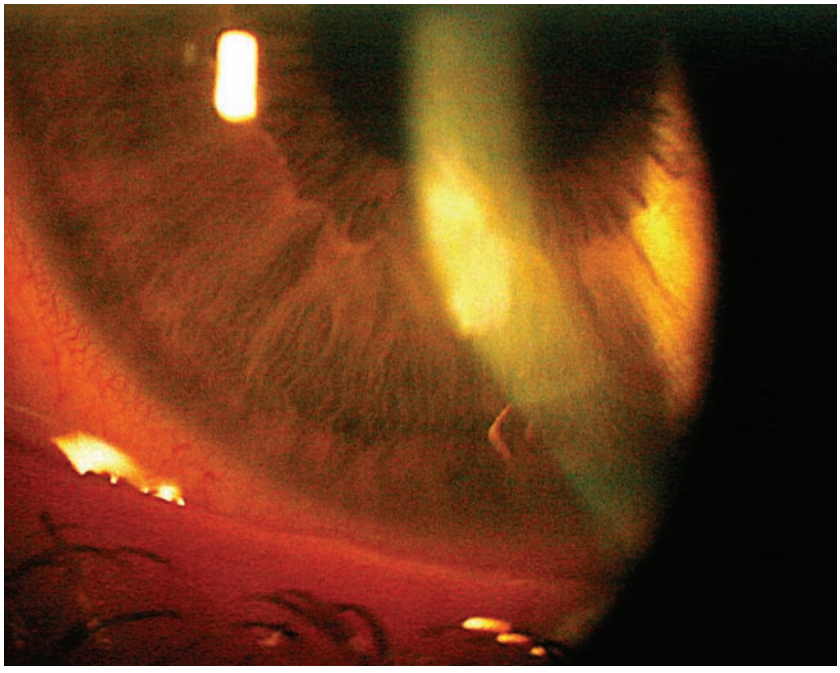
grittiness, irritation and tearing, and may be diagnosed with dry eye disease. Such patients typically will have signs of dry eye and inflammation, and may even show a rapid tear film break-up time, corneal staining and lissamine green conjunctival staining. But, clinical trial recruiters often mistake these signs and symptoms for true dry eye. And unfortunately, no treatment—other than a surgical repair of the conjunctiva—will yield significant sign or symptom improvement.

Most patients with conjunctivochalasis actually can pinpoint where the foreign body or pain emanates from, which typically matches the location of the conjunctival folds. If the patient is able to pinpoint the foreign body sensation that matches the area of conjunctivochalasis, he or she should not be included in a dry eye trial.

Patients with long-standing inflammatory conditions, such as dry eye and allergic conjunctivitis, are prone to develop conjunctivochalasis. Additionally, research has shown a possible association between conjunctivochalasis and immune thyroid disease. A perspective study published in 2006 found that the prevalence of conjunctivochalasis in patients on immune thyroid disease was as high as 88%.[3]

• *RCE.* Another condition that can masquerade as dry eye disease is recurrent corneal erosion (RCE). In these patients, poor adhesion of the hemidesmosomes between the epithelial basement membrane and Bowman's layer results in symptoms of grittiness, dryness, photophobia, tearing, foreign body sensation, blurred vision and, in some instances, irritation or pain.

• *EBMD.* Individuals with epithelial basement membrane dystrophy (EBMD), but no RCE, can display symptoms that mimic dry eye. However, EBMD is not likely to improve with the use of many therapeutic agents. Even with treatment, the presence of maps, dots and fingerprints and staining patterns will remain in patients with anterior corneal dystrophies.



Recurrent corneal erosion, as seen in this patient, is one of the most common dry eye masqueraders observed in clinical practice.

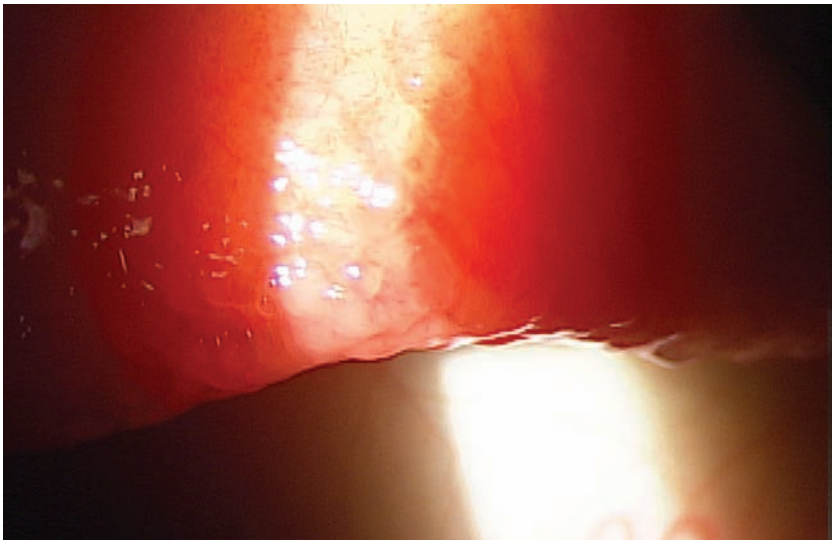
Further, I have reviewed few FDA testing protocols for dry eye that specifically differentiated between symptoms documented at the beginning of the day vs. those observed at the end of the day. From clinical experience, it seems that the symptoms of MGD, RCE and EBMD occur with greater frequency in the morning, while symptoms associated with pure dry eye typically manifest as the day progresses. For example, seeing a patient in the morning for a study visit may elicit a different complaint than a late-day exam.

• *MFS.* Another condition that may present similarly to dry eye disease is mucin fishing syndrome (MFS). Like dry eye, MFS is a chronic condition that causes patients to extract or "fish" strands of mucin from their eyes.

The condition may begin with potential dry eye disease resulting in significant mucin production. The excess mucin causes a visual disturbance and associated symptomatology that is similar to that documented in dry eye.

When patients repeatedly touch their eyes to remove the mucin, they further irritate the ocular surface as well as potentially introduce foreign substances to the eye.[4] Eventually, patients with MFS begin to dig in their eyes habitually. And, as long as fingers continue to come in contact with the eyes, the individual's symptoms will persist—regardless of treatment.

Once again, MFS typically is not considered in the exclusion criteria for any dry eye trial. What makes this even more difficult is that dry eye likely is one of the most common underlying etiologies of MFS,



It is imperative to evert the upper eyelids when screening dry eye patients for giant papillary conjunctivitis, as seen here.

and therefore affected patients likely will be enrolled in a clinical trial for a dry eye medication.

• *FES.* Athough it is a relatively uncommon condition, floppy eyelid syndrome (FES) rarely is included in the exclusion criteria of any dry eye trial. This is because few investigators evert patients' eyelids prior to study enrollment. FES patients often manifest significant ocular surface complications, including advanced superficial punctate keratopathy (SPK), rapid tear film break-up time and conjunctival staining. Further, FES patients typically present with chronic SPK; advanced MGD; and symptoms of burning, stinging, irritation and chronic conjunctival injection.

FES is most common in overweight, middle-aged males who suffer from sleep apnea.[5] Because such individuals have limited eyelid functionality, they have chronic ocular surface inflammation. Furthermore, patients with FES often report spontaneous lid eversion.[6] So, for the purposes of a dry eye investigation, a simple questionnaire might capture this potential finding prior to study enrollment.

Once again, any tested medication is not likely to improve the patient's condition—even though he or she may have many signs and symptoms of dry eye. Unless the upper eyelid is surgically repaired, the symptoms likely will persist.

• *GPC.* Giant papillary conjunctivitis (GPC) is another dry eye masquerader that presents with symptoms of grittiness, irritation, mucin discharge and decreased contact lens wear. Similar to the diagnosis of FES, upper eyelid eversion during the screening examination process will help reveal this condition as well.

• *Salzmann's.* Patients with Salzmann's nodular degeneration (SND) often are enrolled in clinical dry eye studies. In certain individuals, SND's presentation can be quite subtle and may appear as a whitish peripheral haze—not frank nodules. What makes this confusing is that patients with SND exhibit dry, gritty, irritated eyes as well as an associated foreign body sensation, transient blur and rapid tear film break-up time.[7]

Approximately 75% to 90% of all SND cases manifest in white females.[8] This complicates any trial screening process, because dry eye disease is most prevalent in this demographic. Additionally, nearly 63% of all SND cases occur bilaterally—further resembling a presentation of dry eye.[9]

Confocal microscopy reveals that SND lesions are elongated basal epithelial cells and activated keratocytes, particularly in the area of the anterior stroma near the nodules.[10] Occasionally, subbasal nerves and tortuous stromal nerve bundles may be observed. Ultrahigh-resolution optical coherence tomography has uncovered fibrous intraepithelial nodules with significant overlying epithelial thinning in SND patients.[11] This finding may contribute to symptoms that mimic those associated with dry eye disease; however, they will not respond to agents designed to treat ocular surface disease.[11]

• *Asthenopia.* One of the most important and frequently disregarded dry eye masqueraders is asthenopia, described as a collection of conditions that induce ocular fatigue. Affected patients rarely are screened for any clinical trial, and yet demonstrate several symptoms that overlap with dry eye disease. Many patients also may have concurrent dry eye—but if their symptoms are caused by asthenopia, they will not respond fully to the study medication.

Common asthenopic conditions include computer vision syndrome, convergence insufficiency, proprioceptive disparity, fixation disparity, and even exophoria and vertical disparities. Patients with asthenopia typically complain of ocular ache or pain; dryness, redness, grittiness and burning; excessive tearing; visual fatigue with near work, such

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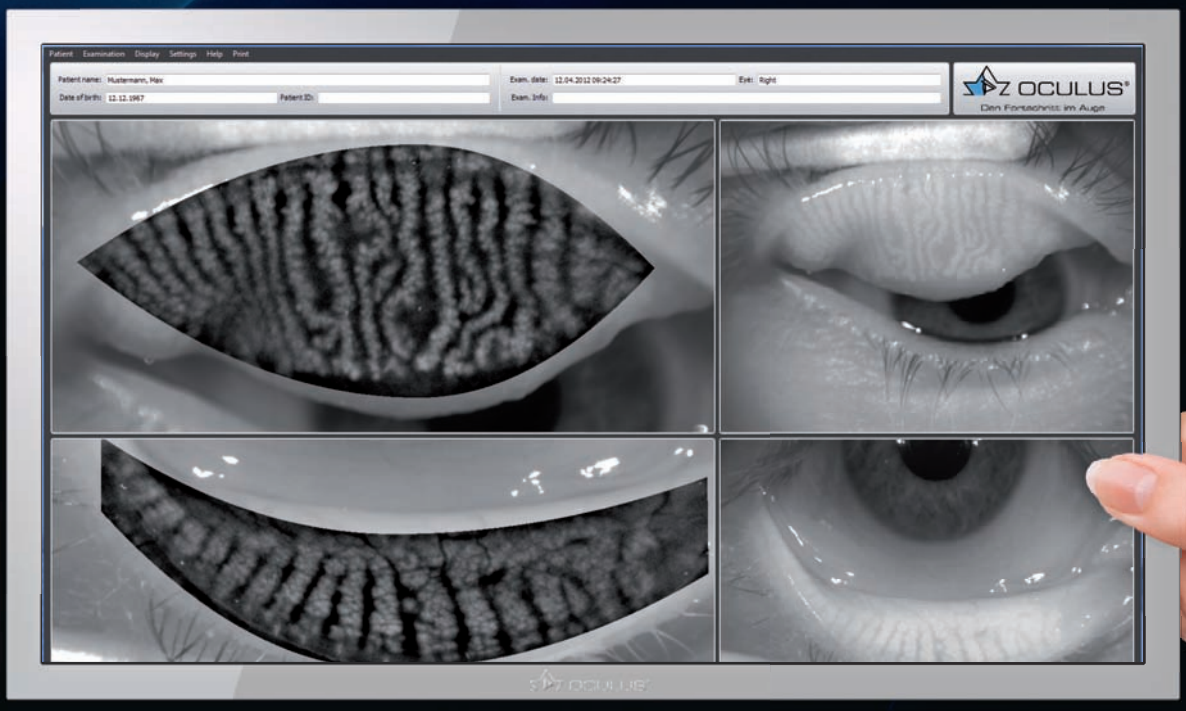

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as computer use; and headaches or an uncomfortable "pulling sensation."[12] The sweeping majority of these symptoms are identical to those experienced by an individual with dry eye.

Patients with dysphoria, fixation disparity, proprioceptive disparity and/or a vertical imbalance between the two eyes often will complain of dryness, grittiness, ocular irritation, visual fatigue, blurred vision and headaches. Many patients who have one of these conditions will test negative for dry eye and will be excluded from a clinical trial. However, in some instances, these individuals will have concomitant dry eye symptoms and will be enrolled in the trial. All too often, however, these symptoms will not resolve with use of the study medication, because the underlying condition is unrelated to dry eye disease.

Limited Correlation Between Signs and Symptoms

Over time, dry eye patients typically experience corneal nerve fiber damage and a subsequent loss of sensitivity. Thus, as a patient's condition progresses, he or she actually may exhibit fewer dry eye symptoms. This association can make proper patient selection very challenging.

Research conducted by Kelly K. Nichols, OD, MPH, PhD, and associates suggested that the signs and symptoms of advanced dry eye disease do not always correlate.[13] Another study indicated that 40% of dry eye patients do not manifest symptoms.[14] Furthermore, patients with neurotrophic dry eye typically will have advanced signs, but few if any symptoms other than blurred vision.[14]

Relying upon the presence of corneal staining as an indicating sign of dry eye disease also may be problematic for trial researchers. If corneal staining is selected as a screening parameter, many early dry eye patients will be excluded. When corneal staining becomes clinically evident, dry eye disease is fairly advanced. This is similar to the presence of visual field defects during a glaucoma screening—by the time a clinician notes a defect, the nerve fiber layer already has been damaged.

Underlying Disease and Systemic Drug Use

Many dry eye patients who are potential candidates for a clinical trial may have contributory underlying systemic diseases. Further, the medications used to control these conditions can further exacerbate ocular dryness. And, as long as patients use these medications and/or have poor control of the underling systemic condition, they will continue to exhibit dry eye symptoms—often irrespective of topical intervention.

Some patients with long-standing acne rosacea, for example, also have advanced MGD and associated dry eye disease. In these instances, patients will pass many of the entrance tests required for a dry eye trial. However, because the meibomian glands are so scarred and damaged, very few—if any— topical agents will modify this condition. Instead, the patient will require a systemic agent, such as doxycycline and/or mechanical treatments to control the underlying rosacea and achieve an optimal result.

Additionally, we often see chronic staining and even persistent epithelial defects in patients diagnosed with type 2 diabetes mellitus. Until those individuals improved glucose control with insulin therapy, very few topical agents effectively treated their dry eye.

Furthermore, patients who are on multiple systemic medications, including diuretics or antihistamines, are much more likely to have dry eye symptoms. As long as these systemic medications are being used, patients in a dry eye trial will experience a limited response to the drug being tested. Patients who are on more than three systemic medications should be excluded from dry eye clinical trials.[15]

Undocumented Disease Variability Between the Eyes

I have been working in the field of dry eye management for my entire career, which now spans close to 20 years. Even after two decades, I've realized that we still have a lot more to learn about the overarching complexity and varying presentation of dry eye disease.

For instance, just within the last few years, we've begun to notice that dry eye patients often demonstrate bilateral symptom variability. In other words, it appears that patients with dry eye often experience compensating effects between the two eyes, such as more pronounced staining or increased tear film osmolarity in one eye vs. the other.

This makes accurate clinical testing of both eyes critical. For example, it is imperative for trial researchers to examine both eyes independently (e.g., not to average Schirmer readings, osmolarity scores or tear film break-up times together, but instead document the highest measurements for each eye individually). Bottom line—a more comprehensively thorough method to measure dry eye severity in FDA trials likely will be required for new dry eye drugs to demonstrate marked success and receive approval in the future.

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A Glimpse at Dry Eye Drugs in the Pipeline

By Katherine M. Mastrota, MS, OD

It has been more than a decade since the first and only dry eye prescription medication, Restasis, received FDA approval. And while pharmaceutical companies have had tremendously limited success bringing a new drug to the US market, excitement is now brewing as we anticipate the approval of several new therapeutic agents for dry eye disease within the next few years.

Currently, there more than a dozen novel, innovative drugs in FDA trials. Any one of these medications could be the next big breakthrough product used to treat your dry eye patients. Here is a review of the most promising dry eye medications across various phases of development:

• ***Lifitegrast.*** SARcode Bioscience completed Phase III testing of this first-in-class, highly selective, small-molecule, integrin antagonist in October 2012. Lifitegrast inhibits the binding of two key surface proteins—lymphocyte function-associated antigen-1 and intercellular adhesion molecule (LFA-1/ICAM-1)—that are integral to chronic T-cell mediated inflammation. This action prevents T-cell migration, adhesion, proliferation and cytokine release, which are all implicated in dry eye disease.

In the Phase III study, lifitegrast 5.0% ophthalmic solution demonstrated a superior reduction in the signs and symptoms of dry eye disease vs. placebo.[1] It significantly improved inferior and total corneal staining scores from baseline, and markedly reduced both ocular discomfort and dryness.

Following completion of the Phase III trial, SARcode Bioscience initiated a year-long safety study (SONATA), and will soon begin a second Phase III confirmatory study (OPUS-2).[2] Data from both SONATA and OPUS-2 will be used to support the filing of a New Drug Application.

• ***CF101***. OpthaliX, the US subsidiary of Israel's Can-Fite BioPharma, is currently evaluating this first-in-class A3 adenosine receptor agonist in a Phase III trial. A3 adenosine receptors are involved in a variety of intracellular signaling pathways and physiological functions. These receptors also help to inhibit degranulation in neutrophil-mediated tissue injury. Further, CF101 modulates key signaling proteins that inhibit inflammatory cytokine/chemokine production and induce inflammatory cell apoptosis.

CF101 is administered orally, and has been tested for the treatment of dry eye disease, glaucoma and uveitis in Phase II studies.[3] The drug also is being evaluated for the treatment of autoimmune inflammatory diseases, including rheumatoid arthritis (Phase IIb) and psoriasis (Phase II/III).

• ***Rebamipide***. This gastroprotective drug stimulates endogenous prostaglandin generation in the gastric mucosa, scavenges free radicals, and is reported to accelerate ulcer healing.[4] In 1990, Japan's Otsuka Pharmaceutical Company first marketed rebamipide as Mucosta tablets for treating gastric lesions and ulcers.

In January 2012, Otsuka launched Mucosta ophthalmic suspension (2.0% rebamipide) as a novel dry eye treatment in Japan.[5] The drug acts to increase the level of mucin in the tear film.

In July 2012, Otsuka and Acucela announced the initiation of a Phase III, multi-center, randomized, placebo-controlled, double-masked, parallel-group study clinical trial to evaluate Mucosta ophthalmic suspension in patients with dry eye syndrome.[5] The companies anticipate the trial to be completed by the end of 2013.

• ***MIM-D3***. In November 2012, Canada's Mimetogen Pharmaceuticals Inc. announced that it received a US-issued patent for MIM-D3—a first-in-class, small-molecule nerve growth factor receptor (TrkA) agonist used to treat dry eye disease. MIM-D3 is a mimetic of nerve growth factor (NGF) that binds specifically to the TrkA receptor.

NGF is a naturally occurring protein found in the eyes that is responsible for the maintenance of the corneal nerves and epithelium, mucin and tear production. NGF shows a potential benefit in dry eye disease management, including neurotrophic effects, corneal healing and mucin secretion.

In June 2011, Mimetogen completed a Phase II, randomized, double-masked, multi-center, placebo-controlled trial designed to evaluate the safety, tolerability and efficacy of MIM-D3. The trial results showed that patients exhibited improved signs and symptoms of dry eye disease as well as excellent safety and tolerability profiles.[6]

• ***RGN-259***. RegeneRx Biopharmaceuticals is developing RGN-259—a thymosin beta 4-based, preservative-free eye drop—as a novel treatment for corneal healing in patients with moderate to severe dry eye disease. Thymosin beta 4 is a naturally occurring peptide found in high concentrations in blood platelets, wound fluid and other tissues.

In June 2012, results from a Phase II trial showed that RGN-259 significantly improved several signs and symptoms of dry eye in 72 patients.[7] In separate studies, RGN-259 effectively promoted corneal healing in patients with chronic, medically unresponsive, non-healing corneal defects secondary to loss of corneal innervation (primarily associated with diabetes and herpes zoster).[8]

• ***Rivoglitazone***. Santen Pharmaceutical's rivoglitazone is a peroxisome proliferator-activated receptor gamma agonist contained in an ophthalmic solution that currently is under investigation for the treatment of dry eye. Peroxisome proliferator-activated receptors represent a group of nuclear receptor proteins that function as transcription factors that regulate the expression of genes.

Santen has initiated Phase II trials of rivoglitazone for the treatment of corneal and conjunctival epithelial disorders associated with dry eye.[9] The company's researchers believe that rivoglitazone enhances the barrier function of the corneal epithelium.[9]

During the next decade, some dry eye medications undoubtedly will receive FDA approval—despite many of the difficulties and limitations outlined above. However, the medications likely will have to be exceptional. Because dry eye disease is one of the most common ocular conditions in the United States, it should not be extremely difficult to populate new, more tightly regulated clinical trials. Because such trials could be both costly and lengthy, it would be ideal to focus upon the key variables outlined in this article—without complicating trial recruitment too extensively.

These issues notwithstanding, new and more effective therapeutic agents for dry eye disease are needed desperately. Hopefully, these recommendations will facilitate success in future FDA trials and, most importantly, provide your dry eye patients with greater relief. ■

Dr. Karpecki is the clinical research director at Koffler Vision Group in Lexington, Ky. He is a paid consultant to SARcode Biosciences, but has no direct financial interest in any products mentioned.

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• ***RX-10045***. This synthetic resolvin analog, developed by Resolvyx Pharmaceuticals, is formulated for topical application to treat dry eye disease. In early studies, RX-10045 has promoted tissue repair of human corneal epithelial cells in vitro.[10]

Resolvins, a group of lipid modulators derived from omega-3 fatty acids, are naturally occurring, small molecules that protect healthy tissue during an immuno-inflammatory response. Additionally, these lipid modulators help to resolve inflammation and promote healing, permitting inflamed tissues to return to homeostasis once the insult has passed. Currently, Rx-10045 is in Phase II testing.[10]

• ***EBI-005***. At the 2012 ARVO Annual Meeting in Fort Lauderdale, Fla., researchers from Eleven Biotherapeutics presented preclininical data on EBI-005, a topically-applied interleukin-1 (IL-1) receptor antagonist protein.[11] Targeting IL-1 is a promising therapeutic approach for the treatment dry eye, because it is a critical mediator of the inflammatory cascade associated with the symptoms of ocular surface disease.

In early December 2012, the company announced initiation of a Phase 1b clinical trial of EBI-005, and it expects to conclude this study during the first half of 2013.[12]

Dr. Mastrota is the center director at Omni Eye Surgery in New York.

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Other Potential Dry Eye Drugs in the Pipeline

- ***Cyclokat*** (0.1% cyclosporine A cationic emulsion, Novagali Pharma)
- ***Restasis X*** (0.1% cyclosporine A, Allergan)
- ***CP-690560*** (tofacitinib, Pfizer)
- ***LX214*** (voclosporin, Isotechnika Inc./Lux Biosciences Inc.)
- ***ISV-101*** (bromfenac in DuraSite vehicle, InSite Vision)

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14th Annual Dry Eye Report

A Lifetime of Dry Eye

Dry eye can strike patients of any age. Do you know the subtle signs and symptoms to look for, and the particular treatments to provide, among patients of different ages?

By Cheryl G. Murphy, OD, Contributing Editor

When you think of a "typical" dry eye patient, who comes to mind? Perhaps a post-menopausal woman? Or a computer programmer? Or a person who suffers an autoimmune disease? True, these are the classic dry eye sufferers, but patients of every age and every stage of life can be affected by dry eye.

At times, the clinical signs may be subtle and the symptoms little to non-existent. However, to ensure every patient's daily comfort, remember to keep dry eye on your checklist of differential diagnoses when examining patients in any stage of life, whether young or old.

With that in mind, let's consider dry eye in each stage of life.

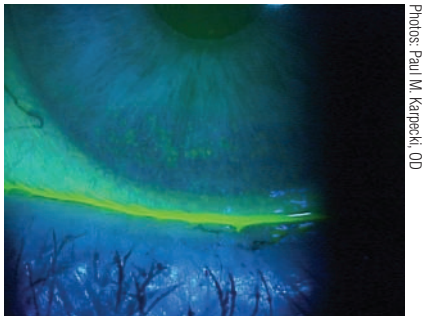
Children

We don't typically suspect dry eye in kids, but they—like patients of other ages—can suffer this daily ailment and all of its accompanying sequelae.

"A diagnosis of dry eye in children may be tricky to pick up on," because children may not readily voice their symptoms, says Ida Chung, OD, section chief of pediatrics and associate professor at SUNY College of Optometry. "The younger the child, the less likely the child will present at an exam with a verbal complaint," she says. So, "an effective eye exam on a child relies on a good history and objective findings."

Take a detailed history that asks parents about specific behaviors and signs that may reveal the child is experiencing dryness, Dr. Chung says. Common symptoms of dry eye that she watches for in kids are blepharospasm, eye rubbing, tearing (or lack of tearing), photophobia, intermittent blurred vision and (rarely) burning.

The clinical signs of dry eye in children can be similar to those in adults, including "decreased tear prism, decreased tear break-up time, punctate epithelial defects that stain with fluorescein dye, and reduced visual acuity," Dr. Chung says.



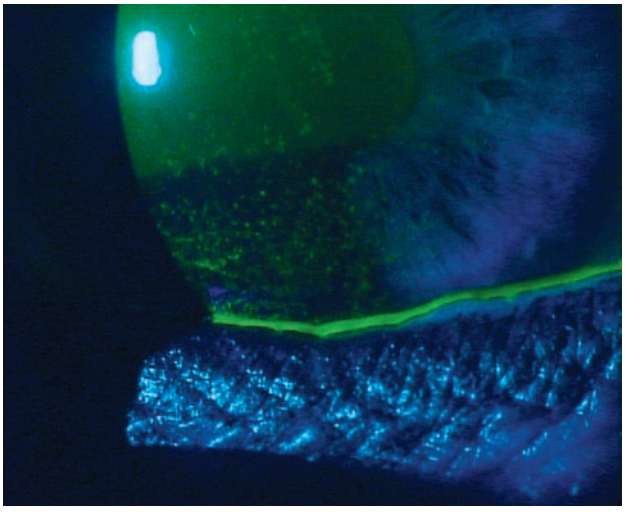
Photos: Paul M Karpecki, OD

Dryness from incomplete blinks can happen at any age.

A clinical pearl just for kids: "Look carefully at the lids, because blepharokeratoconjunctivitis is an

Tips to Examine Kids for Dry Eye

- When biomicroscopy is not possible, "use a Bluminator [Eidolon Optical], which provides pure cobalt blue light and seven times magnification, or a Burton lamp in conjunction with fluorescein dye," Dr. Chung says.
- Another less invasive alternative: "Use a head-borne magnification loupe that provides two to three times magnification and a transilluminator light source, which is better tolerated than the use of a 20D lens," she says.



Minimal tear meniscus, as seen here, indicates reduced tear film quantity, which is highly diagnostic for dry eye.

underdiagnosed condition [in children] that can result in a secondary dry eye," she says.

If dry eye is so uncommon in children, what puts certain kids at risk? "Environmental factors are the most common risk factors for evaporative dry eye for the patients that I see in New York City," Dr. Chung says. "Environmental pollutants are especially prevalent in urban areas, [and] low humidity, especially during the winter months, can put a child more at risk for dry eye."

What else is a risk factor? The use of computers and electronic devices, as well as antihistamines taken by allergy sufferers. "Environmental factors and adverse medicine side effects usually cause mild cases of dry eyes; however, when severe dry eyes are detected (severe enough to cause significant corneal compromise), or you have a case of dry eyes that is not resolving, think something else—there may be an undiagnosed genetic or systemic condition," Dr. Chung says. In those cases, rule out Sjögren's syndrome, diabetes and juvenile rheumatoid arthritis.

To treat dry eye in kids, look to the parents. "Successful treatments for dry eye in kids are usually dependent on the compliance of the parents," Dr. Chung says. She suggests using artificial tear supplements that are more viscous and require less frequent dosing, using a humidifier in the child's bedroom, treating exacerbating conditions like allergies and meibomian gland dysfunction, as well as eliminating environmental factors whenever possible, such as better visual ergonomics when using computers and electronic devices.

Teens and 20s

Like children, patients in their teens and 20s can be overlooked when it comes to a diagnosis of dry eye, says Paul M. Karpecki, OD, of Koffler Vision Group in Lexington, Ky., who frequently lectures and writes about dry eye and eye health. This oversight could be due in part to the fact that, although a teenager may present with the same dry eye symptoms as patient at an older age, they may present with fewer clinical signs.

"With age, the nerves down-regulate, and that's why you often see elderly patients with significant signs but less symptoms—and the opposite with younger patients," Dr. Karpecki says. Because fewer clinical signs present in younger individuals, a thorough history is required.

One etiology in this age group is underlying systemic disease, such as juvenile rheumatoid arthritis. This is a diagnosis that is usually made in childhood, he says, but still needs to be ruled out in teens.

Also inquire about the use of Accutane (isotretinoin, Roche), an acne medication that was taken off the market in 2009 but is still available as a generic. "What is interesting is that I've seen significant dry eyes in patients who were not currently on Accutane but had taken it years prior," Dr. Karpecki says.

Environmental Factors Contributing to Dry Eye in Teens and 20-somethings

- **Caffeine**. "Some studies have shown that small amounts of caffeine help with dry eye and act as a tear stimulant," Dr. Karpecki says. "But excessive amounts could have diuretic effects."
- **Lack of sleep**. "Sleep is a major factor for dry eye disease [in this age group], and is perhaps the main contributor," he says.
- **Alcohol consumption**. "A possible contributing factor for 20-somethings."
- **Improper contact lens wear**. "Contact lens wear usually starts in teens or early 20s, and compliance can worsen during college years, which can lead to dry eye issues."
- **Vasoconstricting topical drops**. "Many teens and college-age individuals want 'white eyes,' so they may overuse vasoconstrictors," Dr. Karpecki says. "The BAK in these agents may lead to more dry eye and ocular surface disease issues."
- **Allergies and antihistamines**. "Allergic conjunctivitis can overlap with dry eye disease," he says. "Allergic conjunctivitis is more common in this age category (as well as in children), and [to make matters worse], taking oral antihistamines can further dry the eye."

Another condition to look for: meibomian gland dysfunction. "I have to admit that I see a lot more MGD in younger patients than I had expected," Dr. Karpecki says. When he sees this in teens, one of the things he asks about is their diet. He believes there is a connection between ocular surface disease and diet, in particular essential fatty acid intake. For example, people who eat more fish also tend to have less ocular surface disease, he says.

Dr. Karpecki adds that environment and habits—like not enough sleep and too much caffeine—really play a big role in teens and young adults who suffer dry eye. (*See "Environmental Factors Contributing to Dry Eye in Teens and 20-somethings," page 62.*)

Accordingly, treatment for teens and 20-somethings with dry eye focuses "primarily on environmental modification and behavioral changes, such as getting more sleep, but don't avoid therapeutic treatments" that address the inflammatory component, he says. Dr. Karpecki gives patients who have taken Accutane as an example: "Many patients who have taken Accutane have significant ocular surface issues, so palliative treatments like artificial tears or environmental management won't help the patient as much—and the signs may even get worse because they don't address the underlying inflammation."

30s and 40s

When it comes to keeping an eye out for dry eye in patients who are in their 30s or 40s, listen for them to say, "My eyes feel dry when I wake up," or "I can't wear my contacts at the office," or "My eyes are tearing a lot," says Jeffrey Anshel, OD, president of the Ocular Nutrition Society and a frequent lecturer on computer vision syndrome.

"The most common sign of dry eye in patients in their 30s or 40s is lipid layer deficiency, which accelerates tear film breakup time," he says.

For patients in their 30s and 40s, "the most common demographic for dry eyes is women approaching menopause," because "aging changes affect the tearing levels due to hormone changes to receptors on the tear glands," Dr. Anshel says.

For women in this age range, motherhood may also bring about dry eye. "If a woman in her early 30s has a newborn, lack of sleep can be a factor as well, which is also common coupled with excessive caffeine intake," he says.

Other patients in this age group who may suffer dry eye are those who "perform hours of work on computers, which has been shown to reduce the blink rate," he says.

Treatment of dry eye in this age group can be as varied as its causes. Among the many factors to address, "the patient should be asked about drugs and any autoimmune diseases, as well as their environmental conditions," Dr. Anshel says.

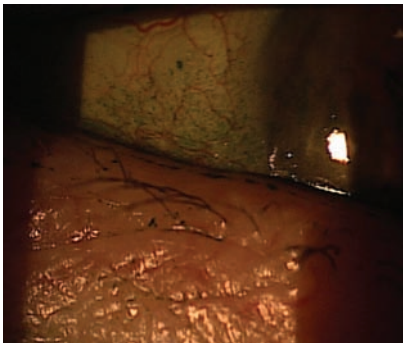
Regarding environmental conditions, he created the "20/20/20" concept for computer users to remember to take frequent visual breaks throughout the workday. (Developed 25 years ago, Dr. Anshel's 20/20/20 rule is: Every 20 minutes, take 20 seconds and look 20 feet away from the screen.)

He also suggests that doctors consider "blood testing to determine the level of nutrients in the body, especially the essential fatty acids, omega-3 and -6." Recommend omega fatty acid supplements if necessary. In addition, "our bodies don't process nutrients as efficiently as we age, so a full-spectrum multiple vitamin is usually suggested as we approach middle age," he says.

Clues for Causes of Dry Eye in 30- and 40-somethings

Dr. Anshel points to several clues to look for related to dry eye in this age range:

- **Hormonal changes:** women approaching menopause.
- **Computer work:** reduced blink rate.
- **New parent:** lack of sleep, excessive caffeine.
- **Drugs/medications:** side effects of dryness.
- **Autoimmune or other systemic disease:** cause dryness.
- **Poor nutritional habits**.



Lissamine green stain reveals devitalized cells that suggest a dry eye diagnosis.

50s and 60s

"Meibomian gland dysfunction is understood to be the leading cause of dry eye," says Caroline Blackie, OD, PhD, senior research scientist at TearScience Inc., in Morrisville, NC. And while the age of onset for MGD has typically been understood to occur between our 40s and 60s, we now know that MGD is also highly prevalent in the younger population. "Dry eye and MGD

Dry Eye Treatments in 50- and 60-somethings

Dry eye in patients of this age can be further complicated by hormonal factors and systemic disease, Dr. Blackie says. So a multi-pronged approach to treatment is frequently necessary.

- **Lubrication**. This includes artificial tears, lipid-based tears, tear gel, lubricant gel, etc.
- **Medications targeted to increase the aqueous production from the lacrimal gland**. "There is a small proportion of dry eye patients for whom aqueous production is the primary cause of their dry eye, so meds, such as Restasis [cyclosporine 0.05%, Allergan], can be helpful if used consistently for several months."
- **Topical anti-inflammatories**. Ophthalmic steroidal and non-steroidal anti-inflammatory drops may be called for to reduce acute and chronic inflammation of the ocular surface. "These meds can accelerate the healing process when used in combination with treatments that address the primary cause of the disease," Dr. Blackie says.
- **Punctal plugs**. In patients with MGD or blepharitis, don't insert plugs until after anti-inflammatory treatment. Otherwise, the plugs will retain the abnormal tears that have high concentrations of pro-inflammatory cytokines.
- **Modify environmental factors and nutrition**. "Extended hours on computers, smartphones, etc., significantly disrupt the natural blink mechanism. Lifestyle, environment and, of course, nutrition have to be a part of the ongoing conversation," Dr. Blackie says. "If the patient doesn't understand that a lifestyle approach is necessary to take control of the condition, she or he is unlikely to achieve full success."
- **Improve meibomian gland function**. Because MGD plays such a central role in dry eye and because it is a chronic, progressive and highly prevalent condition, treatment should always target improvement of meibomian gland function. Consider all and possibly a combination of high-tech, low-tech and at-home options.

in the 50- to 60-age range has most likely, although not always, progressed to a more chronic stage than in that of a younger patient," she says.

Among these older, more chronic patients, "MGD usually presents with some obvious signs," Dr. Blackie says, such as "telangiectasia, epithelial overgrowth and changes to the line of Marx (the mucocutaneous junction)." However, MGD can present in the older age range with no observable clinical signs at the slit lamp, with the exception of reduced meibomian gland function.

Certain factors that put that 50-plus age range at greater risk for MGD and dry eye, as compared to younger patients, include age, chronicity of disease, hormonal changes (particularly for women), polymedicine and increased systemic disease(s), Dr. Blackie says.

Additional influences—such as nutritional, environmental and behavioral factors—are more patient specific and somewhat less age specific, she says.

Treatment for dry eye in this age group tends to follow the same guidelines as for treatment of dry eye at other ages. Because MGD is obstructive in nature, treatment is directed toward removing the blockage and improving meibomian gland function. In her view, the most high-tech method is TearScience's LipiFlow, she says. But other approaches include manual physical expression of the glands, in-office lid margin debridement (in combination with gland expression), intraductal probing of the meibomian glands with Maskin microprobes (Rhein Medical), along with concurrent at-home therapies of lid scrubs, warm compresses and/or commercial heat packs.

If infection is suspected to be a significant contributing factor to a patient's MGD, consider prescribing oral antibiotics because "the anti-inflammatory properties of the antibiotics have been shown to accelerate improvement in MG function," Dr. Blackie says.

However, she emphasizes that gland evacuation is the best place to start when treating MGD and restoring the proper functioning of the glands.

70s and 80s

When patients reach these later decades of life, they are likely on multiple medications. "Dry eye can be made worse by medications patients are using for chronic conditions," says Uyen Dao, OD, who works at Northport VA Medical Center, in Long Island, NY. "Such medicines include diuretics, anticholinergics (such as tricyclic antidepressants and antipsychotics), antihistamines, antispasmodics and antiparkinson medications—and these can worsen dry eye," she says.

Dr. Dao also regularly performs on-site nursing home eye examinations through a private practice. "Androgen deficiency in postmenopausal women and decreased testosterone levels in older men can lead to MGD and dry eye in these patients," she says.

Furthermore, certain medical conditions that are more common in the elderly—including diabetes, rheumatoid arthritis, lupus, vitamin A deficiency, thyroid disorders, radiation therapy and Sjögren's syndrome—are also known to be associated with dry eye.

In particular, patients with Sjögren's syndrome are known to

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suffer from dry eyes and dry mouth caused by chronic inflammation of the mucous membranes. In the eye, this leads to improper functioning of the lacrimal gland and an overall decreased production of tears (which can be confirmed with a Schirmer's test), Dr. Dao says. Primary Sjögren's syndrome occurs alone (without accompanying disorders), while secondary Sjögren's occurs in conjunction with autoimmune disorders such as lupus or rheumatoid arthritis.

Patients with Sjögren's syndrome may go undiagnosed for years. So, if you suspect Sjögren's, comanagement with the patient's primary care physician or referral to a rheumatologist or immunologist is in order.

Dry eye treatments for Sjögren's includes topical lubricants, tear-conserving strategies (such as punctal plugs), slow-release lubricant vehicles (such as Lacrisert [hydroxypropyl cellulose, Valeant]) and Restasis (cyclosporine 0.05%, Allergan).

The treatment regimen of non-Sjögren's dry eye in patients in their 70s and 80s is similar to that of patients in other age groups; however, it may be easier for patients in a nursing home or hospital to adhere to a dosing schedule because it can be organized and administered with the help of health professionals, Dr. Dao says.

Instruct patients to use artificial tears during the day and to apply an ointment at night. Omega-3 supplements may be prescribed and a humidifier can be used during dry winter days and also during the summer when the air conditioning is running constantly and the humidity is low.

A patient who presents with concomitant blepharitis should be treated with lid scrubs, warm compresses and antibiotic ointment.

No matter what age or stage, dry eye is a condition that can significantly affect a patient's quality of life and interfere with daily comfort. Even when there are few to no symptoms present, if conditions that can provoke dryness are left untreated, the patient will ultimately suffer. Remember to look for the root of the problem for dry eye at any age—if one can identify and target treatment toward eliminating the underlying cause of the dry eye, it will stand a better chance of being successfully alleviated.

It is the practitioner's job to recognize the signs and symptoms of dry eye and to provide the education and the customized treatments needed to preventatively and proactively manage this condition in each decade of our patients' lives. ■

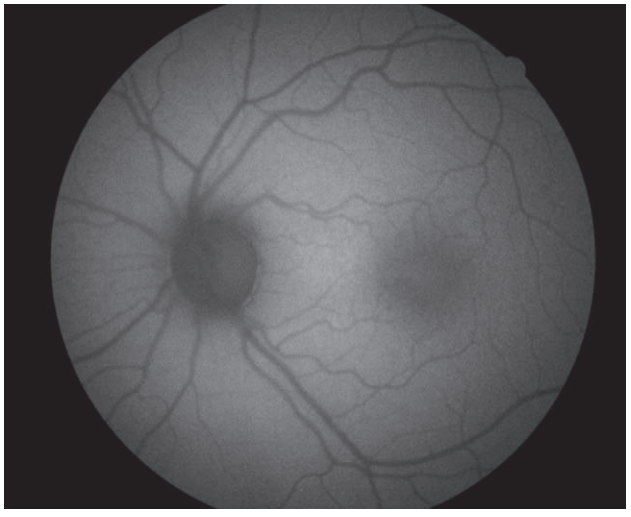
Fundamentals of Fundus Autofluorescence Imaging

New imaging technology reveals a biomarker of retinal disease progression that's not visible to the clinician's eye. **By Khadija Shahid, OD**

Lipofuscin (LF) is a byproduct of phagocytosed photoreceptor outer segments that accumulates in the retinal pigment epithelium (RPE) with age as well as in certain retinal diseases. When exposed to short- to medium-wavelength visible light, LF will autofluoresce. Fundus autofluorescence (FAF) imaging takes advantage of the autofluorescent properties of LF to document its accumulation. The FAF imagery can then be used to predict patterns of disease and progression and can lead to better understanding of disease pathogenesis.

By bridging FAF with additional imaging technologies—including digital red, green, blue (RGB) monochromatic filters, topographical emboss filters and image registration technology, along with high-resolution optical coherence tomography (OCT)—optometrists can further evaluate the ocular fundus in detail to:

- Track temporal changes in LF distribution.
- Detect earlier certain retinal disorders related to LF accumulation.
- Assess risk factors that may affect LF accumulation in the fundus.
- Aid in differentiating diseases using specific LF accumulation patterns.



1. FAF image of a healthy eye: Note characteristic dark, hypofluorescence of the optic nerve and blood vessels as well as the hypofluorescence of the fovea.

The use of these advanced imaging systems offer primary care optometrists further understanding of retinal disease pathogenesis, and may aid in counseling of preventative steps for enhanced patient management.

Release Date: January 2013
Expiration Date: January 1, 2016
Goal Statement: The evolution of ophthalmic imaging has coincided with, and aided, the management of advanced ocular disease. Fundus autofluorescence (FAF) imaging is a relatively new technology that, along with other diagnostic tools, can help the clinician acheive a better understanding of the health of the fundus in order to obtain earlier diagnoses and better predict progression of certain retinal diseases. This course explains how FAF works, and how its results are interpreted, for a variety of retinal disease conditions.
Faculty/Editorial Board: Khadija Shahid, OD
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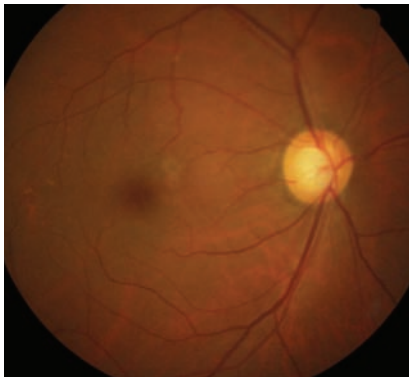
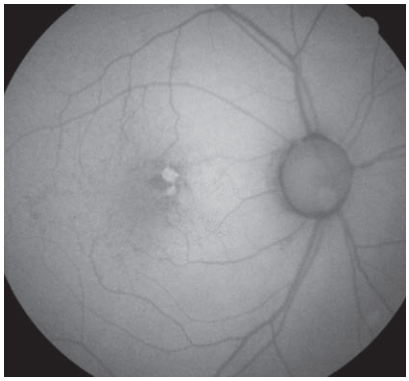


2a. Color fundus photo with pigment changes superior nasal to fovea and drusen temporal to fovea.



2b. FAF image with two focal hyperfluorescent areas correlating to color change in fundus photo. Adjacent hypofluorescence suggests RPE death in an otherwise benign-looking fundus.

Imaging History

Retinal photography has evolved rapidly since its inception in 1959 using an electronic flash tube and 35mm black-and-white film-based fluorescein angiography. By the 1980s, digital retinal imaging became available using charged couple device (CCD) light sensors. More sensitive CCDs, as well as wavelength extraction and digital processing software, have created a revolution in ocular digital imaging.

The 1990s saw the introduction of OCT technology. As high-resolution scanning laser ophthalmoscope (SLO) OCT became more accessible, so did optimal, advanced management of ocular disease.

In the past decade, a new addition to the imaging front is the use of fundus autofluorescence technology.

What is Lipofuscin?

Lipofuscin is a biomarker evident in normal aging and in chronic disease. Its accumulation has been detected in various tissue and lesions associated with neurodegeneration (Parkinson's, Alzheimer's, etc.), nutritional cirrhosis, cardiac failure and RPE degeneration underlying retinal disease, among many other conditions.[1]

This accumulation is evident in ocular disease even before the visual cycle begins to degrade, supporting the theory that LF accumulation can be viewed as an early marker of certain retinal degenerative processes.[1,2]

How Do We Image Lipofuscin?

RPE lipofuscin deposits are visual pathway byproducts with unique autofluorescent properties that can be detected and quantified using imaging devices such as the fundus spectrophotometer, confocal scanning laser ophthalmoscope (cSLO) and FAF camera systems—with the latter two being the most commonly used clinical instruments.[3] Because the approximate spectrum range of LF (wavelength range: 300nm to 600nm) is close to the visible spectrum of light (wavelength range: 400nm to 700nm), these clinical instruments can use visible light to elicit an emission, and safely detect LF in vivo during a routine clinical examination.[1,3]

• ***cSLO.*** The cSLO systems elicit and capture the RPE LF response by using a low-energy laser to excite LF, and a barrier filter to allow only the RPE LF response to pass. The cSLO can perform as many as 30 scans, which are then averaged together using post-processing software.[4] The final result is a single, high-contrast, monochromatic image. The confocal optics and scanning laser help to bypass most of the anterior autofluorescence—for example, in an aging lens—which could interfere with posterior pole imaging.

• ***FAF camera.*** Alternately, a FAF retinal camera system uses a high-energy white flash (300 watt-seconds) and a wideband exciter filter to penetrate ocular media, reach deep within the RPE, and excite any existing LF. The LF response is then able to pass through a wideband barrier filter before reaching the sensor of the retinal camera. Similar to cSLO systems, the result is a single monochromatic image that reveals either the presence or absence of LF. The difference, however, is that FAF images from the retinal camera are not averaged—rather, a single image is captured in real time.

It's important to note that FAF results from both systems have quality limiting factors. As mentioned previously, ocular media opacities, such as an aging lens, can alter or negatively affect FAF posterior pole image results. Additionally, there is neither a uniform protocol (correction of patient refractive error, vertex distance, etc.), nor a standardized manufacture setting that dictates excitation and barrier filter wavelength setting or image processing techniques.

While all FAF systems require digital processing to create the final result, it's important to remember that FAF imaging provides quick, non-invasive access to information related to the health of RPE cells in relationship to LF.

Interpreting FAF results

FAF imagery used to detect and track changes in RPE LF must be interpreted appropriately to best understand ocular health status and to convey this to our patients.

Visually, FAF imagery resembles a fluorescein angiogram (FA) study in that results are represented by a 256 grey-scale value. Low pixel values represent low fluorescent intensities and appear dark, or hypofluorescent. Alternately, high pixel values appear bright, or hyperfluorescent. Unlike FA studies, where signal intensity is determined by circulation, the FAF signal is dependent solely on the presence of autofluorescent material (i.e., LF). Increased concentrations of LF result in very bright, hyperfluorescent signals. Conversely, in the absence of LF, signals appear dark (hypofluorescent).

FAF imaging has been used in healthy subjects as young as two years old. The posterior pole of a healthy ocular fundus has an overall diffuse, mildly hyperfluorescent signal due to the normal levels of LF present in RPE cells. The optic nerve head always appears dark (hypofluorescent) due to the absence of RPE and LF (*figure 1*). Other structures that appear dark are retinal blood vessels (due to signal absorption from blood) and the fovea (due to signal absorption from high densities of macular luteal pigment).

The posterior pole of an unhealthy ocular fundus will have areas of abnormal signal densities (*figures 2a,b*). This could include hypofluorescent signals such as those seen with RPE atrophy and cell death, fresh hemorrhages, exudative lesions, areas of dense hyperpigmentation, and some forms of hard drusen. It also could include hyperfluorescent signals, such as those seen with abnormally high concentrations of RPE LF; for example, visible yellow lesions associated with lipofuscinopathy diseases (Best's, Stargardt's, etc.) are often intensely bright on FAF imaging due to abnormally high levels of LF. Examples of a more mild hyperfluorescent FAF signal could include older hemorrhages (due to fluorphore buildup within the stagnant blood), large, confluent, soft drusen, and basal laminar or reticular drusen that have a unique fluorescent pattern.

When comparing FAF patterns to the corresponding color image patterns of ocular disease, there can be large variability in the findings. The Fundus Autofluorescence in Age-related Macular Degeneration (FAM) Study described these variables in patients during an

FAF Phenotypes in Early AMD[5]

Classification Pattern Name	FAF Result	Color Image Result
Normal	Consistent with a healthy fundus, the posterior pole appears diffusely hyperfluorescent with gradual decreased intensity toward the fovea.	May or may NOT show visible soft or hard drusen.
Minimal change	Minimal irregular background hyper- or hypofluorescence, similar to normal pattern.	May or may NOT show visible soft or hard drusen.
Focal increase	One or more defined hyperfluorescent spot(s) WITHOUT surrounding halo. May or may not have additional defined spots with dark halo.	Multiple hard and soft drusen.
Patchy	One or more less defined hyperfluorescent area(s) with a diffuse halo.	May or may NOT show focal hyperpigmentation or drusen.
Linear pattern	One or more well defined hyperfluorescent line(s) WITHOUT surrounding halo.	Corresponding lines of hyperpigmentation.
Lacelike pattern	Several branching, less defined hyperfluorescent lines that form a lace pattern, with mild diffuse halo.	May or may NOT show corresponding hyperpigmentation lines.
Reticular pattern	Several small areas of poorly defined, hypofluorescent patterns, more commonly noted superior temporal to the fovea.	May or may NOT show numerous small soft or hard drusen, or pigmentary changes.
Speckled pattern	Large area of various hyper- and hypofluorescence that extends beyond the macula, possibly through the entire posterior pole. May appear punctate or linear.	Large areas of pigmentary hypertrophy and atrophy with multiple confluent drusen.

international workshop on FAF phenotyping for early AMD. Subjects were classified into one of eight different phenotypes based on different patterns of autofluorescence.[5] (*See "FAF Phenotypes in Early AMD," page 69.*) The classification scheme illustrates the wide diversity of FAF patterns that are present in just one single disease: early AMD.

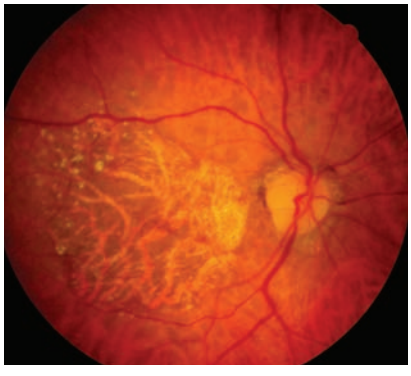
Among the important conclusions of this study: Visible alterations seen on color fundus photography were often poorly correlated with FAF imaging, and these FAF pattern differences were likely indicative of disease progression not yet visible to the clinician's eye. (*See "FAF and Color Fundus Images in Ocular Pathology," below.*) In other words, FAF findings could represent an independent measure of disease activity.[6]

Let's look at the use of FAF for different disease conditions.

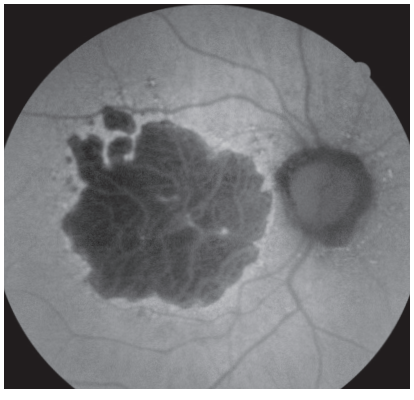
• ***AMD.*** Comparisons of FAF patterns and color imagery in late-stage, dry AMD with geographic atrophy (GA) also demonstrate a variety of FAF phenotypes that are not evident on color fundus photography or other imaging methods (*figures 3a,b*).[7] A classification scale developed by the FAM Study group (*See "FAF Phenotypes in Late AMD," page 71*) relates to FAF patterns at the junctional zone—the area that encompasses the border between the unaffected retina and the edge of a GA lesion. Those cases where more active, larger and more diffuse hyperfluorescent lesions were

FAF and Color Fundus Images in Ocular Pathology

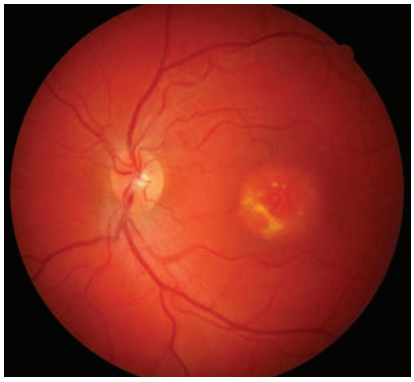
Ocular Pathology	FAF Pattern	Correlation with Color Image Pattern
Choroidal neovascularization	Various patterns of hypofluorescence correspond to hemorrhages, exudates and atrophy. Hyperfluorescent areas correspond to active LF and RPE proliferation.	FAF signal typically extends beyond the edge defined by color fundus image and fluorescein angiography.
Disciform scars, late-stage AMD	Hypofluorescent signal. Some scars show an increased FAF signal at the junctional zone.	Border and extent of scarring is more visible with FAF than color fundus photo.
Stargardt's, Best's, other vitelliform and lipofuscinopathy disease	Hyperfluorescence ranging from intense to moderate pending the LF concentration. Hypofluorescent areas in end-stage and with RPE atrophy.	Visible yellowish flecks on color image correlate well to focal areas of hyperfluorescence. RPE atrophy is not initially visible with color image.
Central serous chorioretinopathy	Acute: hyperfluorescent areas in the active stages. Chronic: hypofluorescent areas.	Acute: FAF highlights edge of detachment that may or may not be visible on color image. Chronic: RPE cell death not visible on color image.
Benign and malignant choroidal lesions	No significant fluorescence. Subtle hyperfluorescence when LF is present over lesion.	Visible LF lesions correlate with FAF hyperfluorescence.
Panretinal photocoagulation	Mostly hypofluorescent signal (RPE destruction), followed by weak hyperfluorescence due to RPE proliferation/scar formation.	Laser scars correlate with hypofluorescence.
Glaucoma	Some show hyperfluorescence in the parapapillary region surrounding the optic nerve.	No visible correlation.



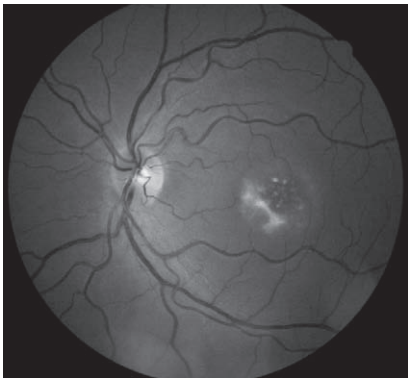
3a. Color fundus image of a patient with extensive geographic atrophy.



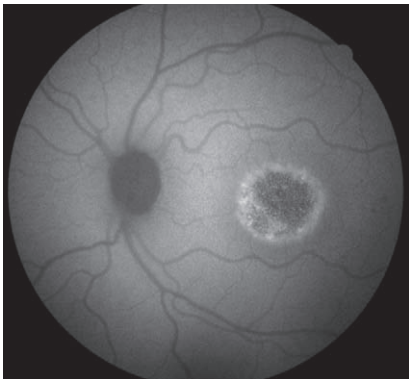
3b. FAF demonstrates dense hypofluorescence. Note the hyperfluorescence at the junctional zone, with a banded pattern.



4a. Color fundus photo of a 33-year-old female with Best's disease. Her best corrected vision is 20/40. Note the yellow deposits in the central macula.



4b. RGB image with green (red-free) filter highlights the retinal layer and the macular deposits.



4c. FAF image demonstrates hyperfluorescence correlating with yellow deposits. Diffuse hyperfluorescence at lesion edge demonstrates the extent of damage and possible future progression.

noted at the junctional zone were more likely to progress over time than those with absent or hypofluorescent lesions at the junctional zone. The progression of GA lesions seemed to be more dependent on the FAF pattern at the junctional zone than any other risk factor being monitored—including size of baseline atrophy, history of smoking, hypertension, diabetes, age, hyperlipidemia and family history.[7] This may help our understanding of unpredictable prognoses in patients with AMD who present with similar baseline clinical findings, yet progress at quite dissimilar rates over the course of their disease. FAF imaging is likely revealing differences at cellular levels that prove these patients are not as similar as we were led to believe with traditional imaging technology.

• ***Retinal dystrophies.*** In other ocular disease, FAF demonstrates variances that may or may not correlate with color fundus imagery. However, a common theme is that the FAF results are indicative of RPE changes occurring on a molecular level that may be precursors to visible, clinically evident disease progression. For example, the evaluation of a patient with Best's disease demonstrates clinically evident, yellow-orange LF retinal lesions (*figures 4a-c*). When comparing the appearance of these lesions on color fundus photos to FAF imagery, there is good correlation between the LF deposits (seen on color image) and bright, hyperfluorescent areas (on FAF image). Sub-clinically, however, FAF shows extensive mottling of hypofluorescence in the macula

FAF Phenotypes in Late AMD[7]

Classification Pattern Name	FAF Result	Description and Prognosis
No Evidence of Hyperfluorescence at Junctional Zone		
None	Hypo- or no fluorescence.	Slow progression of GA lesion.
Evidence of Hyperfluorescence at Junctional Zone		
Focal	Single, small spots of hyperfluorescence at GA border.	Slow progression of GA lesion.
Banded	Almost continuous hyperfluorescent ring around GA lesion.	Rapid progression of GA lesion.
Patchy	Homogenous area of moderate hyperfluorescence at and adjacent to GA border.	Rare occurrence, poor data on progression.
Diffuse	Hyperfluorescent area at and adjacent to border in various patterns, including fine granular, branching, punctated spots, trickling and reticular.	Rapid progression of GA lesion.

where there is RPE cell death, and additional areas of hyperfluorescence at the borders of the lesion, suggesting where the disease may progress in this example.

• *Glaucoma*. FAF patterns in patients with suspected and advanced primary open-angle glaucoma, normal-tension glaucoma, pseudoexfoliative glaucoma and even ocular hypertension have all shown evidence of hyperfluorescence in the parapapillary region of the optic nerve head. In some cases, the amount of hyperfluorescence has been correlated to the severity of the disease, with increasing hyperfluorescence associated with more advanced glaucoma. Histology studies confirm the presence of significant LF accumulation within RPE cells in this region, which may signify degeneration not yet clinically evident.[8]

• *Choroidal lesions*. In cases of choroidal lesions, optometrists are trained to look for several factors that help to differentiate nevi from melanomas and determine the likelihood of growth. These include the presence of subretinal fluid, lesion thickness, visual symptoms, proximity to the optic nerve head, and presence of LF on the surface of the choroidal lesion. FAF imagery has been used to assist in the detection of subtle LF, especially in less visible, deep or amelanotic lesions. Serial FAF imagery of the choroidal lesion has also been used to monitor for changes in LF that indicate a possible tumor growth.

The presence of LF may be found on both choroidal nevi and melanomas; however, a nevus more commonly presents with patchy, distinct hyperfluorescent patterns (*figures 5a,b*), whereas melanomas can present either as patchy or as a diffuse pattern of hyperfluorescence with less distinct borders covering at least 50% of the lesion.

Advanced Posterior Pole Imaging

Autofluorescence technology can be used in conjunction with existing posterior pole imaging techniques to provide a more complete clinical picture. For example, color fundus photography, software-assisted RGB filters, emboss filters and OCT (which are discussed below) all provide valuable information about the overall assessment of each patient case.

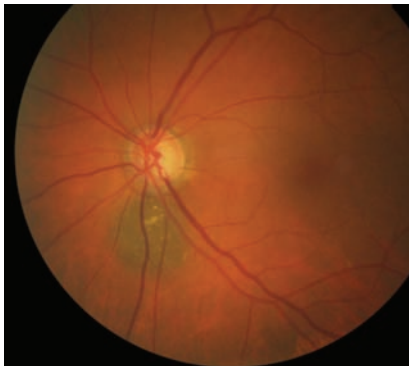
For example, RGB filters originate from a raw (untouched by the camera's co-processor and with full pixel resolution) digital fundus-camera color image that is composed of three color channels of varying wavelength: red (25%), green (50%) and blue (25%). When isolated, each filter becomes a further study of ocular structures within a specific layer of the posterior pole.[9]

By isolating the blue channel (wavelength 490nm to 510nm), the resulting image highlights the superficial nerve fiber layer (NFL) for visualization of dropout, for differentiation of a cotton-wool spot (NFL infarct) from a druse, and to better visualize cup-to-disc ratio.

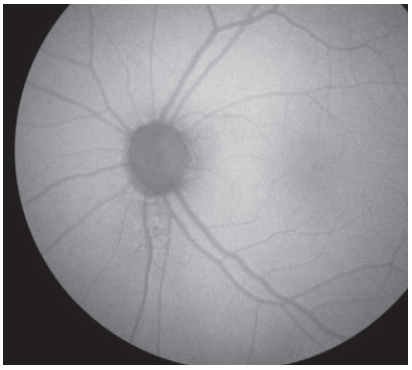
Isolating the green channel (530nm to 550nm) results in high-contrast imagery that highlights retinal structures including retinal hemorrhages or exudates (differentiated from choroidal drusen). Similar to conventional red-free images, the green layer is most helpful in the assessment of vascular disease such as diabetic retinopathy, or artery and vein occlusions.

Finally, when isolating the red channel (wavelength 590nm to 610nm), the result highlights the choroidal layer and allows for choroidal vasculature, RPE and drusen evaluation. The red layer is helpful when studying AMD or when differentiating a flat nevus limited to the choroid from a thick, growing melanoma that has invaded into the retina.

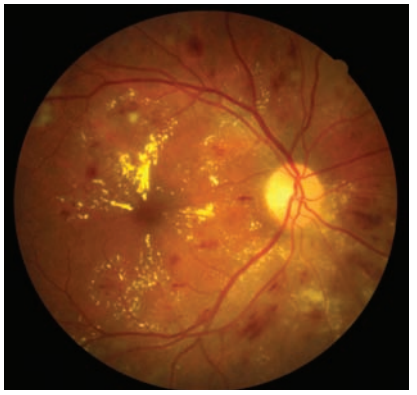
Additionally, an emboss filter is used to address the lack of depth perception when evaluating single-image, digital retinal photos. While stereo imaging can provide some aspect of depth perception, it involves a learned technique and serial imaging. This is one reason why imagery does not substitute for dilated fundus evaluation in which optometrists can view the fundus in stereo, determine ocular health and document pertinent findings. Rather, imagery enhances the information optometrists gather and allows for concise documentation. It is possible to create an embossed, topographical image from a single-color fundus photo or a single RGB channel



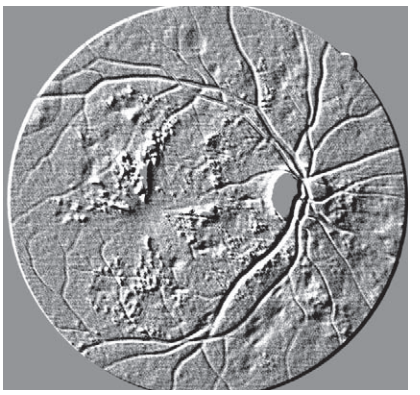
5a. Color fundus image of choroidal nevus with overlying drusen.



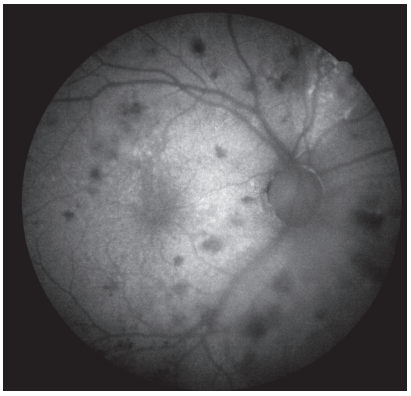
5b. FAF demonstrates minor hyperfluorescent lesions in a patchy pattern, which suggests little to no lipofuscin is present.



6a. Color fundus image demonstrates severe diabetic retinopathy.



6b. Emboss demonstrates significant elevation where the retinal exudate is present.



6c. Areas of hypofluorescence on FAF image correlating with retinal hemes.

image because color images are typically represented by a depth range from eight to 24 bits (*figures 6a-c*).[10] Emboss images create a pseudo-stereo effect with three-dimensional-like representation of elevations or depressions within the patient's posterior pole. The greater the bit depth (z axis) of an image, the higher its resolution and the more information the image yields.

All of the above techniques can be incorporated with image registration patterns and fade-in, fade-out technology, which allows serial images to track any progressive changes over time, and which can be viewed simultaneously over each other to correlate color images, RGB filtered layers, and topographical alterations—all from a single image. By incorporating autofluorescence, one can study underlying posterior pole changes simultaneously—in essence "peeling away" the different layers to investigate what findings lie in which layer and what might possibly become future pathology.

The final piece to advanced posterior pole technology would be the incorporation of high-resolution spectral domain OCT. This non-invasive tool can complete up to 70,000 A-scans per second to create detailed, cross-sectional imagery of the retina, or even anterior segment structures. The OCT has essentially provided the practicing clinician with a microscope capable of high-resolution, histologic views of ocular anatomy in vivo during the course of routine clinical examinations.

One of our main goals as primary eye care physicians is to detect, at the earliest possible point, any degenerative changes that could lead to ocular dysfunction. In an effort to step outside of traditional practice, much of our energy should be directed toward not only the accurate identification, documentation and management of eye disease—which is greatly enhanced with advanced posterior pole technology—but also toward preventative eye care. It is important to educate patients on healthy lifestyle practices and proper nutrition for the best support of ocular health. The use of FAF technology can play an important role in demonstrating pending degenerative changes to the fundus for our patients.

Evidence of changes at the level of the RPE in LF distribution that is demonstrated with FAF imagery has been correlated with early pathology that may not yet be clinically visible. Evidence continues to support the concept that excessive accumulation of LF in the RPE can lead to cellular destruction, retinal aging and visual degeneration. As technologies refine their presence in our practice, we are able to better understand pathology, detect possible at-risk patients earlier, and direct counseling and preventative health care even sooner. This will undoubtedly improve patient care if earlier steps in disease intervention can save or slow the natural progression of ocular disease as we see it today. ■

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OSC QUIZ

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1. Lipofuscin is a byproduct of aging and disease that is most abundant in which ocular structure?
a. Iris.
b. Retinal blood vessels.
c. Optic nerve.
d. Retinal pigment epithelium cells.

2. When exposed to short- to medium-wavelength visible light, lipofuscin will:
a. Autoregress.
b. Autotomize.
c. Autofluoresce.
d. Autolysate.

3. Excessive lipofuscin accumulation can lead to what type of damage?
a. Neurodegeneration.
b. Aneurysm.
c. Retinal detachment.
d. Uveitis.

4. What diagnostic technology is more commonly used to detect lipofuscin in a clinical setting?
a. Fundus autofluorescence camera.
b. Fundus spectrophotometry.
c. Confocal scanning laser ophthalmoscope.
d. Both a and c.

5. Which statement about FAF imaging is true?
a. The wavelength required to excite lipofuscin in the retinal pigment epithelium is in the approximate range of 300nm to 600nm.
b. Visible light can be used to elicit a response from lipofuscin in the retinal pigment epithelium layer to detect lipofuscin in vivo.
c. Confocal scanning laser ophthalmoscope (cSLO) takes a single scan to image and process fundus autofluorescence (FAF) images.
d. Manufacturers of FAF systems use a standardized protocol to dictate excitation and barrier filter wavelength settings.

6. The cSLO bypasses anterior autofluorescence in an aging lens by using:
a. Confocal optics.
b. High-energy (300 watt-seconds) white flash.
c. High-energy laser to excite lipofuscin.
d. RGB filters.

7. The signal intensity of an FAF image is dependent solely on:
a. Blood circulation.
b. Blood concentration.
c. Autofluorescent material.
d. The nerve fiber layer.

8. In the case of a healthy optic nerve, what would you expect to see with FAF imaging?
a. Hypofluorescence of the optic nerve head.
b. Hyperfluorescence of the optic nerve head.
c. Evidence of increased lipofuscin accumulation.
d. A bright, diffuse signal.

9. Which statement about the interpretation of FAF imaging results is true?
a. Increased concentrations of lipofuscin result in very dark signals.
b. Hyperfluorescence is used to describe decreased or absent concentrations of lipofuscin.
c. Hyperfluorescent signals can be due to abnormal, increased concentrations of lipofuscin.
d. Decreased concentrations of lipofuscin can result in very bright signals.

10. Which ocular structure appears brightly hyperfluorescent on FAF imaging?
a. Optic nerve.
b. Retinal blood vessels.
c. Yellow deposits seen in active Best's disease.
d. A healthy fovea.

11. An important conclusion of the Fundus Autofluorescence in Age-related Macular Degeneration (FAM) Study was:
a. Visible alterations seen on color fundus photography often correlate well with FAF imaging.
b. FAF patterns were likely to be indicative of disease progression not yet visible clinically.
c. Subjects with early age-related macular degeneration could be classified into three phenotypes based on FAF patterns.
d. The classification scheme based on FAF patterns illustrates the lack of diversity of FAF patterns in early AMD.

12. The progression of geographic atrophy lesions has been largely associated with several risk factors, including:
a. FAF pattern at the junctional zone.
b. History of pregnancy.
c. Gender.
d. History of glaucoma.

13. According to the FAM Study on late AMD, geographic atrophy with diffuse hyperfluorescent lesions at the junctional zone is more likely to:
a. Progress over time.
b. Unknown.
c. Not progress over time.
d. Convert to wet AMD.

14. Some patients with primary open-angle glaucoma, normal-tension glaucoma, pseudoexfoliative glaucoma and ocular hypertension have shown evidence of which of the following with FAF imaging?
a. Hypofluorescence in the parapapillary region of the optic nerve.
b. Hyperfluorescence in the parapapillary region of the optic nerve.
c. Diffuse hyperfluorescence of the entire optic nerve head.
d. Both a and c.

15. FAF imaging can best assist in differentiating a choroidal nevus from a melanoma by:
a. Identifying the presence of subretinal fluid.
b. Gauging the thickness of the lesion.
c. Assessing visual symptoms.
d. Identifying the presence of LF on the surface of the lesion.

16. In the case of a choroidal nevus, what FAF pattern presents most commonly?
a. Patchy, distinct hyperfluorescence.
b. Diffuse hyperfluorescence that covers at least 50% of the lesion.
c. Hypofluorescence with less distinct borders over the majority of the lesion.
d. Diffuse hyperfluorescence at the borders of the lesion.

OSC QUIZ

17. What wavelength range is associated with the blue channel of RGB filter software?
a. 390nm to 410nm.
b. 490nm to 510nm.
c. 530nm to 550nm.
d. 590nm to 610nm.

18. What posterior pole structure is best highlighted by the green (red-free) channel of a color image?
a. Nerve fiber layer.
b. Choroidal layer.
c. Retinal layer.
d. Retinal pigment epithelium layer.

19. A topographical, pseudo-stereo image created from a color fundus photo or from a single RGB channel is known as:
a. Optical coherence tomography.
b. Emboss.
c. RGB channel image.
d. Color fundus photo.

20. Which statement about high-resolution spectral domain optical coherence tomography is true?
a. It can take up to 70,000 A-scans per second.
b. It can create a cross-sectional image of the retina.
c. The OCT scan is composed of three color channels.
d. Both a and b.

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7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
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9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
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13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
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17.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
18.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
19.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
20.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

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'Can You Spare a Sample, Doc?'

You don't want to deny your patients the drugs they need. But at the same time, you don't want to hand out samples like candy. **Edited by Paul C. Ajamian, OD**

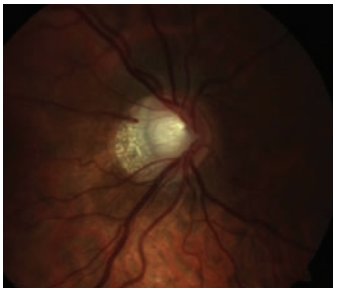
Q **I have a lot of patients asking me if I can "spare any samples" of brand-name glaucoma drugs. How do you handle that?**

A "If the patient specifically requests it, appears in dire need and is not able to get the medicine for a sight-threatening condition, then I will provide a sample," says Robert Pinkert, OD, of Barnet Dulaney Perkins Eye Center in Phoenix.

But this is not what samples are really meant for, Dr. Pinkert admits. "We usually provide a free sample as an initial trial of the drug—especially in a chronic condition like glaucoma—rather than sending the patient to buy a $100 bottle to see if it works," he says. "If it doesn't work, we try something new."

Newly diagnosed patients often misunderstand this. "They may think, 'You gave me the first bottle for free, why not just give me another one?'" Dr. Pinkert says. In this case, "I generally say, 'The samples are reserved for patients who need them on a trial basis. Your insurance should cover most of the cost of your prescription.'"

Keep in mind that the question "Do you have any samples?" is often the patient's way of saying, "I can't afford the medication you just prescribed." So if the patient's insurance does not cover the drug—or the patient doesn't have insurance—this should prompt you to help the patient get on a patient assistance program or to prescribe a less expensive generic drug, Dr. Pinkert says.



The price of the drug has everything to do with compliance.

- ***Patient assistance program.*** Each of the pharmaceutical companies that manufactures glaucoma medications—Alcon, Allergan, Merck and Pfizer—has an assistance program to help indigent patients afford the drugs they need. Usually, your office needs to be the advocate to get the patient on the program. (A list of these and other programs can be found on the Glaucoma Research Foundation's website: www.glaucoma.org/treatment/financial-assistance-and-social-services.php.)
- ***Generic medication.*** "For most patients, I ask them if they would prefer a generic or a brand," Dr. Pinkert says. "In most cases, they're equivalent for garden-variety disorders." (However, in the case of something like a sight-threatening ulcer, stick with the brand, he says. Also, some patients insist on a brand-name drug, and are willing to pay the difference.)

This begs the question: Are generic drugs equivalent to branded drugs? "The answer is, in many cases, they are," Dr. Pinkert says. "But, in some cases, they're not."

Generic ophthalmic solutions—such as generic latanoprost, for example—are expected to have both the same active and inactive ingredients, and in the same concentrations, as the brand-name counterpart. That's OK if you've prescribed Xalatan (latanoprost, Pfizer) for your glaucoma patient. But what if you've prescribed Lumigan (bimatoprost, Allergan) or Travatan Z (travoprost, Alcon)? "Are you really giving the patient the same drug?" Dr. Pinkert asks. "No, you're not—but as a class they work very similarly in most patients. So, the clinical effect is about the same."

In other words, the doctor must often weigh the clinical effectiveness vs. the cost, which is a major factor to compliance, Dr. Pinkert says. "And in chronic care, when the patient is going to be on the drops for many years and the cost is ongoing, we want the best outcome at the lowest cost with the fewest side effects." So, for most patients, generics offer a fair compromise. ■

'This Little Bottle Costs HOW MUCH?!'

Patients are often surprised to find out that a tiny bottle of glaucoma drops can cost $100 or more. Help patients find the least expensive prescription in town by pointing them to a site like GoodRx.com. Type in the name of the drug and your zip code, and up pops a list of nearby brick-and-mortar as well as mail-order/online pharmacies, along with how much each charges for that little drug bottle.

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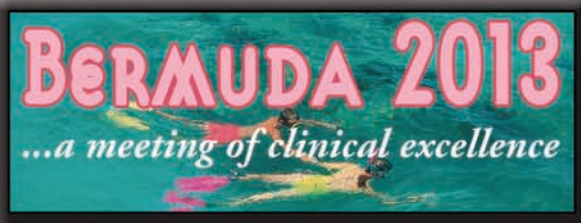

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Artificial Cornea Intelligence

This expert advice can help you reduce complications in patients with keratoprostheses.

Edited by Joseph P. Shovlin, OD

Q One of my patients has had multiple corneal graft failures and is now considering the Boston type 1 keratoprosthesis. What are some of the issues commonly encountered with this device, and do you have any tips on how to reduce such complications?

A The Boston type 1 keratoprosthesis, or KPro (Massachusetts Eye and Ear Infirmary), has undergone many upgrades in recent years, which have improved clinical outcomes and increased its use.[1] "The main complications are difficulty in long-term management and glaucoma issues," says Christopher J. Rapuano, MD, director of the Cornea Service Department and co-director of the Refractive Surgery Department at the Wills Eye Institute in Philadelphia.

Due to these risks, the patient will require rigorous, lifelong follow-up; however, keratoprostheses can produce good outcomes if the eye care provider stays vigilant and the patient remains compliant. "There are easily avoided minor complications in individuals who have had graft failures or who have normal ocular surfaces," says James Aquavella, MD, professor of ophthalmology at the University of Rochester Flaum Eye Institute.

• ***Glaucoma.*** "Advancing glaucoma and blindness from glaucoma are major potential complications with a keratoprosthesis," Dr. Rapuano says. "Most patients—either at time of KPro surgery or prior to it—have a tube shunt implanted, which decreases the chances of permanent glaucoma damage but doesn't eliminate it."

Because traditional tonometry cannot be used with the device in place, it is difficult to measure IOP accurately, and therefore, to follow the glaucoma in these patients. Dr. Aquavella suggests monitoring for IOP elevations using optic nerve head evaluation, visual fields and scleral indentation.

• ***Ocular surface issues.*** Using a bandage contact lens improves device retention, ocular surface hydration and patient comfort and also prevents complications, such as dellen formation, epithelial defects and corneal melt.[1] "Contact lens management problems require refitting, frequent lubrication, and insertion and removal lessons," Dr. Aquavella says. Some patients may need to use the bandage lenses indefinitely; if so, compliance is even more paramount.

• ***Endophthalmitis.*** The incidence of endophthalmitis in KPro eyes has decreased considerably with the use of long-term daily antibiotics, which typically include a fourth-generation fluoroquinolone and topical vancomycin.[2] "Patients need to be very compliant with their contact lenses and with antibiotics permanently to decrease the risk of infection," Dr. Rapuano says.

• ***Retroprosthetic membrane.*** The incidence of retroprosthetic membrane—membrane growth on the back side of the device—in KPro eyes is reported to be between 25% and 65%.[3] "This is mitigated by topical steroids, with increasing dosage from the moment there are changes in the visual axis," Dr. Aquavella says. "Ultimately, YAG laser treatment is easy to do, if necessary."

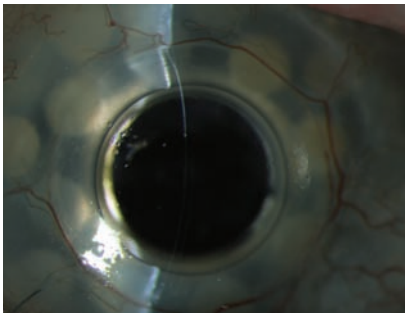

Photo: James Aquavella, MD

This patient has had a keratoprosthesis in place for five years.

• ***Cystoid macular edema.*** "Cystoid macular edema occurs in a small percentage and requires topical steroids," Dr. Aquavella says. "The patient will require antibiotic prophylaxis forever along with follow-up every three months for the first year, and then every six months afterward."

• ***Cellular debris.*** KPro patients are also prone to accumulating cellular debris within and around the PMMA back plate. "Small amounts of debris can severely reduce vision," Dr. Aquavella says. "Debris on the optic can be cleared with routine cleansing or the use of gas permeable lens cleaner." ■

1. Magalhães FP, Sousa LB, Oliveira LA. Boston type I keratoprosthesis: Review. Arq Bras Oftalmol. 2012 May-Jun;75(3):218-22.
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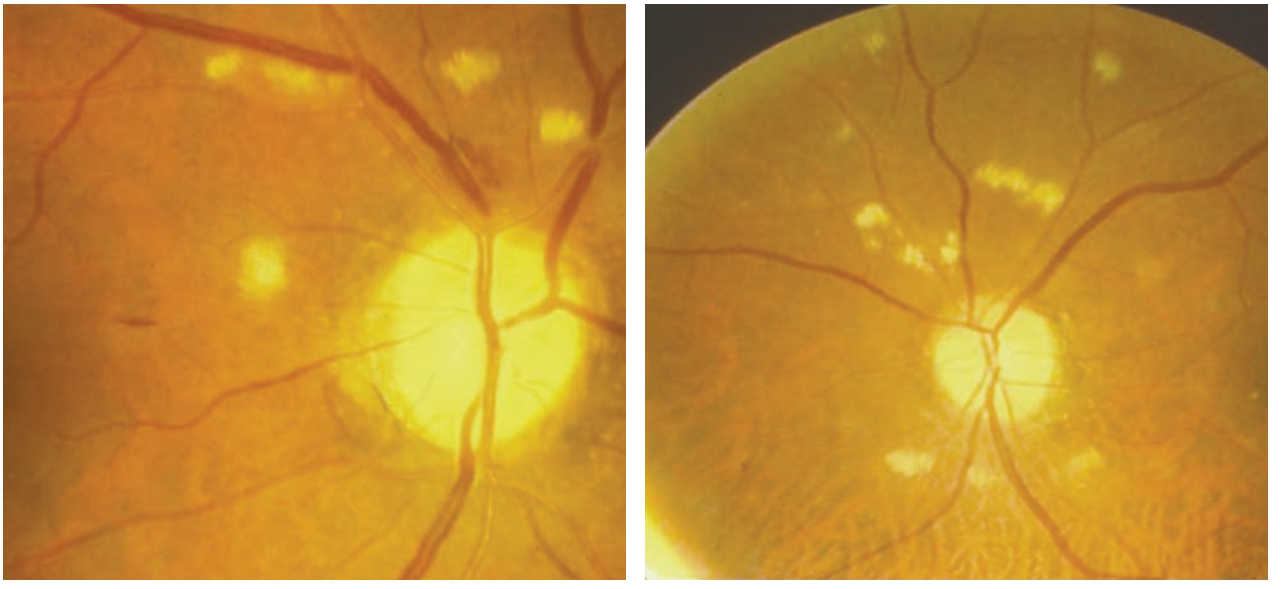
3. Dohlman CH, Colby KA, Belin MW, Todani A. Titanium vs. PMMA backplates for Boston keratoprosthesis: incidence of retroprosthetic membrane. ARVO, 2009;1505/A415.

Beware the Bite of 'the Wolf'

Systemic lupus erythematosus (Part 1): This autoimmune disease affects a wide range of systems in the body, including the eyes.

By Joseph Pizzimenti, OD, and Carlo Pelino, OD



Note the multiple cotton wool infarcts and flame hemorrhages in this patient with systemic lupus erythematosus.

When attacked by organisms and other foreign molecules, the body's immune system responds with an impressive defense.

But when the body mistakes its own cells for invaders, the results can be devastating. In autoimmune diseases, the body has a misguided immune response in which it manufactures T cells and antibodies directed against its own cells and organs.[1]

In this column, the first of a two-part series, we focus on systemic lupus erythematosus (SLE), a common chronic autoimmune disease that affects multiple systems in the body.[1,2]

In SLE and other autoimmune diseases, the immune system's recognition apparatus breaks down—specifically, misguided T cells and autoantibodies contribute to the development of these conditions.[1,2] They begin to destroy healthy cells and tissues, leaving the body unable to perform vital functions and making it vulnerable to attack from actual pathogens.

Let's review some basics of this complex disease, potential signs and symptoms, and how it affects the eye specifically.

The Impact of Lupus

In 1851, doctors coined the name "lupus erythematosus" for a disease frequently characterized by a facial rash that looked like the bite of a wolf (*lupus* means wolf; *erythema* means redness).[4]

There are several categories of lupus, including:

- SLE
- Discoid
- Subacute cutaneous lupus erythematosus
- Drug-induced
- Neonatal

Of these subtypes, SLE is the most common and serious. Estimates of its prevalence vary considerably, but one sizable national review suggested that 161,000 Americans have definite SLE—while as many as 322,000 have definite or probable SLE.[5]

While the disease can affect a wide patient demographic, it does discriminate. SLE affects women more frequently than men and is more common in blacks, Hispanics,

Asians and Native Americans than in whites.[6] Its onset usually occurs between ages 15 and 45, but it sometimes appears earlier or later in life.[6]

Etiology and Patient History

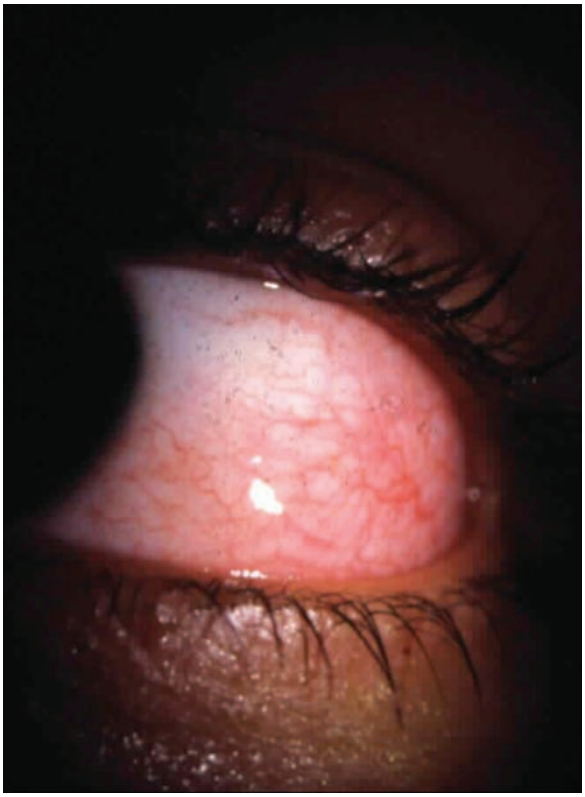
Perform a thorough history and review of systems for patients suspected of having SLE, at minimum covering the following symptoms and signs:[2,3]

- Alopecia
- Anemia
- Arthritis
- Edema
- Fatigue
- Fever
- Pleurisy
- Photosensitivity
- Seizures
- Skin rashes
- Ulcers of the mouth or nose.

The specific etiology of SLE is unknown, although clinicians and researchers consider it to be multifactorial.[2] Previous and current investigations suggest a role for genetic, hormonal, immunologic and environmental factors. This wide range of contributing factors may help explain SLE's variable clinical manifestations.[7-9]

The Eye in SLE

The course of SLE may be unpredictable, with periods of exacerbation and remission. The skin, kidneys, lungs, spleen, joints, mucous membranes, central nervous system and heart are the organs principally affected.[2-4] However, complications from SLE can involve almost any organ system, including ocular tissues.



Episcleritis in another patient with SLE.

SLE may affect the eyes and/or visual system in up to one-third of patients. Ocular manifestations of SLE are mediated directly or indirectly by antibody formation and the creation of immune complexes. Complications may be sight threatening, and virtually every component of the eye and visual pathway may be affected.

Keratoconjunctivitis sicca is the most common finding in the eye, but other ophthalmic sites of involvement include the cornea, conjunctiva, episclera, sclera, uveal tract, retinal vasculature, optic nerve and orbit.[9] In fact, ocular manifestations may be the presenting sign of SLE and can be a useful indicator of underlying systemic disease activity.[9] ■

Stay tuned for part two of this series in the March 2013 issue, where we will discuss the diagnostic workup, treatment and management of SLE.

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2. Diseases of Immunity. In: Kumar V, Cotran RS, Robbins S (eds.). Robbins Basic Pathology. 7th ed. Philadelphia: Saunders; 2003:103-64.
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4. Blotzer JW. Systemic lupus erythematosus I: historical aspects. Md State Med J. 1983 Jun; 32(6):439-41.
5. Helmick CG, Felson DT, Lawrence RC, et al. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part I. Arthritis Rheum. 2008 Jan;58(1):15-25.
6. National Institute of Arthritis and Musculoskeletal and Skin Diseases. Handout on health: systemic lupus erythematosus. Available at: www.niams.nih.gov/Health_Info/Lupus/. Accessed December 27, 2012.
7. Arevalo JF, Lowder CY, Muci-Mendoza R. Ocular manifestations of systemic lupus erythematosus. Curr Opin Ophthalmol. 2002 Dec;13(6):404-10.
8. Sivaraj RR, Durrani OM, Denniston AK, et al. Ocular manifestations of systemic lupus erythematosus. Rheumatology (Oxford). 2007 Dec;46(12):1757-62.
9. Read RW. Clinical mini-review: systemic lupus erythematosus and the eye. Ocul Immunol Inflamm. 2004 Jun;12(2):87-99.

Reported Ocular Complications of SLE[7-9]

Ocular Anatomy	SLE Complication
Lids/lashes	Discoid rash, blepharitis
Ocular surface	Keratoconjunctivitis sicca, recurrent corneal erosions
Episclera/sclera	Episcleritis and scleritis of variable type and severity
Anterior chamber	Uveitis, frequently accompanies episcleritis/scleritis
Posterior segment	Cotton-wool infarct, retinal hemorrhages, hard exudates, retinal vascular occlusions, vasculitis, proliferative retinopathy
Choroid	Ischemia, effusions
Optic nerve	Optic neuritis, ischemic optic neuropathy
Oculomotor disorders	Secondary to vasculitic or ischemic events
Pupil disorders	Horner's syndrome, tonic and light-near dissociation of pupils
Visual pathway	Retrochiasmal disease, intracranial hypertension

Take Your Best Shot

Our patient presented with blurred vision, floaters and poor night vision. What is the most likely diagnosis? **By Mark T. Dunbar, OD**

A 60-year-old white female presented with symptoms of blurred vision (OD > OS) and bilateral floaters that had persisted for the last six months. She also noted increased difficulty seeing at night. Her systemic history was significant for hypertension and high cholesterol, for which she was properly medicated.

On examination, her best-corrected visual acuity measured 20/100 OD and 20/30 OS. Confrontation visual fields were full to careful finger counting OU. Her pupils were equally round and reactive, with no evidence of afferent defect.

The anterior segment evaluation was significant for early nuclear sclerotic and trace posterior subcapsular cataracts OU.

Dilated fundus exam showed a significant vitritis in both eyes. The optic nerves appeared healthy, with small cups and good rim coloration and perfusion OU. The arteries and veins were slightly attenuated. There was no foveal light reflex in either macula. Additionally, we detected a mild epiretinal membrane OU.

On indirect ophthalmoscopy, we noted obvious retinal changes (*figures 1 and 2*). Further, we obtained a spectral domain optical coherence tomography (SD-OCT) scan (*figure 3*).

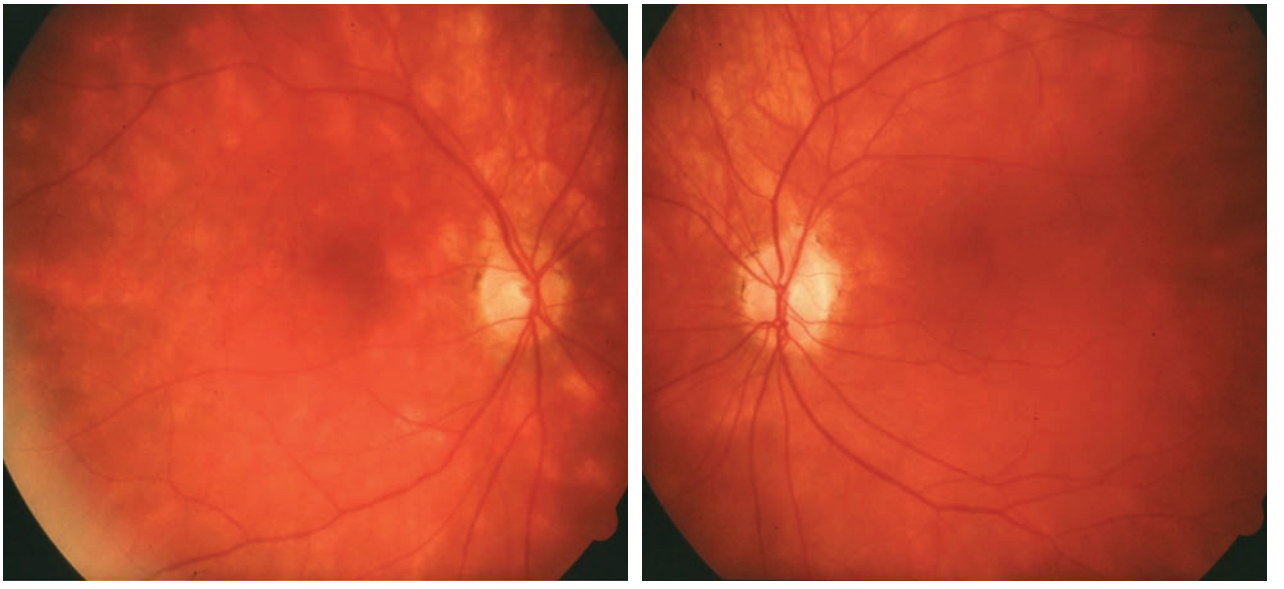
Take the Retina Quiz

1. What does the SD-OCT scan reveal?
   a. Neurosensory retinal detachment.
   b. Cystoid macular edema (CME).
   c. Retinoshisis.
   d. Stage 1 macular hole.

2. At which retinal level are the depigmented lesions located?
   a. Choroid.
   b. Retinal pigment epithelium (RPE).
   c. Sensory retina.
   d. Both sensory retina and RPE.

3. What is the likely diagnosis?
   a. Multifocal choroiditis and panuveitis.
   b. Serpiginous choroiditis.
   c. Vitiliginous chorioretinitis.
   d. Syphilis.

4. What additional tests would help confirm the diagnosis?



1, 2. Posterior pole and midperiphery of both eyes exhibit hazy media, vessel attenuation and hypopigmented spots (OD left, OS right).

a. Fluorescein angiography.
b. Blood testing for HLA-A29.
c. Blood testing for HLA-B9.
d. Angiotensin-converting enzyme (ACE).

5. What is the best treatment option?
a. Corticosteroids.
b. Observation.
c. Immunosuppressive agents.
d. Both a and c.

For answers, go to page 98.

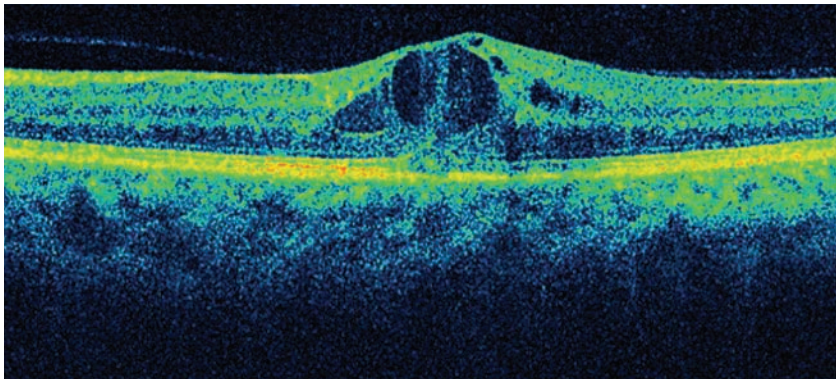
Discussion

We diagnosed our patient with vitiliginous chorioretinitis, a rare inflammatory condition of the choroid and retina. The condition originally was termed "birdshot retinochoroidopathy" in 1980, because the scattered displacement of the associated lesions was reminiscent of a shotgun blast.[1]

Meanwhile, around the same time, J. Donald M. Gass, MD, of the Bascom Palmer Eye Institute in Miami, used the term vitiliginous chorioretinitis to describe the condition because he believed the depigmented retinal lesions resembled skin lesions observed on patients with vitilligo.[2] Since then, both terms have been used interchangeably by academics and practicing clinicians.

In the early reports, vitiliginous chorioretinitis was thought to occur predominantly in women.[1] Today, the condition is understood to occur in both men and women in the fifth to seventh decade of life.[2] The most common symptoms include blurred vision, increased floater volume and photopsia.[2] In advanced disease progression, patients frequently report night blindness and color vision loss.[2]

The hallmark of vitiliginous chorioretinitis is significant vitritis (accounting for the increased floaters) and multifocal patches of depigmented or hypopigmented lesions that may be creamy yellow or orange in color. The ill-defined patches typically are round or oval in shape. Some will appear elongated in a pattern that radiates toward the peripheral fundus.



3. An SD-OCT scan of the right eye. Can you discern any macular changes?

The legions' striking feature is the lack of chorioretinal scaring or hyperpigmentation at the margins, which often are seen in other inflammatory conditions. The disease originates in the choroid and later involves the RPE. Interestingly, there does not appear to be any thinning within the RPE or choroid in the depigmented areas.[2]

The diagnosis of vitiliginous chorioretinitis usually is made based on the clinical presentation; however, there also is a pronounced association with the HLA-A29 antigen. More specifically, at least 90% of patients with vitiliginous chorioretinitis test positive for HLA-A29 upon examination—suggesting an autoimmune mechanism as well as a genetic predisposition.[2] In fact, this association is so strong that you should consider a diagnosis of saroidosis or another granulomatous condition if the patient tests negative for the HLA-A29 antigen.

Vitiliginous chorioretinitis is a chronic, slowly progressive condition that exhibits periods of remission and exacerbation. Typically, patients lose vision from cystoid macular edema (as we documented in our patient), which results from chronic inflammation. As a consequence, management is aimed at quieting the inflammation.

Corticosteroids have been the mainstay treatment option, but have yielded limited success. Patients may note visual improvement as a result of CME resolution, but will not exhibit a decrease in lesion number or severity. Immunosuppressive agents, such as methotrexate, mycophenolate mofetil and cyclosporine, also have been used alone or in combination with corticosteroids for long-term treatment.[2]

We treated our patient with pulsed, high-dose oral steroids and low-dose methotrexate. Her CME resolved and her vision returned to 20/25 OU. However, during the ensuing years, she continued to experience recurrences and exacerbations while on immunosuppressive agents. ■

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Double Trouble III

To wrap up this three-part series, we discuss the case of yet another established glaucoma patient who presented with double vision. **By Joseph W. Sowka, OD, Alan G. Kabat, OD**

In our previous two "Double Trouble" articles (*September and November 2012*), we described two patients being treated for glaucoma who concurrently developed double vision from cranial nerve (CN) VI and CN III palsies, respectively. This month, we complete the trifecta.

A 54-year-old man being treated for primary open-angle glaucoma reported occasionally experiencing double vision. Because the double vision was only intermittent, the patient wasn't overly concerned—he simply wanted to express the complaint.

Upon questioning, he said that the double vision was vertical and became worse when he was reading and/or tired. He did not report any other physical problems and said that he felt well overall. His recent physical examination was normal, save a slightly elevated cholesterol level.

On general inspection, it was easy to see that he had a slight head tilt to the right. Alternate cover testing demonstrated a left hyperphoric deviation, which worsened in right gaze and left head tilt. Based upon this signature motility, he was diagnosed with a left CN IV palsy.

What is CN IV Palsy?

Patients with CN IV palsy typically present with complaints of vertical diplopia that worsens when reading. There may be an inability to look both down and in. There may also be a component of horizontal diplopia, as a lateral phoria becomes manifest due to the vertical dissociation.[1-4] The patient's chin also may be tucked downward as well.

Further, the patient may report greater diplopia or visual discomfort when tilting his or her head toward the side of the palsy. Commonly, the patient develops a compensatory head tilt opposite to the affected superior oblique muscle. Accomodating for these postural changes generally makes the patient more visually comfortable. Ocular motility testing with the alternate cover test will reveal a hyperphoric or hypertropic deviation that worsens upon opposite gaze and same-side head tilt.[5-8]

Patients with CN IV palsy frequently present with concurrent hypertension and/or diabetes.[9-11] In many instances, there will be a history of head trauma immediately preceding development of CN IV palsy. The trauma need not be major, as relatively minor injuries can trigger the event.[2,3,12-14] In cases of longstanding, decompensated CN IV palsy, the inciting trauma may have occurred several years earlier and often is forgotten by the patient.

The fourth nerve especially is prone to trauma, because it exits the brain stem and courses through the subarachnoid space. In contrast to third nerve palsies with an etiology in the subarachnoid space, fourth nerve palsies are rarely caused by aneurysmal compression. The most common causes of damage to the fourth nerve in this region are trauma and ischemic vasculopathy.[3]

Due to the large number of other neural structures that accompany the fourth nerve as it travels through the cavernous sinus and superior orbital fissure, it is unlikely that patients will exhibit isolated fourth nerve palsy due to damage within these areas. More likely, there will be a concomitant palsy of cranial nerves III and VI. Common causes of damage to the fourth nerve in these areas are herpes zoster, inflammation of the cavernous sinus or posterior orbit, meningioma, metastatic disease, pituitary adenoma and carotid cavernous fistula.[15] Trauma to the head or orbit can cause damage to the trochlea with resultant superior oblique muscle dysfunction.

Trauma and vascular disease are considered the main causes of acquired CN IV palsy.[14,15] However, numerous reports of other potential causes of isolated CN IV palsy, include multiple sclerosis, polycythema vera, cat-scratch disease and, rarely, metastatic disease.[15]

Managing Patients with CN IV Palsy

A fourth nerve palsy often presents suddenly, but may result from decompensation of a longstanding or congenital palsy and the onset just seems sudden. In order to differentiate these two types of palsies,

ask for old photographs. A patient with a decompensated, longstanding palsy will present with a compensatory head tilt that often can be identified in photographs. Usually, patients are not even aware of their head tilt.

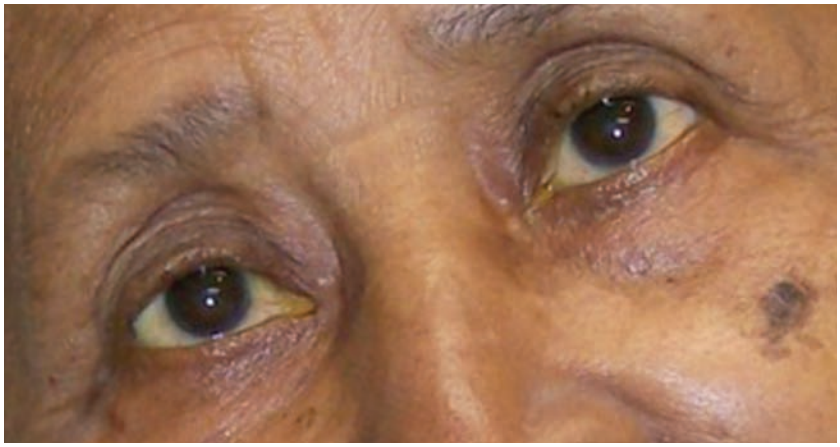
Patients with longstanding, decompensated fourth nerve palsies will have an exaggerated vertical fusional ability. Such longstanding fourth nerve palsies typically have a benign course that requires no further management.

In the case of complicated fourth nerve palsies (those that present with other concurrent neurological dysfunction), the patient should undergo neuroradiological studies. The accompanying signs and symptoms typically dictate the extent of these evaluations.

In the case of isolated fourth nerve palsies caused by recent trauma, the patient should also undergo neuroradiological studies of the head to dismiss the possibility of a concurrent subarachnoid hemorrhage. If the fourth nerve palsy is not associated with recent trauma, a history of past trauma should be investigated.

If the fourth nerve palsy is due to previous trauma and has recently decompensated, the diplopia can be managed by the placement of vertical prisms in spectacles. Further, if the patient is elderly and has a fourth nerve palsy of truly recent origin, an ischemic vascular evaluation should be undertaken to search for diabetes and hypertension.

If, however, the palsy is caused by vascular infarct, then it will spontaneously resolve over a period of three to six months. Usually, no further management beyond periodic observation and either occlusion or press-on prism therapy is required.[2,3] But, in some cases, recovery does not occur.[2,3] In these instances, permanent prism (ground into the spectacle lenses), muscle surgery or botulinum toxin A injections may be considered.[16]



Another patient with a congenital left CN IV palsy and a severe right head tilt.

When encountering isolated CN IV palsy, delay prescribing permanent prisms for at least three months in order to allow for the palsy to recover. Otherwise, glasses with permanent prism correction can induce vertical diplopia, should the palsy recover.

In the case presented here, the patient was educated about his new diagnosis. His recent normal physical exam and lack of other signs or symptoms, along with the head tilt, made us suspect that the patient had a longstanding CN IV palsy that had simply decompensated. We confirmed our suspicions when he produced his driver's license photo (which was several years old), showing the right head tilt.

A 1.00Δ base-down prism over his left spectacle lens made him feel more visually comfortable. Going forward, we instructed him to remain keenly aware of his double vision. Additionally, we informed him that if the problem persisted and affected his quality of life, then we'd add the prismatic correction to his spectacles permanently. ■

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The Miracle of Birth

Amniotic membrane implantation can prevent scarring and corneal haze in patients with advanced ocular surface disease. **By Paul M. Karpecki, OD, and Diana L. Shechtman, OD**

Over the last decade, we have become increasingly knowledgeable about the diagnosis and treatment of various ocular surface diseases. In managing these patients, we've discovered that some conditions do not respond to topical therapy or other conventional treatments.

However, more advanced procedures, such as amniotic membrane implantation, specifically target and effectively marginalize the underlying causes of advanced ocular surface disease.

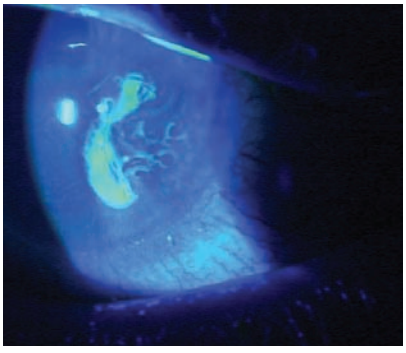
Amniotic Membrane

The amniotic sac that surrounds a baby during gestation exhibits incredible anti-inflammatory, anti-scarring and even antimicrobial characteristics.[1] The amniotic membrane can be harvested from the mother's placenta after the normal delivery of a baby.

The tissue primarily is composed of collagen types IV and VII, fibronectin and laminin—some of the most common components of the ocular surface and cornea.[2] Further, the amniotic membrane contains hyaluronic acid, which has been found to directly inhibit pro-inflammatory cells and suppress T-cell activation.[3] Amniotic membrane also has been shown to stimulate healthy re-epithelialization of damaged ocular surface tissue.[4]

Clinical Indications

Amniotic membrane implantation isn't new to eye care. Interestingly, the technique first was used in the 1940s to treat caustic burns of the cornea and conjunctiva, as well as after symblepharon removal.[5,6]



This patient with a persistent epithelial defect and recurrent corneal erosion recently underwent treatment with ProKera (Bio-Tissue).

Currently, the primary indications for amniotic membrane implantation include keratitis (including neurotrophic keratitis), superior limbic keratoconjunctivitis, persistent epithelial defects, chronic non-responsive superficial punctate keratopathy, infectious corneal ulcers, recurrent corneal erosion, toxic keratitis or chemical burns, Salzmann's nodular degeneration, and even limbal stem cell deficiency.[2,6-10]

The Procedure

Cryopreserved amniotic membrane grafts, such as ProKera (Bio-Tissue), are stored in a freezer, whereas dehydrated implants can be left at room temperature. Before applying the graft, an anesthetic is administered to the affected eye. Additionally, the graft is lavaged with sterile saline solution to remove any preservatives.

First, the graft is inserted under the upper eyelid (while the patient looks down), and then is placed in the lower cul-de-sac (while the patient looks up). Subsequently, surgical tape is applied to the closed eyelids.

Afterward, patients are informed that the less movement they can make with their eyes, the more comfortable the device will be. If the patient has a persistent epithelial defect for example, antibiotics should be prescribed to prevent infection.

Not only is an amniotic membrane implant physically protective like a bandage contact lens, but it can also transfer many of its nutrients to the cornea or ocular surface. In most instances, the graft's protective effects will persist for seven days. However, in more inflammatory conditions, the amniotic membrane may dissolve within three to four days as its nutrients are transferred and exhausted.

For the first few days after implantation, it is important to see amniotic graft patients daily to provide reassurance and assess healing—especially in those with epithelial defects. Fluorescein dye can be instilled on top of the amniotic membrane ring to monitor re-epithelialization, without the need for removal, until the cornea is fully healed or the membrane is no longer intact.

Once treatment is completed, the ring may be removed. This is achieved by topically anesthetizing the eye, then grabbing the edge of the ring with a forceps while the patient looks down.

Amniotic membrane implantation is a very effective and valuable tool for eye care providers who frequently manage patients with significant ocular surface disease presentations.

Such implants facilitate rapid ocular surface re-epithelialization and have been shown to support the expansion of limbal stem cells, which may further aid in the corneal healing process.[11] ■

Dr. Karpecki is a paid consultant to Bio-Tissue Inc. Neither he nor Dr. Shechtman have any direct financial interest in the products mentioned.

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Product **Review**

Color Vision Testing

Anomaloscope

You may have used color vision testing to detect color blindness before, but have you ever used it to diagnose and monitor other clinical eye conditions? Many diseases—such as diabetes, glaucoma, optic neuritis, age-related macular degeneration and cataracts—cause minute changes in color vision.

By recognizing these tiny shifts in color sensitivity, the ColorTrac anomaloscope can help clinicians detect early eye disease, monitor changes over time and manage disease for functional improvement, the manufacturer says. This portable, one-pound device can be easily toted from one exam room to another and used at health fairs and screenings. It doesn't require any special training and is reimbursable through Medicare, the company says.

ColorTrac shows the patient an array of seven match points simultaneously; and the patient is asked to select the vertical pair that is the closest color match. The match-range is centered on the normal point, and is segmented into increments of just noticeable differences, which allow positive, accurate responses in less than 60 seconds, the company says.

Visit www.colortracdx.com.

Color Blindness Test App

With a new app from EnChroma Inc., adults and children can now take a free color blindness test on their smartphone or tablet.

The test presents a series of simple geometric shapes—a circle, square or diamond—each camouflaged by a random pattern of dots of varying size and brightness. The color of the dots is the only visual cue that allows users to identify the shape.

When the test starts, the hidden shapes are very easy to see because there is a stark difference between the foreground and background colors. As the test proceeds, the plates get more challenging. At the end, the app provides users with an assessment of their color vision including the type and extent of the deficiency, if any.

Patients can take the test online at enchroma.com/test or by downloading the free app by searching for "enchroma" in their mobile device app store. Visit www.enchroma.com.

Optical Display

Wall Showcase

Looking for a better way to organize and display your frames? If so, then you might be interested in a wall showcase from Tecno Display.

This preassembled showpiece with tempered glass has eight adjustable 1/4-inch-thick glass shelves, a center support divider, a solid back and locking sliding doors.

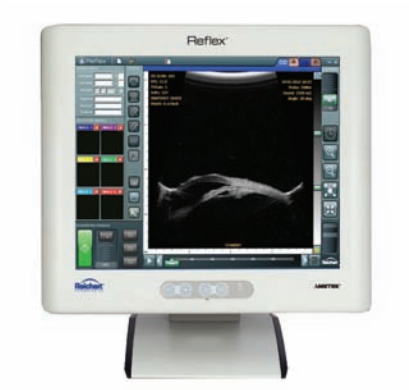
Shown in mahogany, this piece is available in a number of standard finishes and custom finishes upon request. Its dimensions are 81"H x 48"W x 20"D and wheels make it easy to move around the office.

Optional add-ons include micro-halogen spotlights, LED spotlights, clear or mirror back, choice of divider finish and additional shelving.

Visit www.tecnodisplay.com.

Diagnostic Imaging

Reflex Ultrasound Biomicroscope



If you see a lot of glaucoma patients or you specialize in cataracts or corneal disease, Reichert's next-generation ultrasound biomicroscope (UBM) could be a good fit for your practice. Reflex UBM also provides valuable information for the treatment of phacomorphic lenses, plateau iris syndrome, cysts, tumors, retinal tears, cells in the vitreous chamber and vitreous hemorrhages, the company says.

A technician or doctor can perform the test within five minutes, and patients can remain upright in the exam chair throughout the procedure, with no need for a water bath or scleral shell. The device is about the same size as a computer monitor and now features a user-friendly touchscreen and software improvements, Reichert says. Images captured are DICOM-compliant and can be exported to EMR systems. Also, UBM procedures are billable through Medicare.

Visit www.reichert.com.

Payment Processing System

The Revenue Maximizer

If you're looking to simplify your billing, then the Revenue Maximizer patient payment processing system might be able to help. This suite of web-based tools by TransEngen is designed to accelerate patient payments, improve cash flow and reduce bad debt.

It allows the eye care provider's office to tie the patient account, provider, location, department and claim ID to the payment transaction. The system processes major credit and debit cards, provides electronic checking account transactions and converts paper checks to electronic funds transfers. It uses up-to-date security and operational standards to protect card and account holder data, the company says.

This web-based system uses your current computer hardware and Internet connection with a small card reader that plugs into the computer's USB port.

Visit www.therevenuemaximizer.com. ■

Meetings + Conferences

February 2013

■ **6.** *IOA Winter Seminar.* Ritz Charles, Carmel, Ind. Hosted by: Indiana Optometric Association. Email blsims@ioa.org or call (317) 237-3560. Visit www.ioa.org.

■ **6-7.** *MOA Winter Seminar.* Kellogg Hotel & Conference Center, East Lansing, Mich. Hosted by: Michigan Optometric Association. Contact Amy Possavino at amy@themoa.org or (517) 482-0616. Visit www.themoa.org.

■ **8-10.** *3rd Annual Final Eyes CE.* Baptist Hospital Conference Center, Jacksonville, Fla. CE hours: 16. Contact Valerie Fernandez at valierie.fernandez@bmcjax.com or call (904) 202-2080. Visit FinalEyesCE.com.

■ **12-14.** *The Eye Show London 2013.* London ExCeL International Exhibition Centre, United Kingdom. Hosted by: Emergexpo plc. CE hours: 18. Email conference@theeyeshow.com or visit www.theeyeshow.com.

■ **15-17.** *52nd Annual Heart of America Contact Lens Society Contact Lens and Primary Care Congress.* Sheraton Kansas City Hotel and Crown Center, Kansas City, Mo. E-mail registration@thehoacls.org or call (918) 341-8211. Visit www.hoacls.org.

■ **16-20.** *SkiVision 2013.* Viceroy Snowmass Luxury Mountain Resort, Snowmass Village, Colo. CE hours: 23. Email questions@skivision.com or call (888) SKI-2530. Visit www.skivision.com.

■ **21.** *7th Central Jersey Optometric Seminar.* CentraState Medical Center, Freehold, N.J. Time: 7:00 p.m.–10:30 p.m. CE hours: 4. Contact William Potter, OD, at eyedoc2180@aol.com or (609) 947-8545. Visit http://optometryonwest44th.webs.com.

■ **27-March 3.** *SECO International 2013.* Building A, Georgia World Congress Center, Atlanta. CE hours: 300+. Contact Bonny Fripp at bfripp@secostaff.com or (770) 451-8206, ext. 13. Visit www.seco2013.com.

■ **28-March 2.** *MOA Big Sky Conference.* Huntley Lodge, Big Sky Conference Center, Big Sky, Mont. Hosted by: Montana Optometric Association. Contact Executive Director Sue Weingartner at sweingartner@rmsmanagement.com or (406) 443-1160. Visit www.mteyes.com.

March 2013

■ **3-4.** *COVD at SECO 2013.* Time: 8 a.m. - 5 p.m. OMNI Hotel at CNN Center, Atlanta. Hosted by: College of Optometrists in Vision Development. Featured speakers: Carl G. Hillier, OD, FCOVD, W.C. Maples, OD, FCOVD, and Ashley Reddell, OD, FCOVD. Visit www.covd.org. *Registration is separate from SECO 2013.

■ **3-8.** *27th Annual Eye Ski Conference.* The Lodge at Mountain Village, Park City, Utah. CE hours: 20. Contact Tim Kime, OD, at tandbkime@buckeye-express.com. Visit www.eyeskiutah.com.

■ **10.** *6th Annual Evidence Based Care in Optometry Conference.* BWI Marriott, Linthicum Heights, Md. Hosted by: Maryland Optometric Association and the Wilmer Eye Institute. Email moa@assnhqtrs.com or call (410) 727-7800. Visit www.marylandeyes.com.

■ **14-17.** *International Vision Expo & Conference East 2013.* Jacob K. Javits Convention Center, New York, N.Y. CE hours: 350. Visit www.visionexpoeast.com.

■ **16-17.** *7th Annual Conference on Comprehensive Eye Care.* The Sheraton Hotel, Niagara Falls, N.Y. Hosted by: PSS EyeCare. Featured speakers: Ron Melton, OD, Randall Thomas, OD, Paul Karpecki, OD, and Deepak Gupta, OD. CE hours: 18. Email education@psseyecare.com or call (203) 415-3087. Visit www.psseyecare.com.

■ **24.** *"Practicing Full Scope Primary Care Optometry: 2013 and Beyond."* Tinley Park Convention Center, Tinley Park, Ill. Hosted by: Illinois Optometric Association. Featured speaker: Pamela Lowe, OD. Email ioa@ioaweb.org or visit www.psseyecare.com.

April 2013

■ **12.** *American Conference on Pediatric Cortical Visual Impairment.* Time: 7:30 a.m. - 5:00 p.m. Children's Hospital & Medical Center, Omaha, Nebr. Contact CME Coordinator Sara M. Olsen, MEd, at solsen@childrensomaha.org or (402) 955-6070.

■ **12-13.** *OAOP Annual Spring Congress 2013.* Embassy Suites & Conference Center, Norman, Okla. Hosted by: Oklahoma Association of Optometric Physicians. Visit www.oaop.org.

■ **12-14.** *American Optometric Society 4th Annual Meeting & CE Seminar.* Westin Riverwalk, San Antonio, Texas. Hosted by: American Optometric Society. Visit www.optometricsociety.org.

■ **13-14.** *5th Annual Symposium on Ocular Disease.* Crowne Plaza, Tyson's Corner, Va. Hosted by: PSS EyeCare. Featured speakers: Deepak Gupta, OD, and Kimberly Reed, OD. CE hours: 18. Email education@psseyecare.com or call (203) 415-3087. Visit www.psseyecare.com.

■ **19-20.** *Educational Meeting 2013.* Mission Inn, Howey-in-the-Hills, Fla. Hosted by: the Florida Chapter of the American Academy of Optometry. Featured speakers: Carlo Pelino, OD, Albert Woods, OD, and John McClane, OD. CE hours: 10. Contact Arthur T. Young, OD, at eyeguy4123@msn.com or (239) 542-4627.

■ **19-21.** *WFOA Spring Seminar 2013.* Hilton Sandestin Beach Golf Resort & Spa, Destin, Fla. Hosted by: West Florida Optometric Association. Contact Jennifer Major, OD, at wfoatreasurer@gmail.com. Visit www.wfoameeting.com.

■ **24-29.** *11th Annual Education Conference.* Hilton Embassy Suites Kingston Plantation, Myrtle Beach, S.C. Hosted by: New Jersey Chapterof the American Academy of Optometry. CE hours: 16. Featured speakers: Diana Shechtman, OD, and Carlo Pelino, OD. Contact Dennis H. Lyons, OD, at dhl2020@aol.com or (732) 920-0110.

■ **26-28.** *28th Annual Morgan/Sarver Symposium.* DoubleTree Hotel, Berkeley Marina, Berkeley, Calif. Hosted by: University of California, Berkeley, School of Optometry. CE hours: 20. Email optoCE@berkeley.edu or call (800) 827-2163. Visit http://optometry.berkeley.edu/ce/morgan-sarver-symposium.

May 2013

■ **1-4.** *2013 Annual Educational Conference & Exposition.* Hilton Garden Inn, Missoula, Mont. Hosted by: Montana Optometric Association. Contact Executive Director Sue Weingartner at sweingartner@rmsmanagement.com or (406) 443-1160. Visit www.mteyes.com.

■ **2-4.** *MWCO Annual Congress.* Caesar's Palace, Las Vegas. Hosted by: Mountain West Council of Optometrists. Contact Tracy Abel, CMP, at tracyabel@earthlink.net or call (888) 376-6926. Visit www.mwco.org.

■ **9-10.** *117th Annual Meeting and Spring Seminar.* DeVos Place, Grand Rapids, Mich. Hosted by: Michigan Optometric Association. Contact Amy Possavino, at amy@themoa.org or call (517) 482-0616. Visit www.themoa.org.

■ **17-19.** *2013 AZOA Spring Congress.* Hilton Tuscon El Conquistador Golf & Tennis Resort, Tucson, Ariz. Hosted by: Arizona Optometric Association. Contact Kate Diedrickson, at kate@azoa.org or call (602) 279-0055. Visit www.azoa.org.

■ **17-29.** *Nova Southeastern University's 17th Annual Eye Care Conference & Alumni Reunion.* NSU College of Optometry, Fort Lauderdale, Fla. Contact Vanessa McDonald at oceaa@nova.edu or visit http://optometry.nova.edu/ce.

June 2013

■ **7-9.** *Ocular Symposium: Pearls in Ocular Diagnosis.* Holiday Inn Golden Gateway, San Francisco. CE hours: 24. Contact Lorraine Geary at ocularsymp@aol.com or call (415) 278-9940.

■ **13-16.** *Maui 2013.* Wailea Beach Marriott Resort & Spa, Maui, Hawaii. Hosted by: *Review of Optometry*. Meeting chair: Paul Karpecki, OD. CE hours: 14. Contact Lois DiDomenico at ReviewMeetings@Jobson.com or (866) 658-1772. For more information, visit www.revoptom.com/conferences.

July 2013

■ **19-22.** *Bermuda 2013.* Fairmont Hamilton Princess, Bermuda. Hosted by: *Review of Optometry*. Meeting chair: Paul Karpecki, OD. CE hours: 14. Contact Lois DiDomenico at ReviewMeetings@Jobson.com or (866) 658-1772. For more information, visit www.revoptom.com/conferences.

August 2013

■ **3-5.** *Annual Educational Retreat 2013.* South Seas Island Resort, Sanibel, Fla. Hosted by: Southwest Florida Optometric Association Inc. CE hours: 14. Contact Brad Middaugh, OD, at swfoa@att.net or (239) 481-7799. Visit www.swfoa.com. ■

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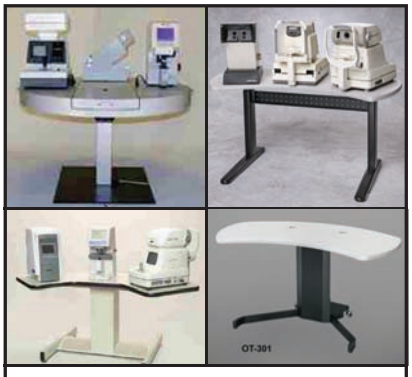

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Vitrectomy with Membrane Peel

The timing of this procedure depends on the patient's symptomatic course.

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

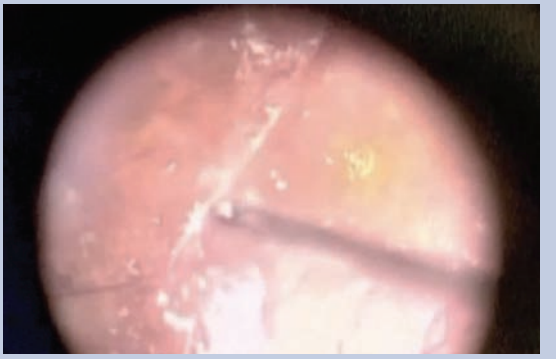

Photo and video courtesy of Alan Franklin, MD, PhD

Go to www.revoptom.com or scan the QR code at left to see video footage of the procedure.

On The Web ›› View a narrated video of an epiretinal membrane peeling procedure.

Vitrectomy with membrane peel is the most common vitreoretinal surgery billed to the Centers of Medicare and Medicaid Services. The procedure is typically performed to intervene in the event of epiretinal membrane (ERM) formation or vitreomacular traction syndrome that presents with visually significant symptoms.

An ERM is a semitranslucent, avascular, fibrocellular membrane located along the inner surface of the retina's internal limiting membrane (ILM). In most instances, ERM formation is seen over or around the macula. Clinically, you may document a loss of foveal reflex, parafoveal light reflection (which looks similar to cellophane), wrinkling of the retinal surface, localized intraretinal hemorrhages or alteration of the parafoveal vasculature (increased tortuosity). Macular edema and/or pseudoholes may also be associated with ERM development.

Some ERM patients are asymptomatic; however, most affected individuals report distorted vision or scotomas that are repeatable on Amsler grid testing. Because some ERMs slowly worsen over time, patients typically experience a gradual reduction in visual acuity.

The first clinical sign of ERM formation tends to be an unnatural macular appearance. Although fluorescein angiography can be used to help diagnose ERM, OCT has become the gold standard; its high-resolution imaging of the vitreoretinal interface detects even the subtlest membrane.

Not all membranes require treatment. The risk of surgical intervention for mild ERMs that have little to no visual impact isn't justified. Typically, the patient's symptomatic course will dictate the timing of surgical intervention. In many cases, mild membranes are simply monitored over time for progression. But once the patient's perception of visual distortion begins to impact his or her quality of life, you should recommend surgical consultation.

Historically, the only viable treatment for ERM was vitrectomy surgery. Although relatively successful, recurrence rates were as high as 16%. Recently, surgeons have begun to also peel the ILM from the retina after vitrectomy, to decrease the risk of recurrence. This additional measure, in essence, reduces the recurrence rate to 0%.

To preserve the anatomic integrity of the retina, ILM removal must be executed with extreme caution. Surgeons may use various imaging devices and intraoperative dyes to help visualize the ILM, and then subsequently peel it off the retina.

In vitrectomy with membrane peel procedures, the instruments are usually inserted 4mm behind the limbus. The surgery is performed under local anesthesia with very small incision ports that do not require suturing. Visual recovery varies from patient to patient, but can be dramatic the very next day.

The most common surgical complications include infection (roughly one in 1,000 procedures), retinal detachment (roughly one in 100 procedures), cataract progression in phakic eyes, bleeding and diplopia. Although such complications are relatively rare, open discussion of the possible risks often helps to gauge the patient's desire for surgical intervention. ■

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Diagnostic **Quiz**

Back to the Suture

By Andrew S. Gurwood, OD

History

A 76-year-old white female presented for an urgent visit with a chief complaint of bilateral foreign body sensation and irritation (OS > OD) that had persisted for two weeks. In the past, we saw the patient for complaints of seasonal ocular allergy and mild dry eye.

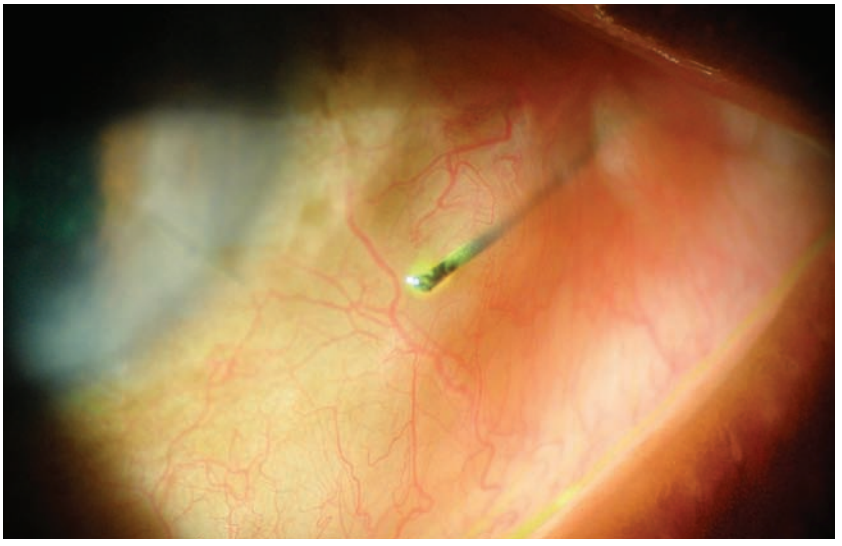
Additional ocular history included bilateral cataract extraction and blepharoplasty three years earlier. She denied any exposure to foreign bodies or harmful substances. Current ocular medications included Pataday (olopatadine hydrochloride 0.2%, Alcon) and artificial tears, as needed.

She had no contributory systemic history and reported no allergies to medications.

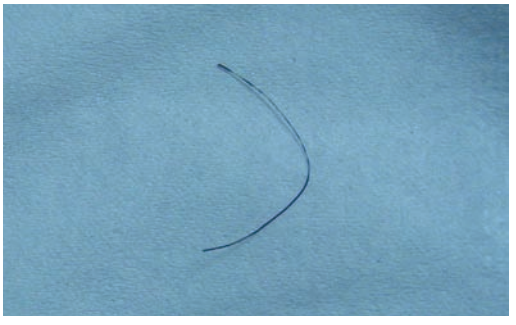
Diagnostic Data

Her best-corrected visual acuity measured 20/20 OU. External examination was normal, with no evidence of afferent pupillary defect. Refraction uncovered mild hyperopia with negligible changes to her habitual spectacle prescription. Biomicroscopy revealed normal lids and lashes OU.

Corneal findings included minimal inferior punctate staining OU and an irregular tear film. The bulbar conjunctiva was white and quiet OU. The anterior chambers were unremarkable.



We uncovered the presence of a foreign object in our patient's left eye. How did it get there?



Her IOP measured 16mm Hg OU. An undilated, 90D fundoscopic examination showed quiet grounds and normal posterior poles OU.

Your Diagnosis

How would you approach this case? Does this patient require any additional tests?

What is your diagnosis? How would you manage this patient? What's the likely prognosis?

To find out, please visit www.revoptom.com. Click on the cover icon for this month's issue, and then click "Diagnostic Quiz" under the table of contents. ■

Thanks to Marc D. Myers, OD, of Coatesville, Pa. for contributing this case.

Retina Quiz Answers *(from page 82):* 1) b; 2) a; 3) c; 4) b; 5) d.

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