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August 15, 2012

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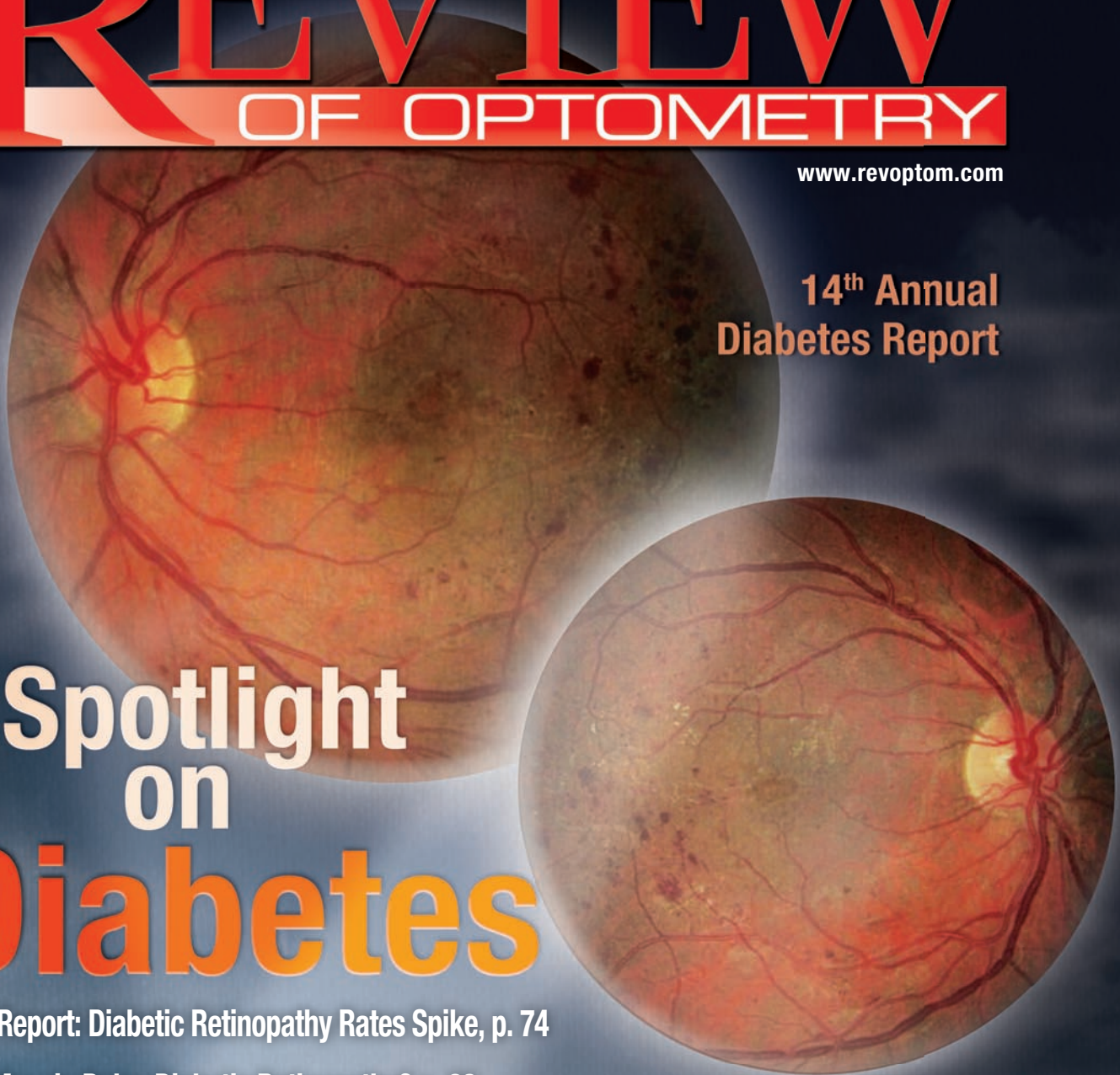

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VOL. 149 NO. 8 ■ AUGUST 15, 2012

IN THE NEWS

Salus University announced that **Rear Admiral Michael H. Mittelman, O.D., M.P.H.**, will succeed **Thomas L. Lewis, O.D., Ph.D.**, as the university's next president. Currently serving as Deputy Surgeon General of the Navy and Deputy Chief of the Bureau of Medicine and Surgery, Dr. Mittelman will complete his service with the Navy before starting as Salus University president sometime in late spring or early summer 2013. Dr. Mittelman earned his optometry degree from the **Pennsylvania College of Optometry** in May 1980. He is past president of the Armed Forces Optometric Society and he recently received the 2012 Distinguished Service Award from the American Optometric Association.

In 2011, **55% of physicians** had adopted an **electronic health record (EHR)** system, and nearly one-half of physicians currently without an EHR system plan to use one within the next year, according to a new study from the **Centers for Disease Control and Prevention's National Center for Health Statistics**. The vast majority of physicians who use EHR systems (85%) reported being somewhat satisfied (47%) or very satisfied (38%) with their system. Among EHR adopters, 74% believe that using their system enhanced overall patient care.

Just a reminder: August is **National Children's Vision & Learning Month**. Are you helping those kids get ready to go back to school?

New Drug for Obesity Carries Ocular Risks

Qsymia—which includes topiramate—can cause myopic shift and angle closure. **By John Murphy, Executive Editor**

Optometrists know the ocular side effects of Topamax (topiramate, Janssen), which is used to prevent epileptic seizures and migraine headaches. Interestingly, when researchers analyzed patients who used this drug, they found that subjects also lost weight.

In mid-July, the FDA approved Qsymia (phentermine/topiramate extended-release, Vivus) to control weight in adults with a body mass index (BMI) of 30 or greater (obese) or adults with a BMI of 27 or greater (overweight) who have at least one weight-related condition, such as hypertension, type 2 diabetes or high cholesterol.

Approximately one-third (35%) of American adults (more than 78 million people) and nearly 17% of teenagers and children (about 12.5 million) are obese, according to the Centers for Disease Control and Prevention.

"We see a fair number of patients with seizure disorder who are on Topamax," says Jimmy D. Bartlett, O.D., chairman and CEO of Pharmakon Consulting Group. "But considering the number of people who are overweight and obese in the United States, the sheer number of potential patients who could be on Qsymia will increase the risk for optometrists to see adverse ocular effects in the office."

Induced myopia and angle-closure glaucoma are the two main adverse ophthalmic effects of topiramate, Dr. Bartlett says.

If your patient is on Qsymia, keep an eye on the patient's refractive error and watch for any indication of increasing myopia, he says. Consider gonioscopy to inspect the anterior chamber angle for evidence of angle closure.

If a patient comes in with acute symptoms of blurred distance vision or pain along with increased intraocular pressure, it could indeed be secondary angle-closure glaucoma, Dr. Bartlett says.

This is different from primary angle-closure caused by pupillary block. So, "absolutely avoid pilocarpine," he says. Instead, "utilize a cycloplegic, like cyclopentolate or homatropine, to relieve the secondary angle closure, followed by the normal methods for reducing acutely elevated intraocular pressure, such as beta-blockers, carbonic anhydrase inhibitors or alpha-2 agonists."

See also Glaucoma Grand Rounds, "New Diet Drug Also 'Slims' Eyes," page 104.

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TPA Bill Fails in Puerto Rico—Again

By Stefania Paniccia, M.S.

Puerto Rico is the only jurisdiction in the United States and its territories where optometrists are not permitted to use any therapeutic pharmaceutical agents (TPAs).

And, it looks like that won't change any time soon.

In mid-May, optometrists in Puerto Rico submitted a bill (P.S. 2634)—their seventh attempt in 15 years—to gain TPA privileges. On June 25, the commonwealth's legislature was scheduled to vote on the bill. On the steps of the Capitol building in San Juan, optometrists from Puerto Rico and students from the InterAmerican University of Puerto Rico School of Optometry (myself included) demonstrated our support for the bill and exercised our right to petition the government.

However, the Democratic process was halted when Senate President Thomas Rivera Schatz decided the bill should not be heard due to an insufficient number of favorable votes. I sat helpless in the public area of the Senate chamber as I watched democracy suffocate before my eyes and P.S. 2634 dissolve without due process. Such a sad event, and made even more unjust considering that the Puerto Rican government desires statehood but will not grant its citizens eye care that is equivalent to what other U.S. citizens receive.

Optometrists and optometry students stand in front of the capitol in Puerto Rico in support of their seventh TPA bill.

"Optometry has made every effort to amend the current law to best reflect scientific advancements and expansion of optometric education and scope of practice," says Angel Romero, O.D., dean of academic affairs at the IAUPR School of Optometry and one of the demonstrators in support of the bill. "Every attempt by optometry to amend the law has received fierce resistance from organized groups that do not have the welfare of the Puerto Rican people in their best interest. These groups have succeeded, and thus have kept optometric scope of practice decades behind our stateside colleagues."

Although this attempt was unsuccessful, we will continue to fight for our patients, our communities and ultimately our profession. The status sought by optometrists in Puerto Rico is not only for recognition, but also for the right to fully perform the profession for which we have been trained and to provide full-scope eye care to the many millions of people in Puerto Rico who desperately need it.

Ms. Paniccia is a fourth-year student at InterAmerican University of Puerto Rico School of Optometry.

New Optometric Society Has an Eye on CXL

Corneal collagen crosslinking, or CXL, is one of the hottest topics in eye care today. But, exactly how CXL is performed? What equipment is required? Where can you refer patients for this as-yet-unapproved procedure? Fortunately, a brand new group—the Optometric Crosslinking Society (OCXLS)—has the answers to these questions. The OCXLS was founded with one primary goal in mind: To inform all interested optometrists about CXL.

"We want to be the singular information source on CXL for all O.D.s and optometry students in the United States," says Andrew S. Morgenstern, O.D., the society's founder and president. "Currently, there is not a great deal of uniform information about CXL, which doctors are performing the procedure, the necessary instrumentation or the associated financial considerations. We intend to provide all of that information and more via meetings and lectures, our website and various social media outlets."

For more information, go to www.ocxls.org; send an email to CXLsociety@gmail.com; or follow the organization on Facebook at www.facebook.com/OCXLS and @CXLsociety on Twitter.

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Study: Alzheimer's Linked to Diabetes

For the first time, researchers have found direct experimental evidence that diabetes is linked to the onset of Alzheimer's disease. A large part of the study successfully employed an examination of the retina for signs of Alzheimer's disease.

This opens up the possibility of detecting Alzheimer's in the eye. "Our findings indicate that scientists may be able to follow the onset and progression of Alzheimer's disease through retinal examination, which could provide a long sought-after early warning sign of the disease," said study co-author Peter Frederikse, Ph.D., of the department of Pharmacology & Physiology at the University of Medicine and Dentistry of New Jersey (UMDNJ).

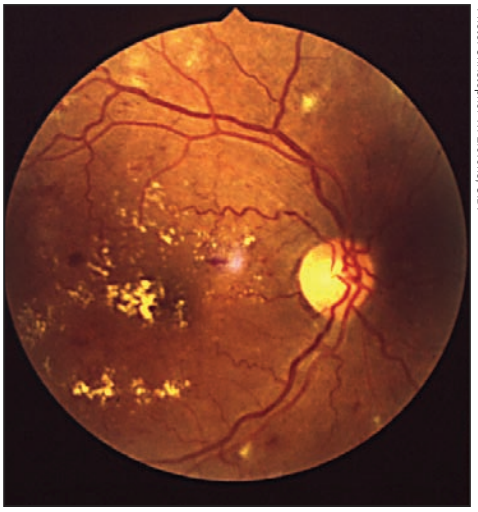

Photo: Christopher W. Lievens, O.D.

New research has found a link between diabetes and Alzheimer's disease. This opens up the possibility of using retinal exams to detect Alzheimer's in patients with diabetes (such as this one).

Prior research has shown that levels of brain insulin and its related receptors are lower in individuals with Alzheimer's disease. This recent study, published online in the *Journal of Alzheimer's Disease*, may suggest that diabetes actually appears to instigate Alzheimer's.

UMDNJ researchers collaborated with scientists from Northwestern University in Illinois and employed a physiological model of Alzheimer's neuropathology in diabetic rabbits. At the study's completion, they found a five-fold increase in amyloid-beta peptides—a hallmark of Alzheimer's disease—in the brain cortex and hippocampus. They also found significant amyloid-beta accumulation in ganglion and inner nuclear cell layers of the retina.

By contrast, when diabetes was not present, they detected no observable pathology in either the brain or the retina.

"Our study identifies emergence of AD [Alzheimer's disease] pathology in brain and retina as a major consequence of diabetes; implicating dysfunctional insulin signaling in late-onset AD, and a potential relationship between [amyloid-beta]-derived neurotoxins and retinal degeneration in aging and diabetes, as well as AD," the researchers wrote.

"In light of the near-epidemic increases in Alzheimer's disease and diabetes today, developing a physiological model of Alzheimer neuropathology has been an important goal," said Chinnaswamy Kasinathan, Ph.D., another member of the research team. "It allows us to identify a potential biomarker for Alzheimer's disease and may also make important contributions to Alzheimer drug testing and development."

Bitel CL, Kasinathan C, Kaswala RH, et al. Amyloid-β and Tau pathology of Alzheimer's disease induced by diabetes in an animal model. J Alzheimers Dis. 2012 Jul 11. [Epub ahead of print]

FDA Panel OKs Lucentis for Diabetic Macular Edema

The FDA's Dermatologic & Ophthalmic Drugs Advisory Committee voted unanimously to recommend approval of the 0.3mg dose of Lucentis (ranibizumab injection, Genentech/Roche) for treatment of diabetic macular edema (DME). The majority of the committee also recommended the 0.5mg dose.

The FDA generally follows advisory committee recommendations, although it is not required to do so.

The committee based its recommendation on data from Genentech's Phase III trials, RIDE and RISE, which evaluated the efficacy and safety of Lucentis in patients with DME. The primary endpoint was the percentage of patients who could read an additional 15 letters (three lines) or more on the eye chart after 24 months of treatment compared to a control group.

The 0.3mg dose achieved this endpoint in 38.6% of 125 patients, and the 0.5mg dose improved it in 50% of patients. By comparison, 14.4% of patients in the placebo group achieved this improvement.

Currently, there are no drugs approved to treat DME. The conventional treatment is laser photocoagulation, which can slow or stabilize vision loss. But few patients obtain improved vision.

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Vision Dangers in Drinking Water

By Cheryl G. Murphy, O.D., Contributing Editor

Do you know what's in your drinking water? Young and prenatal children who were exposed to the neurotoxic chemical tetrachloroethylene in drinking water in the 1970s are now, as adults, showing vision deficiencies—particularly in color discrimination. Unfortunately, this chemical is still in use today.

Researchers at Boston University's School of Public Health (BUSPH) performed a retrospective study of adults who grew up in an area of Cape Cod that is known to have had a water supply contaminated with tetrachloroethylene (also known as perchloroethylene—"perc"—or PCE). This dangerous chemical had leached into their water supply in the 1970s due to a faulty vinyl liner in the town's pipes.

The researchers found that those who had lived in that area, and who had been exposed to PCE during gestation to age five, now suffer a statistically significant reduction in color discrimination with Farnsworth testing.[1]

Also, when compared to those who were not exposed to the contaminated water, these adults had reduced contrast sensitivity to intermediate and higher spatial frequencies, although the differences proved to not be statistically significant.

"To the best of our knowledge, this is the first study to assess the associations between prenatal and early childhood exposure to PCE and adult vision," the authors wrote. "Exposure to PCE via drinking water during these critical periods of development may be associated with long-term subclinical visual dysfunction in adulthood, particularly color discrimination."[1] Further studies are needed to understand the wider implications of this finding, they added.

Today, discharge of PCE—from factories and dry cleaning facilities—is the major source of drinking water contamination across the country. The EPA has set a maximum contaminant level goal of zero for tetrachloroethylene in drinking water, though it has an enforceable maximum contaminant level limit of 0.005mg/L. The EPA describes tetrachloroethylene as a "colorless, organic liquid with a mild, chloroform-like odor."[2]

Over time, people who consume drinking water containing PCE (in excess of the maximum contaminant level) have a higher risk for liver problems and certain cancers.

Workers in the dry cleaning industry are frequently exposed to the chemical, which is used as an aerosol product. Occupational exposure to PCE on a regular basis via inhalation puts workers at a greater risk for liver and kidney damage, cancer, reproductive and developmental abnormalities as well as color visual impairment.[3] Adverse neurological effects among adults following exposure even to low PCE levels are well documented and include decreases in attention, cognitive function and memory.

1. Getz KD, Janulewicz PA, Rowe S, et al. Prenatal and early childhood exposure to tetrachloroethylene and adult vision. Environ Health Perspect. 2012 Jul 11. [Epub ahead of print]
2. United States Environmental Protection Agency website. Basic Information about Tetrachloroethylene in Drinking Water. Updated May 21, 2012. Available at: http://water.epa.gov/drink/contaminants/basicinformation/tetrachloroethylene.cfm.
3. Gobba F, Cavalleri A. Color vision impairment in workers exposed to neurotoxic chemicals. Neurotoxicology. 2003 Aug;24(4-5):693-702.

Myopia on The Bounty

Photo: HMS Bounty Organization LLC

Descendants of the famed mutineers of the British ship HMS Bounty have some of the lowest rates of myopia in the world. New research suggests they could help unlock the genetic code for the disease.

The Norfolk Island Eye Study examined eye problems in descendants of the Bounty sailors and their Polynesian wives who settled at Pitcairn Island after the mutiny in 1789, and later moved to Norfolk Island. Almost half the islanders can trace their ancestry back to the original Pitcairn population of just nine British mutineers, 12 Tahitian women and six Tahitian men.

"We found the rate of…myopia [among the Pitcairn descendants] is approximately one-half that of the Australian population, and as a result would be ranked among one of the lowest rates in the world," says David Mackey, M.D., of the Centre for Eye Research Australia. By contrast, other Norfolk Island residents had approximately the same rate of myopia as the Australian population.

Genetic differences in the island's inhabitants could lead to breakthroughs in the causes of myopia, which is increasing in prevalence in Australia, Dr. Mackey says. Future studies may allow for the identification of genes that differ between the two populations.

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People Would Rather Lose a Limb Than an Eye

Almost 70% of people around the globe would rather take a decade off their life or sacrifice a limb than go blind—but less than a third of those polled take basic steps to preserve their eyesight, according to a global opinion poll released by Bausch + Lomb. The company surveyed 11,000 consumers in the United States, Brazil, China, France, Germany, India, Italy, Japan, Russia, Spain, and the U.K. about their awareness, attitudes and behaviors related to eye health.

Key results from the "Barometer of Global Eye Health" survey found:

- If forced to choose, people would rather lose their sense of taste (79%), hearing (78%), one of their limbs (68%) or 10 years off their life (67%) than their eyesight.
- 75% of people would rather have their pay cut in half than have a permanent 50% decline in their quality of vision.
- 68% say they are knowledgeable about eye health, but just 21% had regular eye exams over the past five years.
- For those who did not have regular eye exams, 65% said they had not visited an eye doctor because they did not have any symptoms, and 60% because they had clear vision.
- Women are more likely than men to take steps to protect their vision, such as wearing sunglasses (81% vs. 77%), eating a healthy diet (82% vs. 75%) and refraining from smoking (79% vs. 73%).
- Married people are more likely than single people to have had a comprehensive eye exam in the past year (46% married vs. 38% single).

B+L hopes this research will help eye care professionals emphasize the need to expose and correct consumers' misconceptions about eye health.

For detailed results of "The Barometer of Global Eye Health," visit www.bausch.com/barometer.

FDA Asserts B+L 'Misbranded' PureVision2

The FDA, in a warning letter sent to Bausch + Lomb in early June, accused the company of marketing its PureVision2 contact lenses with an explicitly unapproved indication. The FDA referred to the device as "misbranded" and called for the company to immediately cease marketing PureVision2.

The letter stated that the company sought FDA approval for a revised labeling statement indicating the devices were "HD High Definition" and "ComfortMoist," yet the FDA denied this request in May 2011.

In response to the letter, B+L says it acted in good faith in regard to the claims in question, but respects the FDA's position and is actively working to resolve these concerns. The company has altered its marketing materials to reflect the FDA's concerns and is working with suppliers and customers to update their websites. It also stopped marketing PureVision2 and PureVision2 For Astigmatism as extended wear lenses. ■

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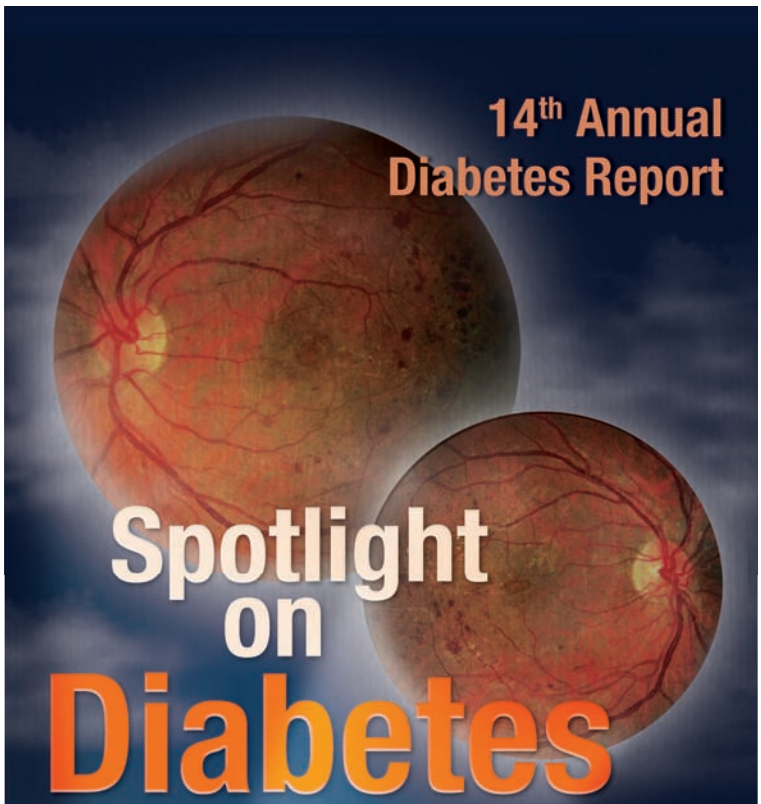

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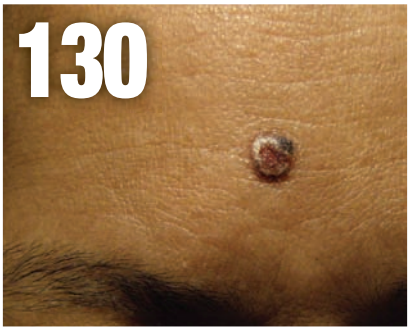

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*Post-hoc analysis of combined data from two studies using a contralateral conjunctival allergen challenge (CAC). Based on a scale of itching scores of 0-4, with 0 as no itching and 4 as severe itching. Ocular itching was evaluated 3 minutes after allergen challenge at onset and at 16 hours.

†(N=85; 95% CI=48.8, 70.5) ‡(N=82; 95% CI=48.3, 70.4)

References: 1. IMS Health, IMS National Prescription Audit™, August 2010 to February 2011, USC 61500 OPHTH ANTI-ALLERGY. **2.** Data on file.

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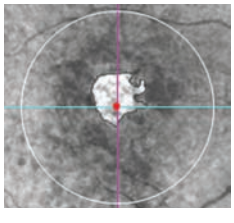

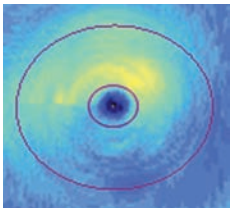

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Gluttons for Punishment

America's overeating is fueling a diabetes epidemic that shows no signs of slowing down. Here's why you should intervene.

By Jack Persico, Editor-in-Chief

Politicians sometimes tout "American exceptionalism"—the notion that we're uniquely different from the rest of the world because our cultural values of freedom and equality are enshrined in our constitution. This makes us a beacon of inspiration for emerging nations (so the theory goes). It's good fodder for stump speeches, even if our boasting does leave other countries feeling a bit slighted, especially those that share our values and would argue that there's nothing too unique about us after all.

But lately, one way we differ from the rest of the world *has* been harder and harder to avoid.

We're fat. Exceptionally fat.

A recent study of worldwide population characteristics found that although Americans comprise only 5% of the world's population, we account for nearly one-third of its obesity.[1] Compare that to Asia, which represents 61% of the global population but only accounts for 13% of global obesity.

That's not just embarrassing, it's unsustainable—for individuals and the environment. "If every country in the world had the same level of fatness that we see in the USA, in weight terms that would be like an extra billion people of world average body mass," Ian Roberts, one of the study authors, told the BBC. Some beacon of inspiration we are!

OK, so we overindulge. That's bad, but it's only half the story. We also don't burn off those extra calories nearly as much as we should. Another recent study found that roughly 41% of adults in America don't take part in enough physical activity.[2] Published in *The Lancet* last month, the study said that physical inactivity actually results in as many deaths as smoking.

"We should maintain cigarette smoking as public enemy number one, but we should move physical inactivity right up next to it," John Thyfault, Ph.D., of the University of Missouri, told WebMD.

And what do obesity and inactivity often lead to? Diabetes, of course. The *Lancet* study found that 8.3% of type 2 diabetes cases in the U.S. are linked to inactivity—and again we eclipse the worldwide average, which is 7.2%.

As we discuss this month on page 74, a new report finds that 26 million Americans currently have diabetes and triple that number have "pre-diabetes." At this pace, one third of our population will have diabetes by 2050. The report also finds that diabetic eye disease nearly doubled in the last decade.

It all adds up to a public health crisis—and that's where you come in. As the doctors who perform the vast majority of routine eye exams, you're in a unique position. More often than not, ophthalmologists see these patients after the horse has left the barn, when they've got full-blown proliferative diabetic retinopathy. But you're able to intervene at a point in patients' lives when lifestyle changes would still be able to interrupt the progression from diabetes to diabetic retinopathy to PDR.

It may seem impolite to bring up systemic health concerns like obesity in otherwise asymptomatic patients, but those are the people with whom you have the best chance of "bending the curve" on those grim charts predicting ever-increasing rates of DR.

I know that during a busy clinic day it's hard to find the time to address diet and lifestyle concerns, especially with an unreceptive audience (and for a consult that lacks a CPT code, no less). But it's a message your patients need to hear. Too often, we fail to connect the dots between our actions (and inactions) and their consequences for our long-term health. As I write this, the Olympics are showcasing the world's finest physical specimens, performing at their peak. Meanwhile, we Americans watch on the couch with a bag of Cheez Doodles.

Whenever the opportunity presents itself at your practice, try to instill a sense that, at least in our health risks, we're not as exceptional as we think. ■

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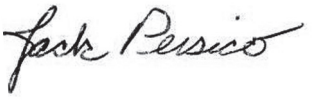
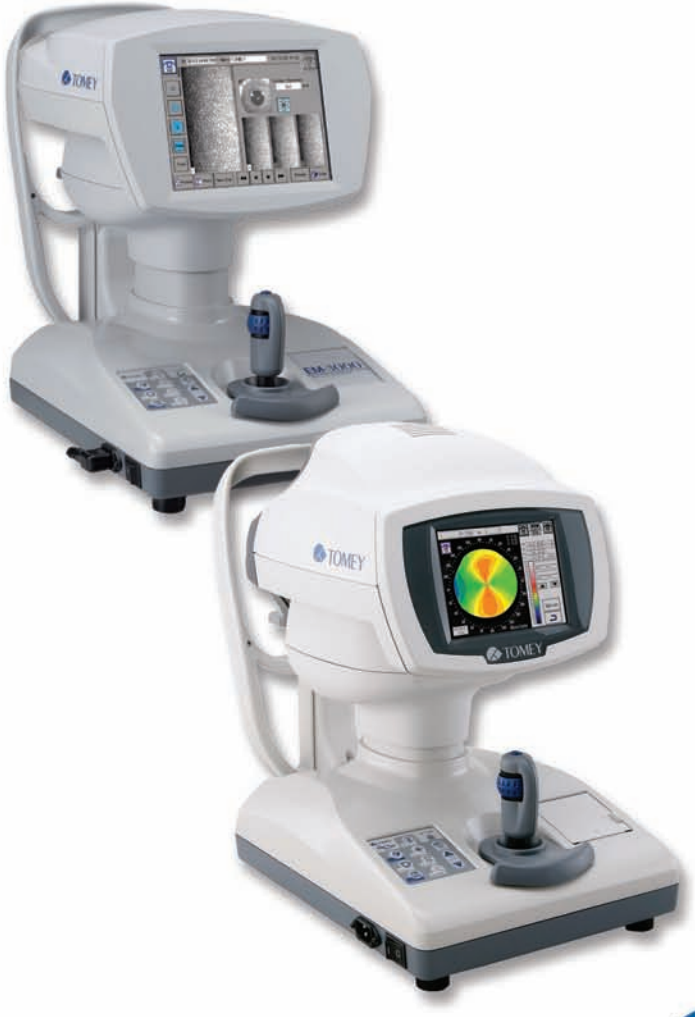


"AFTER MUCH THOUGHT, I CHOSE TOMEY."

"After I observed how many optometrists were upgrading their practice, I joined the group."

– Jay Stockman OD, New York Vision



"I have been an optometrist for over thirty years. In addition to a very successful private practice, *New York Vision,* I also have a contact lens manufacturing business, not to mention I am a senior examiner for the National Board of Examiners. As you can see, I get around a lot.

"Recently, on some of my visits to my clients, I noticed that many of them have set up an exam room with new Tomey equipment. The equipment is a **Specular Microscope** and a five-in-one **Auto Refractor/ Auto Keratometer Plus.** When I asked my clients why they chose this brand of equipment, I was told that it was the best on the market.

"After looking into how high reimbursement was, I could see how using this duo really made a significant contribution to their bottom line. I decided to order the equipment myself. I can't wait to start using them so I'll not only have the best diagnostic tools for my patients, but also will get a nice reimbursement for my practice."

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I'm so bored, I'm writing rhymes. What the heck, it fills the time.

By Montgomery Vickers, O.D.

Even in moments of forced lethargy, I can still produce one fine poem:

Lord, I am bored.

2 p.m., Thursday: Renee is checking in a new patient right now, and with the efficiencies we've developed by incredible investments in technology and advanced practice management policies that can only come from 32 years of consistent effort, that check-in should take only 45 minutes or so.

It's so boring, now I'm snoring.

2:03 p.m.: What else can I do? If I get all involved with catching up on my charting, and I am almost up to 1984, Renee will just interrupt me at the same time I perform an Internet search to see if I have to update a deceased patient's chart and, of course, I have not checked Facebook for 14 minutes and I may miss someone's important post that they are now leaving to go grocery shopping.

I'm so lazy right now that my mind is hazy, and how.

2:11 p.m.: There are, of course, other important duties I have as the Clinical Director of Vision Associates Incorporated—but the trash cans are not really full, so I would rather wait until the day ends. I could, I guess, read a journal article relevant to my career. Unfortunately, I left this month's issue of *Guitar World* at home in my porcelain office.

Perhaps I should call my legislator and try again to explain that we treat glaucoma, not guacamole. Maybe I should clean the instruments? No, just did that in 2009. I know: I'll call Mom! Great, now I'm bored and anxious, too.

Time flies when you're heaving sighs.

2:16 p.m.: Where did I put my rubber bands? I just had one on my desk last month. I seem to recall aiming it at the window and shooting myself in the forehead. I was so embarrassed that I proceeded to tell all my ensuing patients about it. I have learned that patients like it when I do something inane.

Then again, waiting here is driving me to the point that, if I could find that rubber band, I might be insane enough to try it again. I'd probably hit my eye. That would not be as entertaining to my patients—to see me rush out of the door in pain to visit my colleague down the street. I'll just let sleeping rubber bands lie.

I need a diversion. Perhaps a cat that's a Persian.

2:21 p.m.: While waiting for patients to get ready to be healed of their ocular afflictions, I check my e-mail seven or eight hundred times. Did you know that I have a rich uncle from Nigeria? And there are optometry jobs posted in Dallas? All I have to do is join the Air Force. Nobody's ever bored in the military, right?

Ate a mint that's heaven sent.

2:29 p.m.: Eating is a very good way to pass the time while waiting to see a patient. The bad news is that Renee has taken control of the icebox and my Jell-O shots have been replaced by carrots and olive medleys from around the globe. Is this what I am reduced to—eating what a goat would eat?

The patient is ready and her name is Betty.

2:32 p.m.: Hmm. My Mom's name is Betty. Could it be...?

"Hi, Mom. You look so 'purdy.' Yes, I know, my shirt is dirty."

No longer bored... But, oh, good Lord. ■

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Don't Overdo Diabetes Testing

Diabetes happens. Don't let these reimbursement mistakes happen, too.

By John Rumpakis, O.D., M.B.A., Clinical Coding Editor

Diabetes and its accompanying ocular manifestations are among the most common disease states in the optometric practice. Within this presentation lies great opportunity to help patients proactively with regular examinations and diagnostic procedures—but it also holds the temptation to see the patient as a bank account due to the frequent number of visits and the potential for over-testing.

Know the 'Medical Necessity'

Before we get into the specifics of diabetes and the special testing that comes with it, let's revisit the concept of medical necessity. Medicare's website defines "Medical Necessity" as: "*Services or supplies that are proper and needed for the diagnosis or treatment of the patient's medical conditions, are provided for the diagnosis, direct care and treatment of the patient's medical condition, meet the standards of good medical practice in the local area and aren't mainly for the convenience of the patient or the physician.*"

What does that mean in practical application? It means that the patient's medical record must clearly demonstrate that the service, procedure or test ordered was absolutely necessary to diagnose, treat or monitor the patient's condition.

Diabetes is one of the few diseases for which the Centers for Medicare & Medicaid Services (CMS) has adopted a true philosophy of preventive care. For example, in 2008, CMS allowed the provision of an annual comprehensive eye examination (92004/92014) solely with the systemic diagnosis of diabetes (250.XX) *even in the absence of diabetic retinopathy*. This is the only systemic condition (that I'm aware of) for which an annual comprehensive exam is allowed without apparent ocular complications.

Two Things to Watch For

One of the most common mistakes that O.D.s make for individuals with diabetes is ordering many special ophthalmological procedures simply because the patient has a diagnosis that can be tied to these procedures. Unfortunately, this compulsion for further testing is often unwarranted. (Simply having a "covered diagnosis" does not equate with the medical necessity of the test itself. For example, fundus photography may be a covered procedure when accompanied by the diagnosis of diabetic retinopathy—but only when the clinician actually sees a change that meets the definition of medical necessity as it relates to the patient's specific case.) This compulsion can then turn into angst when the carrier requires a review of the records to determine the medical necessity of all of the testing performed.

When O.D.s in such a situation contact me, I ask why they ordered the test in the absence of clinical information. The usual response: "Because I was afraid of missing something." The doctor is well intentioned, but misinformed.

Here's the bottom line: Your patients' medical insurance is not the same thing as your malpractice insurance. Ordering and performing special ophthalmological tests in the absence of establishing medical necessity for those tests in the medical record is simply inappropriate, and the monies paid by a carrier are definitely recoverable in a post-payment review.

The other area that requires special attention is the interpretation and report. This document must contain specific information about the reason why you ordered the test, your specific interpretation of the test results, a comparative analysis with previous test results (if any) and, most importantly, how the test impacted the course of care and outcomes for the patient. Properly done, an interpretive report in itself tells the story of why the test was ordered and performed, and what it added to the care profile of the patient.

Diabetes is a very common disease state, but unique in the preventive care allowed in the eyes of medical carriers. If you concentrate on taking care of the patient, rather than allowing the temptation of economics or fear of medicolegal jeopardy to drive decisions, and if you make sure that your medical record is meticulously documented and accurately reflects the "what and why" of the care you provided, then the medical coding of the case will take care of itself. ■

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Infiltrative Keratitis and Gram-Negative Bacterial Resistance to PQ-Aldox Lens Care Products

The rate of infiltrative keratitis especially with daily wear silicone hydrogel lenses has been reported with greater frequency.[1-4] Infiltrative keratitis is associated with several factors[1-8] including lens care solutions,[9,10] lens type,[1,3] smoking,[5] and bacterial bioburden.[5-8] **Contact lens associated infiltrative keratitis (CLAIK) has been reported at higher rates in particular with polyquaternium (PQ)-Aldox (myristamidopropyl dimethylamine) based Multi-Purpose Solutions (MPS).[1-5,9]**

Notably, CLAIK has repeatedly been associated with one PQ-Aldox MPS, Opti-Free RepleniSH in independent studies.[1-4] In one report, this solution was being used in 71% of CLAIK cases.[3] **Importantly, there has been no demonstrated correlation between transient, solution related corneal staining and inflammatory keratitis.[11]**

Low levels of lens case contamination may occur with any MPS or peroxide system in asymptomatic patients, but gram-negative contamination was reported highest with Opti-Free RepleniSH.[12] Recent scientific findings in patients using lens care solutions with CLAIK, demonstrate case contamination with certain gram-negative clinical isolates, the predominant species being *Stenotrophomonas maltophilia* and *Achromobacter.*[13] These gram-negative bacteria have also been cultured in the lens cases of patients using PQ-Aldox MPS.[12,14] **Additional research has shown that these clinical isolates are resistant to a PQ-Aldox MPS and can re-grow during storage in PQ-Aldox MPS in as few as 6 days.[14-16]** Non-Aldox PQ-based MPS, such as those containing PHMB, and peroxide lens care solutions have demonstrated excellent biocidal efficacy against these same clinical isolates.[14-17] Table 1 presents biocidal efficacy against clinical isolates of *Achromobacter* and *Stenotrophomonas* when exposed to PQ-Aldox MPSs and a PHMB-PQ MPS.[18] Lens care solutions that are ineffective against these clinical isolates may be prone to case contamination and CLAIK may result directly from these bacteria and/or their endotoxins being repeatedly exposed to the ocular surface.[14]

Further investigation is warranted to understand the causality between infiltrative keratitis events and the use of PQ-Aldox MPS products. **The inefficacy of PQ-Aldox MPS against clinical isolates cultured from CLAIK patients should be considered by eye care practitioners in recommending lens care systems for their patients.**

Log Unit reduction		
	Achromobacter*	Stenotrophomonas*
Biotrue® MPS (PHMB-PQ)	2.9	3.5
OPTI-FREE PureMoist (PQ-Aldox)	0.1	1.2
OPTI-FREE RepleniSH (PQ-Aldox)	0.0	1.3
OPTI-FREE Express (PQ-Aldox)	0.2	1.2

Table 1. MPS Biocidal Efficacy Against Achromobacter and Stenotrophomonas Clinical Isolates Associated with CLAIK[18]

CLAIK has the potential of creating a significant economic burden on patients[19] and may contribute to patients choosing to stop wearing lenses. **Switching patients to MPS products with broad antimicrobial efficacy and proven biocompatibility, along with recommending appropriate lens and lens case cleaning regimens,[6] may help to prevent CLAIK, minimize risk for future recurrence[20] or contact lens drop out.**

Biotrue® MPS from Bausch + Lomb has proven biocompatibility and also demonstrates excellent disinfection efficacy compared to competitive multi-purpose solutions,[21,22] even against clinical isolates such as *Stenotrophomonas* and *Achromobacter, which are known to be associated with corneal infiltrative keratitis.*

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HL 5784-1

2012 "New Look" Office Design Contest

Objective: *Review of Optometry*'s "New Look" Office Design Contest recognizes optometric practices that incorporate functionality, optimum use of space and stylistic appeal with up-to-date clinical technology.

Eligibility: Newly built offices and office remodels or expansions completed between January 1, 2010 and June 30, 2012 are eligible to enter the 2012 "New Look" Office Design Contest.

Judging: Entries will be judged by a panel of fellow optometrists who have been previously recognized for their expertise in office design.

The contest will be divided into two categories:

- Renovation of Existing Office
- New Office/Expansion

Awards: "Office Design of the Year" will be awarded to the best overall facility, based upon functional design, efficient interior space planning, style and appropriate integration of optometry equipment and technology. There will be a total of four winners—one small practice and one large practice will be selected from each category.

Each winner will receive an engraved office plaque recognizing the practice's achievement, in addition to editorial coverage online and in the December 2012 print edition of *Review of Optometry*. Honorable mentions will receive online coverage.

How to Enter: Send your completed contest entry form and three to four high-resolution images to Senior Editor Colleen Mullarkey. Images should illustrate the contest's design principles. They must be no less than 300 dots per inch (dpi) and should be saved as .tif or .jpg files.

- ***Email:*** cmullarkey@jobson.com
- ***Mail:*** Review of Optometry
  New Look Office Design Contest
  11 Campus Blvd., Suite 100
  Newtown Square, PA 19073

All entries must be received by September 15, 2012.

2012 Office Design Contest Entry Form

PLEASE PRINT OR TYPE

Name and Title: _____

Practice Name: _____

Address: _____

City: _____ State: _____ Zip: _____

Phone: _____ Project Completion Date: _____

Website: _____ Email: _____

DESIGN BASICS

Contest Category: ☐ Renovation of Existing Office ☐ New Office/Expansion

Practice Size: ☐ Small (Gross Revenue < $400,000) ☐ Large (Gross Revenue > $400,000)

Estimated Total Project Cost: _____

Total Net Square Footage of Practice: _____

Entries that do not meet all requirements or are not received by the deadline of September 15, 2012 will be disqualified.

Submission of an entry constitutes consent to use the entrant's name and/or photograph, including posting on the *Review of Optometry* website and/or related print and electronic publications, without compensation unless prohibited. All photos become property of *Review of Optometry* and will not be returned. Only one entry per office will be accepted.

Entries must be composed of original, authentic, unpublished material and must be the sole property of the entrant, not previously submitted in any other contest. *Review of Optometry* is not responsible for lost, late, misdirected, incomplete, or postage-due entries. Submission of your photo gives consent for *Review of Optometry* to place the image in its image bank on a nonexclusive basis for noncommercial use.

Signature: _____ Date: _____

Questions or Concerns: Please contact Colleen Mullarkey, Senior Editor/Web Editor, at cmullarkey@jobson.com or (610) 492-1005.

2012 Office Design Contest Entry Form

DESIGN OBJECTIVES

Explain how your office design incorporates these concepts and explain any obstacles you overcame. Please submit your responses to each of the following four questions, limiting each response to 150 words.

1. Function: How does your new office/remodel improve efficiency for your staff and effectiveness with your patients?

2. Optometric Equipment: How was currently installed optometric equipment integrated into the overall design of your facility? List pertinent upgrades that were made and/or additional components that will be added in the future.

3. Ergonomics: How has your new office improved the ease of providing eye care? Consider specific design decisions made regarding the layout of your business and clinical work areas (especially the exam rooms and front desk), placement of equipment and computer components, and positioning of doctor(s) and staff.

4. Aesthetics: How does the look of your new office/remodel affect or improve your staff and patient experience? How has your new office design attracted new business and/or expanded your patient base?

JOSHUA MARC LAHIFF, OD

A Growing Practice Is Built on Happy Patients

Garnering new patients through **word-of-mouth referrals** is more than a great marketing strategy—it is the key to success that endures the test of time.

Successful practices see more patients

Focusing your practice on getting new patients rather than dollars per patient is a more pragmatic—and preferable—business approach. This is true for any practice. In a 2009 study of independent practices, it was reported that the practices with the greatest gross revenue see three times as many patients as the average practice. Furthermore, there is little difference in the gross revenue per exam between rural and urban settings, small and large practices.[1]

Increased patient traffic and improved exam productivity have major impacts on practice revenue. In fact, another 2009 study found that, on average, 52% of a practice's gross revenues come from exam fees alone.[2] This constitutes nearly 2/3 of gross income.[1,2]

According to Dr Josh LaHiff, practicing optometrist in Cheyenne, WY, increasing patient traffic is all about providing patients with an outstanding experience: "You want to be able to provide them [patients] with such an experience that they're going to invest more in your clinic." Dr LaHiff should know. He sees at least 30 patients per day. Which is impressive considering there are 28 other eye care professionals in Cheyenne, a town with a population of only 50,000.

Success is built on a happy patient experience

Increasing patient traffic through referrals is often as simple as delivering excellent service and the healthiest products. "To be the best, you have to use the best. If you wow the patient, treat them like gold, that's how you really generate those referrals," says Dr LaHiff.

That's why Dr LaHiff believes the most effective approach to achieving profitable, long-term patient relationships is to "do what's best for the patient, even if it may not be the most profitable option for the doctor initially, because it is what is right, and the profit will come as an annuity in return visits and the happiness of the patient."

High patient satisfaction inside and outside of the practice is the most effective catalyst for generating new patients. This can be especially true for contact lens patients. A survey of 1086 patients found that those who are happy in their contact lenses are nearly 2x more likely to recommend their eye doctor than those who are unhappy in their lenses.[2]

ACUVUE® Brand—proven to keep patients satisfied

The proven method to achieve satisfaction is to use a product with consistently successful results. Dr LaHiff believes that, "When you use a product that you know is going to work time and time again, it cuts your chair time down and it's easier for you, your staff, and your patients to put them in something that's comfortable."

ACUVUE® Brand delivers outstanding comfort and consistent patient satisfaction. In fact, 9 out of 10 ACUVUE® patients are satisfied in their contact lenses.[2]

And happy ACUVUE® wearers refer their eye doctor: 8 out of 10 satisfied ACUVUE® OASYS® Brand Contact Lens and 9 out of 10 1-DAY ACUVUE® MOIST® Brand Contact Lens wearers would recommend their doctor to others.[2]

According to Dr LaHiff, "The more you use a superior product, the better the experience is going to be for the patient." And that's been a proven strategy for the success of his practice. ■

Joshua Marc LaHiff, OD, is a partner and practicing optometrist at Cheyenne Vision Clinic in Cheyenne, WY, and clinical instructor for the Illinois College of Optometry in Chicago, IL. He received his doctor of optometry degree with honors from the Pacific University College of Optometry in Forest Grove, OR.

Dr LaHiff is a member of numerous associations and serves as a speaker and professional consultant for several medical companies, including VISTAKON® Division of Johnson & Johnson Vision Care, Inc. He was compensated for this article.

References: 1. Management & Business Academy: Practice Profile Report, 2009. **2.** Data on file. Johnson & Johnson Vision Care, Inc. 2011-2012.

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The expanded CE program will focus on disease diagnosis and treatment, clinical applications of new products and business solutions. **By Jane Cole, Contributing Editor**

Optometrists who attend this year's Vision Expo West meeting in Las Vegas can choose from more than 200 hours of clinical CE content and an additional 100 hours of business-related courses geared to help you boost your bottom line. This year's meeting will be held at the Sands Expo & Convention Center. Education courses will be offered from Sept. 5-8, and the exhibit hall will be open from Sept. 6-8.

"The International Vision Expo & Conference team constantly looks for ways to enhance the attendee experience at our show," says Tom Loughran, vice president for Reed Exhibitions, which co-owns International Vision Expo with The Vision Council. "This year, we've enhanced a variety of programs and events to do just that—from our concerted effort to collect and promote exhibitors' show specials to additional networking opportunities and parties to expanding the number of travel and entertainment discounts offered," he says.

In addition, they've expanded the conference program with new sessions like Government Regulations and Sales U "to help organizations' stay abreast of the latest requirements and hone their skills in this turbulent economy."

Here's a snapshot of key meetings and courses you won't want to miss:

• ***Courses led by Ron Melton, O.D., and Randall Thomas, O.D., M.P.H.*** Their sessions include:

– *On Call with Melton and Thomas (Friday, Sept. 7, 2:45 p.m. – 4:45 p.m.).* Gain a better understanding of challenges encountered during weekend call in the tertiary care environment.

– *Glaucoma Grand Rounds (Friday, Sept. 7, 5:00 p.m. – 6:00 p.m.).* This team-taught course will

Vision Expo will "double down" on CE opportunities for optometrists at this year's meeting, offering over 350 hours to choose from.

share numerous patient presentations in a case-study/grand rounds format. Emphasis will be on the diagnostic work-up, assessment of risk, selection of drug therapy and follow-up care.

– *Melton and Thomas Treatment Guidelines (Saturday, Sept. 8, 8:30 a.m. – 11:30 a.m.).* The Melton-Thomas team will discuss some common and not-so-common disorders often seen in the primary care optometric practice. Current information and recommendations are discussed regarding approved drugs and treatment modalities as well as off-label use of drugs.

• ***Assessing & Selecting Managed Vision Care Plans (Wednesday, Sept. 5, 12:15 p.m. – 1:15 p.m.), Mark Johnson, A.B.O.C., N.C.L.E.C.*** This course will help you evaluate different managed care plans to determine which are right for your practice.

• ***What's New in Eye Care? (Wednesday, Sept. 5, 1:30 p.m. – 3:30 p.m.) Moderator Kelly Kersick, O.D.*** This course will be a discussion of EHR benefits, challenges and processes of implementing into today's optometric practice.

• ***Corneal Collagen Cross-Linking in Management of Corneal Ectasia (Wednesday, Sept. 5, 3:45 p.m. – 5:45 p.m.), Melissa Barnett, O.D.*** In patients with progressive keratoconus and post-LASIK corneal ectasia, collagen crosslinking (CXL) is a new treatment that uses riboflavin and ultraviolet light to strengthen the cornea. This course offers a comprehensive overview of CXL for the eye-care practitioner.

• ***EHR Survival: Deployment and Rewards, (Thursday, Sept. 6, 2:45 p.m. – 3:45 p.m.), Phillip Gross, O.D.*** This one-hour course will discuss the critical issues and steps involved when deploying an integrated EHR system, whilefocusing on the realities, challenges and rewards. Real-world examples will help you successfully prepare and execute your own transition to EHR.

• ***Point-Counterpoint Glaucoma (Friday, Sept. 7, 8:30 a.m. – 10:30 a.m.), Robert Wooldridge, O.D., and Murray Fingeret, O.D.*** This course will review controversial areas in diagnosis and management of glaucoma. Items to be addressed include: choosing the best adjunctive medication, contemporary models of care, the role of surgery as an initial therapeutic modality and new diagnostic instruments (optic nerve imagers, newer perimeters).

• ***Using the OCT Beyond the Macula (Saturday, Sept. 8, 8:30 a.m. – 9:30 a.m.), Diana Shechtman, O.D.*** This lecture presents challenging cases in which OCT analysis played a key role in the evaluation of retinal conditions that go beyond macular disease.

• ***Board Certification Review.*** Courses include how to prepare

(continued on page 89)

Mark Your Calendar for These Special Events

Wednesday, September 5th, 7:30 a.m.-12:00 p.m.

• Eyefinity's "Focus 2012: Embracing the 'i' Craze," Eyemax Theatre, Conference Area (Room 503). This program will explore how today's optometric practice is being transformed by new mobile technology. The keynote address is "The Future of Health: Where Can Technology Take Us?" presented by Dr. Daniel Kraft, a Harvard University and Stanford University trained physician and scientist. A panel discussion focusing on the growth of mobile technology throughout the optometric industry will follow.

Thursday, September 6th

• Association of Practice Management Educators (APME) Business Meeting, Level 3 (Room 3401B), 8:00 a.m. – 6:00 p.m.

• National Association of Optometrists & Opticians, Level 3, Room 3003, 7:30 a.m. – 12:00 p.m.

• Professional Eye Care Associates of America (PECAA) Business Meetings, Level 3 (Room 3401A), 1:00 p.m. – 5:00 p.m.

• Doctorfest: Medical & Scientific Theater (Booth 4121), 5:00 p.m. – 6:00 p.m.

• Canada Reception with Breton Communications, located in the Club Vision Lounge, 5:00 p.m. – 6:00 p.m.

• Humana Reception, Conference Area (Room 606), 5:30 p.m. – 7:00 p.m.

• Opening Night Kick Off Party, TAO Nightclub, 10:30 p.m. – 11:30 p.m. Guests who present their badge and ID will receive complimentary admission and one-hour of open bar from 10:30 p.m. – 11:30 p.m.

Friday, September 7th

• Independent Doctors of Optometric Care (IDOC) Luncheon, Conference Area (Room 606), 12:00 p.m. – 2:00 p.m.

• Vision Source Experience, Level 3, (Rooms 3404, 3405, 3406), 3:00 p.m. – 6:00 p.m.

• Job Search Meet and Greet, Medical & Scientific Theater, 5:00 p.m. – 6:00 p.m.

• Southern California College of Optometry, Palazzo Hotel, Celebrity Car event, time TBD.

• O.D.s on Facebook Party co-hosted with Dr. Alan Glazier, Lavo Nightclub, 6:00 p.m. – 7:00 p.m.

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Give Kids' Ocular Allergies a 'Time Out'

Allergy is more common in children than in adults—and can be more difficult to diagnose and treat. **By Andrea Thau, O.D., Ida Chung, O.D., and Scott Richter, O.D.**

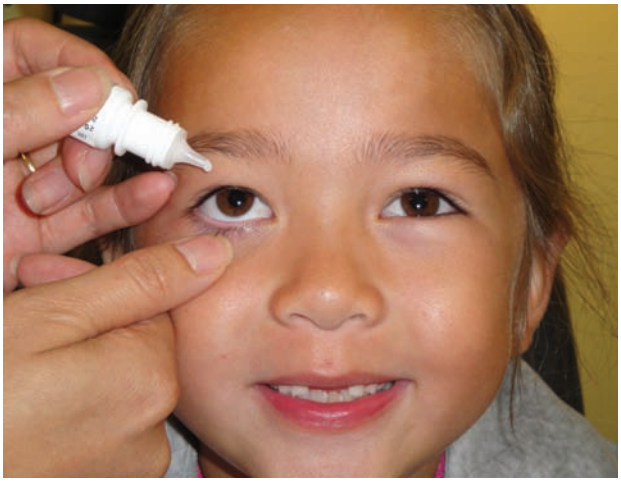
We see patients with external allergic reactions almost every day. This isn't surprising, considering it occurs in an estimated 20% to 25% of the population—and in up to 40% of children.[1] But confirming and then treating a diagnosis of allergy is more difficult in children, for all the obvious reasons. Because we need to differentiate and diagnose allergic presentations with such frequency, here is a brief review of allergic conjunctivitis revolving around the pediatric population.

Pathophysiology

Allergic reactions generally arise from prior exposure to an allergen, resulting in sensitization. Upon re-exposure, allergen-specific IgE antibodies bind to receptor sites in mast cells. This causes a release of chemical mediators from the mast cells and mast cell degranulation. While there are a number of agents released from the mast cells, histamine plays a major role in the "immediate allergic response," resulting in increased vascular permeability, chemosis and redness.[2]

With degranulation there is some degree of accompanying inflammatory response, with IgE cross-linking leading other mediators to recruit cells for a "late allergic response," some six to 12 hours later.

The allergic immune response is complex and, in children, may begin before a baby is born. Allergic disease in infants is influenced by the maternal immune response during pregnancy. Genetic, environmental, nutritional and immunologic factors during pregnancy play a role in a child's propensity to develop allergic sensitization and subsequent allergic disease.[3]



The key to instilling drops in children is to not frighten or trick them. But once you broach the subject, be ready to instill the drops. The longer you wait, the harder it will be.

What's the Diagnosis?

As always, making the appropriate diagnosis leads to the best choice of treatment. This is no different for allergic disease in children. Children have active immune systems, and allergic reactions in children, with varied presentations, are quite common. We must first differentiate allergic disease from viral, bacterial and dry eye presentations. (*See "Basic Differential Diagnoses for Ocular Allergy," page 38.*) Then, we must differentiate the type of allergic reaction. Most ocular allergic reactions (greater than 80%) are mild, but allergic reactions can range from mild to severe conditions, such as atopic keratoconjunctivits and

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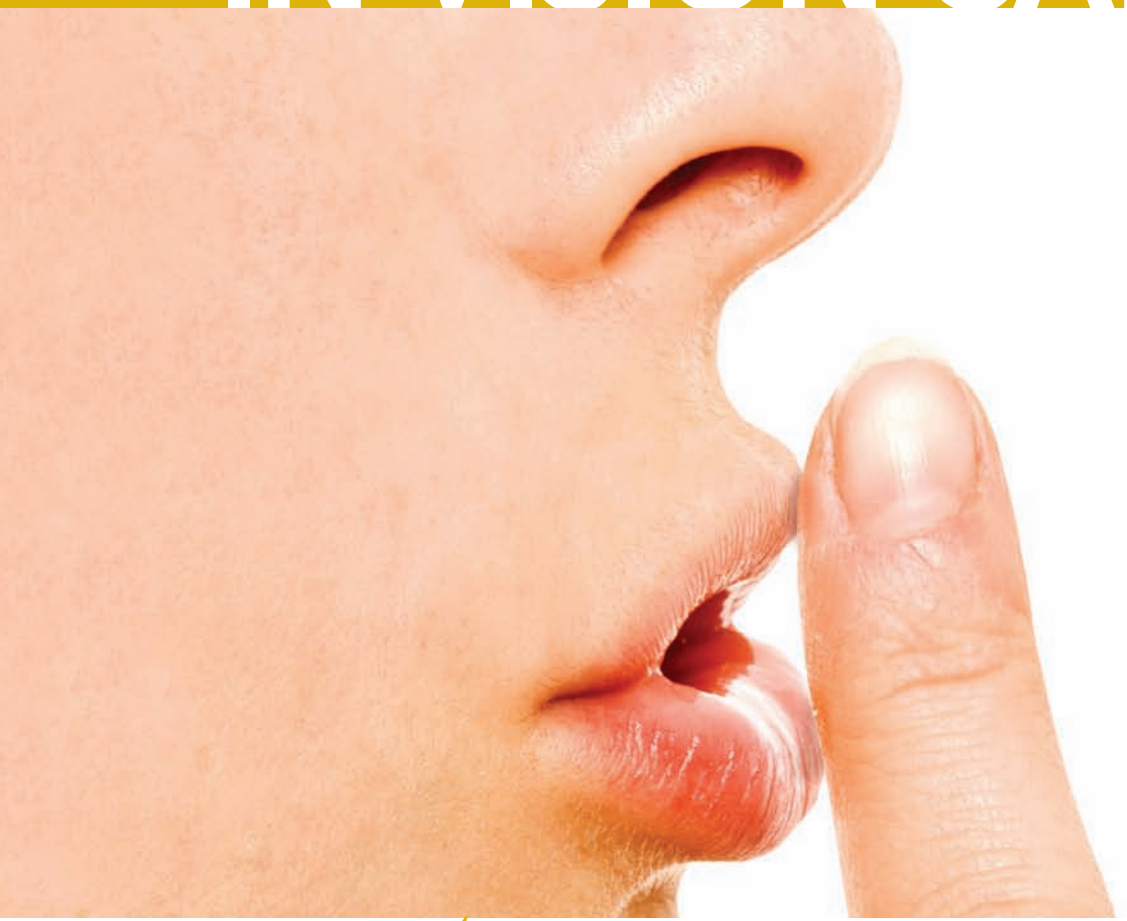

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Basic Differential Diagnoses for Ocular Allergy

	Allergy	Viral	Bacteria	Dry Eyes
History	allergen exposure	upper respiratory infection	other family member	systemic disease
Duration	often chronic	recent onset	acute recent onset	chronic
Symptoms	itching	foreign body sensation, irritation, pain	irritation, low-grade pain	sandy, foreign body sensation
Laterality	bilateral	bilateral; may be asymmetric	unilateral or bilateral	bilateral
Hyperemia	mild to moderate	often moderate to marked	moderate to marked	mild
Discharge	watery, stringy	watery	mucopurulent	tearing
Papilla	marked	mild	mild to moderate	minimal to mild
Follicles	moderate	moderate to marked	varied	minimal to mild

vernal conjunctivitis, which can have more serious consequences.

• ***History***. Differential diagnosis starts with history. Admittedly, history can be misleading, with the limitation that it must sometimes be obtained from the parents, but it can lead directly to the correct diagnosis. Remember that none of the following is a rule inviolate, only a general guide.

Ask pointed questions to obtain specific answers. A history of exposure to a known allergen for that patient—for example, "My daughter was playing with her friend's Persian cat"—can lead to a diagnosis of acute allergic conjunctivitis. The history that "my child gets red watery eyes every year at this time," can lead to a diagnosis of seasonal conjunctivitis (associated with seasonal exposure to known allergens). The history that "my child's eyes are red and tearing all year long," can lead to a diagnosis of chronic allergic conjunctivitis (with the known or unknown allergen being environmental in nature). Kids who suffer from perennial allergic conjunctivitis often have a history of allergic rhinitis, atopic dermatitis and/or asthma.

Because most viral and bacterial conjunctivitides are of limited duration, the chronicity of the problem may point to allergy—unless you can locate a source of re-infection, such as in canaliculitis or chronic blepharitis.

While dry eye in children may cause long-term problems, there usually is an associated systemic etiology or the presence of chronic blepharitis. Although only about 1.5% of healthy children have dry eye symptoms, the presence of a symptom such as burning is an indication of possible systemic disease.[4] A health history of systemic disease, such as rheumatoid juvenile arthritis or Sjögren's syndrome, may point to dry eye, but a history of asthma or atopic dermatitis may lend credence to a diagnosis of an allergic etiology. Conversely, a history of recent upper respiratory infection, or of a recent eye infection in a sibling, may point to a bacterial or viral etiology.

• ***Patient symptoms***. The symptoms presented aid diagnosis as well. Complaints of itching are strongly associated with allergic conjunctivitis, while complaints of burning and foreign body sensation are associated with dry eye. Complaints of lids matted together, along with irritation and soreness, point to a bacterial etiology. Irritation and pain suggest a viral etiology. Photophobia is more commonly associated with the corneal involvement seen with viral etiologies.

Remember that allergic conjunctivitis is almost always bilateral, while viral conjunctivitis is bilateral but often asymmetric, and bacterial conjunctivitis can be unilateral. The bilateral symmetry of dry eye problems in children vary based on the etiology.

In short, symptoms of itching with a stringy and watery discharge suggest allergy. Watery discharge and photophobia suggest a virus. Mucopurulent discharge suggests a bacterial cause. And, dry eye is usually accompanied by tearing.

• ***Clinical signs***. The diagnosis of allergic conjunctivitis is a clinical decision that is not based on laboratory findings. So, the clinician must garner a series of careful observations to construct a presentation, which then leads to the appropriate diagnosis.

In acute allergic conjunctivitis, bilateral lid edema is common in association with marked conjunctival chemosis. Bilateral lid edema is also seen with viral conjunctivitis, but usually with a lesser degree of conjunctival chemosis. As with many allergic diseases, clinical manifestations decrease with age. Thus, chronic allergic conjunctivitis leads to milder lid edema and conjunctival chemosis over time. Kids who suffer from chronic allergic conjunctivitis can develop dark discoloration below their eyes, called allergic shiners.

If possible, evert and inspect the upper lid. This may not be possible with all children, of course. But, if you pull up the lid high enough and you look from down below, you can often get an idea of the underlying conjunctival mosaic. Children with allergic conjunctivitis usually show a marked papillary reaction, but also demonstrate some follicles. The absence of petechial conjunctival hemorrhages—as is sometimes seen in viral disease or in association with the vascular fragility caused by some bacteria—helps to rule against an infectious etiology.

Pay careful attention to the papillary reaction, as the degree of the reaction can point to a more serious diagnosis. Severe papillary reaction asymmetrically involving the upper tarsal plate may indicate vernal conjunctivitis.

Vernal conjunctivitis is a recurrent seasonal disorder seen most frequently in young males and more prevalent in warmer climates.[5] It is characterized by an extensive papillary reaction disproportionately involving the upper tarsal conjunctiva, limbal involvement with the accumulation of eosinophils and cellular debris forming yellow/white mounds (Horner-Trantas dots), and may lead to corneal neovascular intrusion and the development of a sterile corneal shield ulcer (so called because of its shape).

Atopic conjunctivitis often starts at a young age, but can be seen in the pediatric or adult population. It is seen in association with asthma and atopic dermatitis. Check the periocular skin and lids for an atopic reaction, such as redness, blisters, drying or thickening with a superficial crusting rash. There is a marked papillary reaction, which sometimes is manifested more inferiorly.

Hyperemia is typical in any form of conjunctivitis. In the common form of allergic conjunctivitis, it is usually mild. Marked hyperemia is more common in viral and bacterial conjunctivitis.

Corneal involvement in allergic conjunctivitis is most often minimal or limited to superficial punctate keratopathy. In the rare extremes of vernal or atopic disease, corneal involvement is significant, with the potential for a permanent effect on vision.

What's the Treatment?

Making the appropriate diagnosis directs us to the appropriate course of treatment. Remember, prevention or avoidance of the allergen is always the first and usually the most effective course of action. For children who are allergic to cat dander, do not let them play with cats. For children with seasonal

Topical Ophthalmic Anti-Allergy Medications

Brand Name	*Generic Name*	*Manufacturer*	*Pediatric Use*	*Dosage*
Alamast	pemirolast potassium 0.1%	Vistakon Pharm.	3 years	q.i.d./.b.i.d.
Alaway (OTC)	ketotifen fumarate 0.025%	Bausch+Lomb	3 years	b.i.d.
Alocril	nedocromil sodium 2%	Allergan	3 years	b.i.d.
Alomide	lodoximide trometh-amine 0.1%	Alcon	2 years	q.i.d. up to three months
Bepreve	bepotastine besilate 1.5%	ISTA Pharm.	2 years	b.i.d.
Claritin Eye (OTC)	ketotifen fumarate 0.025%	Schering-Plough	3 years	b.i.d.
Crolom	cromolyn sodium 4%	Bausch+Lomb	4 years	q.i.d.
Elestat	epinastine HCl 0.05%	Allergan	3 years	b.i.d.
Emadine	emedastine difumarate 0.05%	Alcon	3 years	q.i.d.
Lastacaft	alcaftadine 0.25%	Allergan	2 years	q.d.
Opticrom	cromolyn sodium 4%	Allergan	4 years	q.i.d.
Optivar	azelastine hydrochloride 0.05%	Meda	3 years	b.i.d.
Pataday	olopatadine hydrocloride 0.2%	Alcon	3 years	q.d.
Patanol	olopatadine hydrocloride 0.1%	Alcon	3 years	b.i.d.
Refresh Eye Itch Relief (OTC)	ketotifen fumarate 0.025%	Allergan	3 years	b.i.d.
Zaditor (OTC)	ketotifen fumarate 0.025%	Alcon	3 years	b.i.d.

allergies, try to limit their exposure to known allergens—is going to the botanical garden really a good idea at this time in the season? For children with chronic allergy, consider removing potential environmental allergens of dust mites or mold spores.

Once exposed, the treatment reflects the severity of the presentation.

• ***Mild presentations.*** For mild allergic presentations, artificial tears and cool compresses work well. The artificial tears help flush allergens out of the eye, and decrease rubbing by increasing comfort. Cool compresses decrease swelling and blood flow to the area, thereby inhibiting the allergic response. Artificial tears and cold compresses do not treat the underlying allergic response, but can provide relief from ocular symptoms in mild cases. This may be the most appropriate choice for very young children under age two when there is no pediatric-approved topical ophthalmic drug.

Topical vasoconstrictors are of limited value, although more effective when used in combination with a topical antihistamine. Mast cell stabilizers prevent degranulation, thus interrupting the allergic cascade at a later point. This also means that mast cell stabilizers have a slower onset of action—typically five to 14 days.[2] For this reason, they are best used in advance of the known approach of seasonal allergic conjunctivitis.

• ***Moderate presentations.*** Today, the mainstay of treatment is the family of combination antihistamine-mast cell stabilizers. A number of these medications are available, most approved for children age three and older, and two approved for children as young as two years of age. These have a rapid therapeutic onset, and they can be safely used over an extended period of time. They provide effective relief for most forms of allergic conjunctivitis. (*See "Topical Ophthalmic Anti-allergy Medications," at left.*) Allergy meds with daily or twice-daily dosing obviously are better for both kids' and parents' schedules.

Antihistamine is the fast-acting component and the mast cell stabilizer is slower acting but prevents the allergic cascade from occurring. For those children who suffer yearly from seasonal allergies, starting a topical antihistamine-mast cell stabilizer six weeks ahead of allergy season can often prevent a child from having seasonal ocular allergic reactions.

• ***Severe presentations.*** The use of steroids is reserved for when it is clearly indicated, such as for acute and chronic forms of allergic conjunctivitis, and then only for a limited period. A topical steroid may help the patient with a marked reaction to an allergen (i.e., acute allergic conjunctivitis) more rapidly regress toward a normal baseline.

Acute reactions can be treated with a steroid for a short period of time, usually three to five days. This can then be followed up with the use of an antihistamine-mast cell stabilizer. In seasonal chronic disease, breaking the cycle of allergic reaction may take longer, requiring steroids for seven to 10 days.

In the more severe forms of allergic conjunctivitis, such as vernal or atopic keratoconjunctivits, the use of a mild steroid, such as

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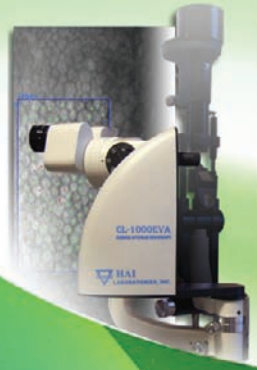

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fluorometholone or loteprednol, is indicated.[6] In these cases, we must increase comfort and delimit the progression of these diseases as quickly as possible. Treat with a steroid for seven days at minimum; however, the steroid may be required for even longer—two weeks or more—to reach a reasonable baseline. After improvement of symptoms, you can switch the patient to an antihistamine-mast cell stabilizer combination drop.

With adjunctive corneal involvement, an aggressive approach is warranted (e.g., concurrent use of prednisolone eye drops q.i.d. and a topical antibiotic drop as indicated).[5] When there are concurrent signs of allergy, such as conjunctival chemosis or eyelid edema along with itching, use steroids.

Be mindful, as always, of the steroid's adverse effects, including increased intraocular pressure, viral infections and cataract formation.

While allergic eye disease is most often not serious or vision threatening in nature, it can profoundly affect a child's quality of life (and the parents' as well).

Considering its prevalence in the pediatric population, it's up to us to recognize these signs and symptoms, and intercede in an appropriate manner. ■

Dr. Thau is the owner of a group practice in New York, which has a special emphasis on children's vision and vision therapy. She is a Trustee of the American Optometric Association and a founder of AOA's InfantSEE public health program. She is also an associate clinical professor at the SUNY College of Optometry. Dr. Chung is an associate professor at SUNY, specializing in pediatric optometry. She is president-elect of the College of Optometrists in Vision Development. Dr. Richter is an associate professor at SUNY within the Pediatric, Infant's Vision and Ocular Disease Services.

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Corneal Oxygen Deficiency and CL Wear

By Richard C. Orgain, O.D.

THE CORNEA NEEDS OXYGEN TO STAY HEALTHY. AS contact lens specialists, we are often faced with corneal oxygen deprivation issues that arise from overwear of low oxygen permeability contact lenses.

Fortunately, there is a contact lens option available that offers a significant reduction in signs and symptoms associated with corneal hypoxia: silicone hydrogels (SiHys).

HEMA LENSES & CORNEAL OXYGEN DEFICIENCY

We all encounter patients who complain of lens awareness, sporadic blurring of vision and red, dry-feeling eyes. These signs and symptoms are almost always worse later in the day and while dryness may often be the main symptom, they all may be traced to inadequate oxygen to the cornea.

I most often see corneal oxygen deprivation issues with wearers of HEMA contact lenses. All HEMA lenses have relatively low Dk/t and therefore may create some amount of corneal edema, especially if worn for very long hours or overnight. In a three-year clinical study comparing 30-night continuous wear of SiHy lenses with daily wear of two-week replacement low-Dk/t HEMA lenses, patients wearing SiHy lenses showed significant reductions in limbal and conjunctival redness, corneal neovascularization and also showed less myopic progression.[1] The study also reported significantly fewer symptoms of lens awareness, redness, dryness, photophobia and blurred vision vs. the low-Dk/t HEMA group.[1] The body of knowledge has grown supporting a connection between the decreased levels of available oxygen to the cornea caused by low-Dk/t contact lens wear and negative impacts on the signs of corneal health and patient symptoms.[2]

SIGNS & SYMPTOMS

With all of my contact lens patients, I first look for lens movement, lens edge impingement and any build-up on the lens surface. If I note any injection around the limbal area or stromal edema, I know their cornea's oxygen needs are not being met. Sometimes the signs of corneal oxygen deficiency can be difficult to spot, so I try to listen closely to what patients say about their current lenses, even if the symptoms are vague.

I spend quite a bit of time discussing proper hygiene, wearing schedules and the importance of regular replacement schedules with all (HEMA and SiHy) lens wearers, but most of us have come to accept the fact that we're going to have noncompliant patients. I've learned that scolding and threatening won't do any good. Only common sense discussions and education will change their habits. When I examine patients who are known for their noncompliance, I try to fit them with a higher-Dk product such as AIR OPTIX® AQUA contact lenses and stress that long-term eye health requires their cooperation.

The remedy is a little easier when we're talking about patients wearing HEMA lenses, because switching them to the healthier option of SiHy lenses will surely make a difference to both their health and contact lens wearing experience. I routinely dispense trial SiHy lenses—even to HEMA patients who aren't symptomatic—because I know their overall corneal health will benefit from a more breathable contact lens. I probably have a bias for monthly replacement schedules, as I have fewer overall complaints from these patients. My first choice is AIR OPTIX® AQUA contact lenses. Monthly replacement lenses such as these tend to maintain comfort and have deposit resistance all month long.

TIME FOR AN UPGRADE?

Almost anyone can be a successful contact lens wearer. All it takes is the right lens and proper care.

As far as the right lens goes, fit new patients and upgrade patients wearing older lens options to a higher-Dk contact lens such as AIR OPTIX® AQUA contact lenses. SiHy lenses offer the latest advances in material science and lens design. Not only do they provide higher oxygen transmissibility, but they also offer other features that allow for a better lens-wearing experience.

And as for proper care, if you can communicate the benefits of compliance to patients and the bigger picture of how their actions (or inactions) affect their long-term eye health, there's a better chance of them behaving in a healthy way that leads to a better contact lens experience.

Patience, understanding and real listening will build a lasting relationship that can't be eroded by changes in health insurance, employment or even moves away from your practice.

Dr. Orgain is certified by the Tennessee State Board of Optometry in the treatment of ocular disease and has served as a clinical investigator on different contact lens designs.

1. Bergenske P, Long B, Dillehay S, et al. Long-term clinical results: 3 Years of up to 30-night continuous wear of lotrafilcon A silicone hydrogel and daily wear of low-Dk/t hydrogel lenses. Eye Contact Lens. 2007; 33(2):74-80.
2. Dillehay SM. Does the level of available oxygen impact comfort in contact lens wear?: a review of the literature. Eye Contact Lens. 2007;33(3):148–155.

*AIR OPTIX® AQUA (lotrafilcon B) contact lenses: Dk/t = 138 @ -3.00D. Other factors may impact eye health.

Ergonomics in the Exam Lane

Optometry doesn't have to be a pain in the neck. Taking the right ergonomic approach not only improves your health, but also the health of your practice.

By Colleen Mullarkey, Senior Editor

When you're faced with a full day's caseload, it can be easy to sacrifice comfort in the interest of saving time. You might quickly lean over the slit lamp rather than taking a seat and using it properly, or reach for the ophthalmoscope instead of getting up to grab it. While these may seem like minor shortcuts, they can take a major toll on your body over the course of a workday.

A recent survey of Australian optometrists revealed that 82% of respondents experienced some form of work-related physical discomfort. The neck, shoulders and lower back are the most common problem areas.[1]

"I was amazed at how many optometrists told me that they had experienced discomfort and put up with pain for many years, but no one was able to provide an explanation," says lead author Jennifer Long, B.Optom (Hons), M.Safety Sc., an optometrist and certified professional ergonomist from the School of Optometry and Vision Science at the University of New South Wales in Sydney.

Back-Breaking Work

The explanation involves many components—including the nature of the work itself. With so many instruments and pieces of equipment involved in a comprehensive eye exam, it's no surprise that repetitive stress injuries are common. Research has suggested that clinical tasks such as slit-lamp examinations, ophthalmoscopy and refraction frequently contribute to discomfort.[2]

Or, in other instances, they may exacerbate an existing musculoskeletal issue, which was the case for Rebecca Hutchins, O.D., a behavioral optometrist in Niwot, Colo. She suffers from brachial outlet syndrome, causing tightness in her neck as well as numbness and tingling in her right arm, hand and wrist. After observing Dr. Hutchins perform a typical eye exam, a professional ergonomist told her that

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1 LOWER IOP BY 30%[1]

2 SUSTAIN IT UP TO A FULL DAY[1,2]

INDICATIONS AND USAGE:
TRAVATAN Z® Solution is a prostaglandin analog indicated for the reduction of elevated intraocular pressure in patients with open-angle glaucoma or ocular hypertension.

DOSAGE AND ADMINISTRATION:
One drop in the affected eye(s) once daily in the evening.

IMPORTANT SAFETY INFORMATION:

WARNINGS AND PRECAUTIONS:
Pigmentation: Pigmentation of the iris, periorbital tissue (eyelid) and eyelashes can occur. Iris pigmentation likely to be permanent.

Eyelash Changes: Gradual change to eyelashes including increased length, thickness and number of lashes. Usually reversible.

ADVERSE REACTIONS:
Most common adverse reaction (30% to 50%) is conjunctival hyperemia.

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

Before prescribing TRAVATAN Z® Ophthalmic Solution, please read the brief summary of prescribing information.

References:
1. Lewis RA, Katz GJ, Weiss MJ, et al. Travoprost 0.004% with and without benzalkonium chloride: a comparison of safety and efficacy. *J Glaucoma*. 2007;16(1):98-103. **2.** Gross RL, Peace JH, Smith SE, et al. Duration of IOP reduction with travoprost BAK-free solution. *J Glaucoma*. 2008;17(3):217-222.

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(travoprost ophthalmic solution) 0.004%

TRAVATAN Z®

(travoprost ophthalmic solution) 0.004%

TRAVATAN Z® (travoprost ophthalmic solution) 0.004%
Initial U.S. Approval: 2001

Brief Summary of Prescribing Information

1 INDICATIONS AND USAGE

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

2 DOSAGE AND ADMINISTRATION

The recommended dosage is one drop in the affected eye(s) once daily in the evening. TRAVATAN Z® (travoprost ophthalmic solution) should not be administered more than once daily since it has been shown that more frequent administration of prostaglandin analogs may decrease the intraocular pressure lowering effect.

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

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

3 DOSAGE FORMS AND STRENGTHS

Ophthalmic solution containing travoprost 0.04 mg/mL.

4 CONTRAINDICATIONS

None

5 WARNINGS AND PRECAUTIONS

5.1 Pigmentation

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

Iris color change may not be noticeable for several months to years. Typically, the brown pigmentation around the pupil spreads concentrically towards the periphery of the iris and the entire iris or parts of the iris become more brownish. Neither nevi nor freckles of the iris appear to be affected by treatment. While treatment with TRAVATAN Z® (travoprost ophthalmic solution) 0.004% can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly.

5.2 Eyelash Changes

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

5.3 Intraocular Inflammation

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

5.4 Macular Edema

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

5.5 Angle-closure, Inflammatory or Neovascular Glaucoma

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

5.6 Bacterial Keratitis

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

5.7 Use with Contact Lenses

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

6 ADVERSE REACTIONS

6.1 Clinical Studies Experience

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

The most common adverse reaction observed in controlled clinical studies with TRAVATAN® (travoprost ophthalmic solution) 0.004% and TRAVATAN Z® (travoprost ophthalmic solution) 0.004% was ocular hyperemia which was reported in 30 to 50% of patients. Up to 3% of patients discontinued therapy due to conjunctival hyperemia. Ocular adverse reactions reported at an incidence of 5 to 10% in these clinical studies included decreased visual acuity, eye discomfort, foreign body sensation, pain and pruritus.

Ocular adverse reactions reported at an incidence of 1 to 4% in clinical studies with TRAVATAN® or TRAVATAN Z® included abnormal vision, blepharitis, blurred vision, cataract, conjunctivitis, corneal staining, dry eye, iris discoloration, keratitis, lid margin crusting, ocular inflammation, photophobia, subconjunctival hemorrhage and tearing.

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

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Pregnancy Category C

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

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

There are no adequate and well-controlled studies of TRAVATAN Z® (travoprost ophthalmic solution) 0.004% administration in pregnant women. Because animal reproductive studies are not always predictive of human response, TRAVATAN Z® should be administered during pregnancy only if the potential benefit justifies the potential risk to the fetus.

8.3 Nursing Mothers

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

8.4 Pediatric Use

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

8.5 Geriatric Use

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

8.6 Hepatic and Renal Impairment

Travoprost ophthalmic solution 0.004% has been studied in patients with hepatic impairment and also in patients with renal impairment. No clinically relevant changes in hematology, blood chemistry, or urinalysis laboratory data were observed in these patients.

16 HOW SUPPLIED/STORAGE AND HANDLING

TRAVATAN Z® (travoprost ophthalmic solution) 0.004% is a sterile, isotonic, buffered, preserved, aqueous solution of travoprost (0.04 mg/mL) supplied in Alcon's oval DROP-TAINER® package system.

TRAVATAN Z® is supplied as a 2.5 mL solution in a 4 mL and a 5 mL solution in a 7.5 mL natural polypropylene dispenser bottle with a natural polypropylene dropper tip and a turquoise polypropylene or high density polyethylene overcap. Tamper evidence is provided with a shrink band around the closure and neck area of the package.

2.5 mL fill	NDC 0065-0260-25
5 mL fill	NDC 0065-0260-05

Storage: Store at 2° - 25°C (36° - 77°F).

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

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the clinical tasks required of optometrists were some of the worst repetitive motion behaviors he'd ever seen.

And he's not the only one who thinks so. A recent study in *Ophthalmology* found that eye care providers suffer from more musculoskeletal disorders than family practice physicians.[3] Results showed that eye doctors reported a higher prevalence of neck pain (46% vs. 21%), hand/wrist pain (17% vs. 7%) and lower back pain (26% vs. 9%).[3]

Performing repetitive tasks and continuing to work while injured increases the risk of experiencing severe discomfort.[1] That's why it's important to address pain as soon as possible and take preventive measures to avoid injury in the first place.

Develop a Game Plan

There are many ways to assess ergonomic health in your exam lane—the choice depends on your personal preference, your practice and your budget. When evaluating your practices and techniques, ask yourself:

- Is this comfortable?
- Is this efficient?
- How can we make it better?

"Some people are very aware of their own bodies," Ms. Long says. "If they videotape themselves, it might provide them with enough information to identify how they can improve their practices and techniques."

Or, you may prefer to have a colleague observe you and provide suggestions. However, if your colleague works in the same practice, he or she may be too close to identify any problem areas or offer new solutions. In this case, it may be best to seek professional resources outside the practice. "An outsider might be able to provide a different perspective on an issue or come up with a novel solution based on their experience working in other industries or with other optometry practices," Ms. Long says. "However, they would need to spend some time with the optometrist or in the practice to understand the nature of the job."

To address specific physical injuries, you may want to consult a specialist. Dr. Hutchins, for example, sees a chiropractor, an osteopath and an acupuncturist monthly. For a more comprehensive approach, occupational therapists or ergonomists can perform jobsite analyses to help you make any necessary changes to your workspace and schedule.

Game-Winning Strategies

- ***Use proper body mechanics.*** Try to relax your shoulders. Keep your elbows tight to your waist and your body as close to your core as possible rather than reaching or extending. When you're using loose lenses, try to keep a neutral wrist position instead of pinching the lenses.

"When you have your wrist in a neutral position, it puts less strain on the joint," says Jennifer Kaldenberg, M.S.A., O.T.R./L., director of occupational therapy services at the New England Eye Institute and the New England College of Optometry in Boston. "If you have pain or fatigue in your dominant hand, you can support that arm with a table or your other arm."

"I was amazed at how many optometrists told me that they had experienced discomfort and put up with pain for many years, but no one was able to provide an explanation."
—Jennifer Long, B.Optom (Hons), M.Safety Sc.

Karen Jacobs, Ed.D., an occupational therapist, certified professional ergonomist and clinical professor of occupational therapy at Boston University, tells her clients to fill out a week-long time log and highlight any areas during which they experience musculoskeletal discomfort. "That helps us to better identify what's going on so we can start to make changes to the tasks that seem to be more problematic," she says.

Ergonomists can help to devise strategies to improve comfort and efficiency, even before problems manifest. Physical aspects like posture are only part of the problem, Ms. Long says. A broader solution also addresses cognitive factors, such as task complexity, and organizational components like stress, workload and time pressures.

- ***Switch things up.*** "If your exam rooms are set up with all the equipment on the right or all on the left, then it's much more repetitive motion on only one side," Dr. Hutchins says. She suggests setting up some exam rooms in a right-handed fashion and some in a left-handed fashion to distribute the work more evenly.
- ***Take advantage of adjustability.*** Get as much adjustability from your equipment as you can, as opposed to sitting or standing in an awkward position. "Instead of bringing yourself to the patient, use the chair—raising or lowering it—so you can properly position yourself and maintain a neutral spine position," Ms. Kaldenberg says.
- ***Be mindful of technology use.*** "Holding an iPad or other tablet for an extended period of time may

cause discomfort," Dr. Jacobs says. "They are wonderful tools, but they should not be a complete substitute for inputting reports." You might want to consider using voice recognition applications or software.

When you are using mobile devices or notebook computers, be mindful of your posture. Turn your chair all the way around to the screen, rather than twisting between the notebook computer and the patient, as that can cause neck strain. "A laptop riser allows you to raise the screen so you're not straining your neck or head excessively," Ms. Kaldenberg says. Once you have the laptop raised, it's also important to use an external keyboard and mouse to ensure proper positioning.

• ***Put your best foot forward.*** Long periods of standing can really take a toll on your lower back, which is why supportive footwear is very important. "I know a lot of women wear high heels, and they do not give the proper support," Dr. Hutchins says. "I did not go to athletic shoes, but I wear very, very dorky-looking, lace-up shoes and they really make a difference." Invest in good carpet padding to ensure you're getting support from the surface you're standing on as well.

• ***Take a seat.*** Try to alternate between sitting and standing. Don't sit side-saddle on a stool or a chair, and avoid arching your back. "Try to maintain '90-90-90'—keep your feet flat on the floor, your knees bent at a 90° angle and your hips at 90° angle," Ms. Kaldenberg says. "If you can sit appropriately, you won't fatigue as quickly."

Dr. Hutchins invested in a saddle stool, which has helped to take pressure off of her legs while sitting for long periods of time, and it tips forward so she doesn't have to lean in as much. "It goes up high enough for me to look into the slip lamp, and low enough to write and talk with the patient," she says.

• ***Give yourself a break.*** Move throughout the day and give yourself a positional break every 30 minutes or so. "After I get the distance refraction, I tell the patient they can take a break while I write it down," Dr. Hutchins says. "It might only be for a minute, but I'm giving myself a postural break because I'm no longer leaning forward."

Break up your workday by alternating tasks. Dr. Jacobs suggests scheduling administrative work or a meeting in between clinic to free yourself from using the equipment periodically.

"My staff has been very supportive of 'our' baby," says Christine Sindt, O.D., who is nearing the end of her third trimester. "I don't think I could get through the day without their tremendous effort and help. Having a supportive staff not only helps clinically but also emotionally."

Visit www.revoptom.com to read "What to Expect When You're Expecting" to get ergonomic tips for pregnant optometrists.

Long-Term Goals

Making these adjustments to your practice may take extra time, but it can end up saving you physical pain or an early retirement in the long run. "Our field specifically—but the world, in general—needs to realize that the means and the ends do not add up," Dr. Hutchins says. "If optometrists are working to the point where they're messing up their bodies, they're not going to be able to achieve their professional goals and provide optimal patient care."

Ms. Long believes that shift in thinking starts by educating the next generation of optometrists so they can recognize the signs and symptoms in themselves and adjust their practices and techniques. "Talking about work-related discomfort is important so that optometrists are aware that this issue exists, can seek appropriate help and implement change to improve comfort," she says. ■

1. Long J, Naduvilath TJ, Hao LE, et al. Risk factors for physical discomfort in Australian optometrists. Optom Vis Sci. 2011 Feb;88(2):317-26.
2. Long J, Yip W, Li A, et al. How do Australian optometrists manage work-related physical discomfort. Clin Exp Optom. 2012 Apr 24. doi: 10.1111/j.1444-0938.2012.00711.x. [Epub ahead of print]
3. Kitzmann, AS, Fethke NB, Baratz KH, et al. A survey study of musculoskeletal disorders among eye care physicians compared with family medicine physicians. Ophthalmology. 2012 Feb;119(2):213-20. Epub 2011 Sep 16.

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Important information for AIR OPTIX® AQUA (lotrafilcon B) contact lenses: For daily wear or extended wear up to 6 nights for near/far-sightedness. Risk of serious eye problems (i.e. corneal ulcer) is greater for extended wear. In rare cases, loss of vision may result. Side effects like discomfort, mild burning or stinging may occur.

References: 1. Based on a post-launch evaluation in which 88 eye care practitioners refit over 400 patients in AIR OPTIX® AQUA contact lenses. Alcon data on file, 2011. **2.** Nash W, Gabriel M, Mowrey-Mckee M. A comparison of various silicone hydrogel lenses; lipid and protein deposition as a result of daily wear. *Optom Vis Sci*. 2010;87:E-abstract 105110. **3.** Compared to HEMA contact lenses; based on the ratio of lens oxygen transmissibilities; Alcon data on file, 2010. **4.** Dumbleton K, Richter D, Woods C, et al. Compliance with contact lens replacement in Canada and the United States. *Optom Vis Sci*. 2010;87(2):131-139. **5.** Compared to 2-week replacement lenses; based on self-reported lens replacement time and third-party industry pricing information; Alcon data on file, 2012.

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Should Your Practice Have An Extern?

More importantly, are you willing to share your experience—and in return, gain a sharper appreciation of your skills? If so, the answer is yes. **By Glenn S. Corbin, O.D.**

If you're not currently a clinical preceptor for one of the optometry schools, maybe you should become one. I've served in this capacity for optometry students for the past 30 years, hosting more than 250 externs in my practice during that time, so I have insights to share about the pros and cons of bringing in externs. (But the pros far outnumber the cons.)

Photos: Len Smith/Turning Point Media

Optometrist Glenn S. Corbin (center) discusses clinical findings with externs Aikaterini Koukas and Joseph Ellwood. Dr. Corbin doesn't give his externs all the answers. He asks them their opinions to engage them in patient care.

Do You Have the Right Stuff?

Before you become a preceptor, reflect on who you are and how you perceive your practice. Are you a very independent practitioner who prefers not to share his or her knowledge with others, or your patients for that matter? If so, this role is not for you.

But if you possess a lot of self-confidence in your ability to teach students, in addition to having a solid academic and clinical knowledge base, preceptorship might serve you well (and vice-versa).

Although your training and experience place you in a position to know more than the students, that doesn't necessarily make you a good candidate to teach them. You have to be willing to engage them in all aspects of how you provide patient care, and be open to their opinions and approaches to diagnostic and therapeutic decisions.

Remember, this person will be in your office on a daily basis for three or six months, working closely with you and your staff, so know what you're getting into.

In addition, your patients must be willing to accept that an extern will participate in their care. Only you can make that work. In a teaching hospital or university setting, patients expect to have "student doctors" participating in their care. A teaching practice is no different. Patients usually appreciate the level of expertise that a teaching facility offers, knowing that the doctor will ultimately

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Externs should learn not only about clinical care, but about all aspects of private practice. A stint at the front desk shows externs how a practice "really" works.

review their findings, confirm the testing and see them in person to make the decisions on the direction of their care. In my practice, having externs—I refer to them as "interns" because they're in my office—elevates our practice in the minds of our patients.

What Do Externs Do?

My externs do everything that I do; they practice comprehensive, full-scope optometry. When they first arrive in our office, they receive a one-week orientation to get acquainted with our practice. This gives them time to get to know our staff and the protocols that we follow. Throwing them into patient care immediately would be detrimental to them and to our patients—a formula for disaster.

To start, I give each a thorough tour of the office, and then have them spend several days at my front desk. The reception area is the entry point to our practice, so it's a natural place to start. Also, it's a hotbed of activity, where the extern learns all about the "business" of optometry. As a preceptor, your obligation is to not only expose them to clinical care but to train them on all aspects of private practice.

The externs' time at the front desk gives them an opportunity to hear how the phones are answered, to see how appointments are made and to learn the protocols that my front desk staff uses to triage and address patient issues over the phone. The exposure to the front desk check-in process gives them the opportunity to learn about insurance plans and how we differentiate vision care plans from medical plans. They also observe how patients check out at the end of their visit and learn about preappointing and collection of fees.

Next, the externs shadow my clinical techs and learn the pretesting protocols for the various patient encounters—general exams, contact lens exams, medical eye health visits, emergencies, specialty testing visits, and so on. Once they have some observational opportunities, they switch roles with the techs to perform the pre-testing, while my techs observe and advise them.

After the externs have completed this orientation, they spend some time observing one or more of the doctors performing exams. Practices differ in their culture, so the externs need to understand our approach to patient care and how we communicate with our patients. This is critical to integrating an extern successfully into a private practice. For instance, I use very specific language when explaining test results to patients, which the extern would not pick up without shadowing me. I also review the visual test results with the patient, and prescribe lens designs and applications. This discussion with the patient is critical to yielding a higher rate of eyewear purchases.

No matter how much care my externs provide to my patients, I participate at every level. I introduce my extern to the patient and engage them both in the discussion to initiate the exam.

As I examine patients, I discuss clinical findings with the externs and I ask them their opinions to engage them in the patients' care. Often I ask them to observe through the slit lamp and we discuss the findings in front of the patient, explaining in layman's terms so the patient is part of the discussion.

At this point, the extern should be ready to start participating in patient care directly. After I exit the exam room, the extern performs a comprehensive refraction, visual analysis and slit lamp exam. They then report their findings to me and we discuss a treatment plan with regard to visual correction. The extern can then discuss the final prescription lens and eyewear

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Externs should receive hands-on training of testing protocols for various patient encounters—general exams, contact lens exams, medical eye health visits, emergencies, specialty testing visits, and anything else you can throw at them.

options with the patient.

Next, the tech comes in to perform tonometry and dilate the patient, based on the dilation protocol that I provide. Once the patient is dilated, my extern performs a complete ocular health exam, including slit lamp funduscopy and binocular indirect ophthalmoscopy. Afterward, I repeat the eye health testing and perform a final consultation with my patient, along with my extern. Occasionally, a patient prefers to see only you, "the doctor," not the extern, but that is rare and perfectly acceptable.

In addition to patient care, the externs must adapt to our specific record-keeping forms, protocols, computer software and equipment. Of course, they need to feel comfortable seeing the patients as they learn new administrative protocols. I also have my externs continuously researching diagnoses to help them learn by stimulating discussion. They assist me by drafting my reports to other physicians—a great way for them to review charts, increase their knowledge base and learn what is required to effectively communicate with other doctors, not only eye care practitioners.

Good and Bad

The first few weeks can be trying and frustrating as we break in a new extern. "Patience is a virtue," I remind myself. Once orientation is complete, life is much easier.

Most externs I've encountered over the years have been good or stellar, but occasionally I've had a student who is not up to par. This is an extern whose skills lag significantly behind other externs at the same stage of training. Or, this could be a student whose character is a mismatch for your site—a possible detriment to patient care.

I have found, to my dismay, that my colleagues who had these

Tips from the Externs

What advice do externs have for you about the process of bringing them into your practice? Dr. Corbin's externs offer these suggestions.

- **Expect an inquisition.** "First and foremost, a doctor and his staff should be prepared to ask and answer a lot of questions," says Joseph Ellwood, class of 2013 at Pennsylvania College of Optometry. "No amount of education can make up for what a student lacks in experience. I've learned so much by engaging in dialogue with Dr. Corbin and his staff. Also, working with different doctors gives us the opportunity to see there are often several 'correct' solutions to a presenting problem. I love when doctors ask for my opinion and then give their reasoning for their approach. I am a firm believer in the Socratic method."
- **Discuss cases daily.** "The most important factor for a successful rotation with an extern is communication," says Aikaterini Koukas, also class of 2013 at PCO. "Discussing cases daily is important in the growth of any extern; it opens the opportunity for us to ask questions and further research topics for that day. Even if it's just discussing cases during lunch or at the end of the day, it strengthens our ability to manage patients."
- **Show them the ropes.** "During the first week, it's important for the extern to learn the protocol for patient care," Ms. Koukas says. "The more exposure we receive about how patient care is managed, the faster we are to adapt. Letting an extern observe for one to two days with the doctor helps us understand the flow of patient care."
- **Talk dollars and sense.** "In addition to clinical experiences, Dr. Corbin frequently discusses the business aspects of his practice," Mr. Ellwood says. "Not every doctor who accepts an extern owns a practice, but I'm sure they all have insights to offer on the business of optometry."

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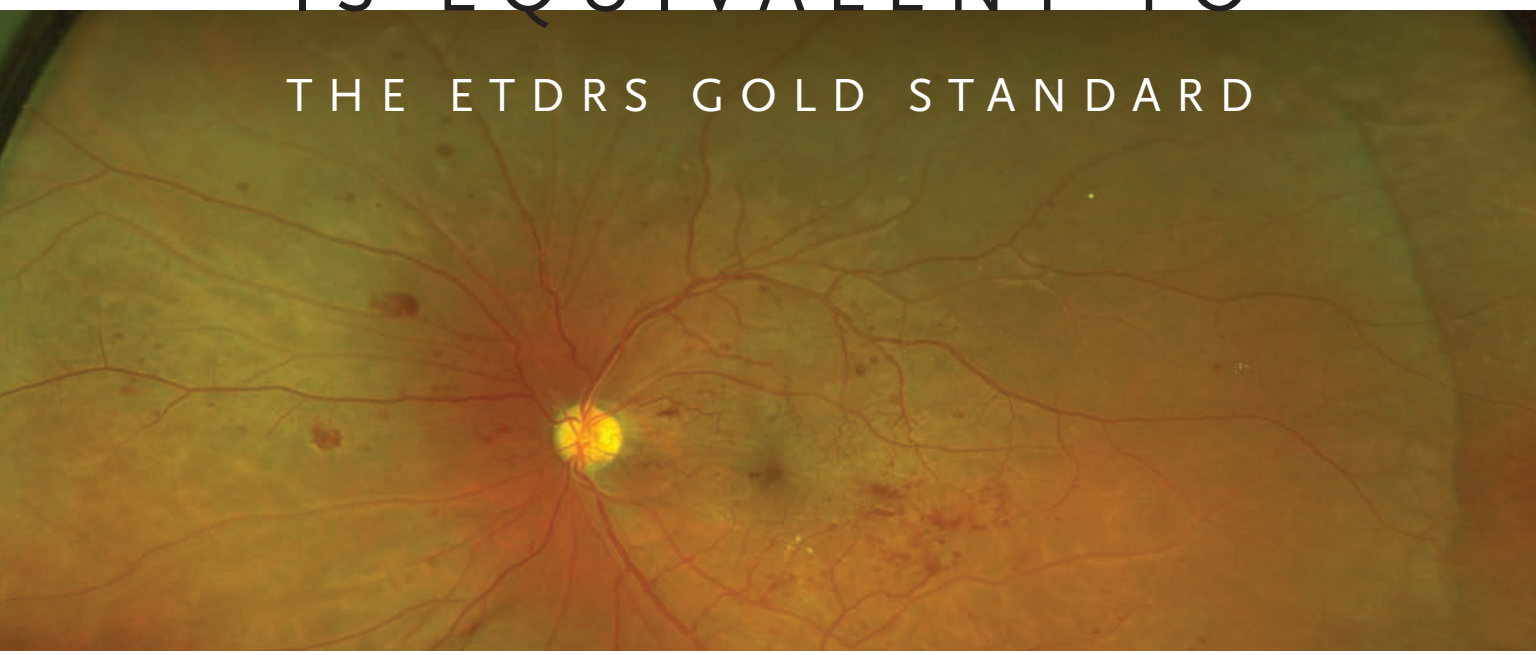

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"Nonmydriatic ultrawide field images compare favorably with dilated ETDRS photography and dilated fundus examination (by a retinal specialist) in determining DR and DME severity: however they are acquired more rapidly.[1]"

American Journal of Ophthalmology, May 2012

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Dr. Corbin has hired five former externs who are now full-time doctors in his practice. Pictured (from left) are: Michael Burkhart, O.D., Amanda Legge, O.D., Dr. Corbin, Kerry Burrell, O.D., Karen Heaney, O.D., and Heidi Sensenig, O.D., M.S.

student externs, prior to them coming to my practice, either paid no real attention to them or were just too lenient and pushed them through. Note that I said "too lenient," not "too nice." This is a true disservice to the student and our profession.

My expectations for all my externs are the same, but I make some adjustments based on the time of their rotation with me—for example, I treat a third-year student slightly differently than a first-quarter fourth-year student. That being said, some students require more intense training than I should have to provide. I first make every effort to give the student the benefit of the doubt and try to provide extra mentoring. But, if the extern has exhausted my patience and my practice's ability to move them to the next level, I then contact the college to send the student back for re-assessment and additional on-campus training. But this is rare.

I'm very proud when I see how so many of my prior externs have gone on to be very successful in academia and/or private practice. It's extremely rewarding to know that I have participated in their success. I have seen many of my former externs throughout the years at conferences, continuing education events and have had the pleasure of attending their lectures as "their" student.

What's in it for You?

If you have a busy office and are practicing up to today's standards both with state-of-the-art technology and clinical scope, becoming a clinical preceptor can bring your practice to the next level. It requires patience, hard work and commitment, but the rewards are plentiful.

I've had the distinct privilege of hiring five of my former externs (one partner and four associates) as full-time members of my practice. I had the ability to spend months working side by side with them, getting to know their personalities and their ability to deal with a variety of situations, and observing their clinical knowledge and skill level. I also share in business discussions with them throughout their rotation and I get to know how knowledgeable they are in this area, as well as their ability to communicate with patients, other health care providers and vendors.

This opportunity allows me to select the best docs to hire, a far better situation than just interviewing and checking references. Bringing on an extern is a great way to have another "doctor" working in your practice to test the waters.

But being a preceptor isn't merely useful as a way to find a new employee. My experience as a preceptor has made me a better practitioner. To be a good educator, I must stay at the top of my game in every aspect of optometric practice. I also learn from my externs. They bring up issues that they learned in school or at other sites, triggering me to further investigate and learn new ideas or techniques. I hate to reinvent the wheel and am anxious to learn how someone else approaches patient care so I can improve my care and efficiency.

What's in it for Your Patients?

When you hear "teaching hos-

Criteria to Be a Preceptor

What does it take to qualify to be a preceptor? Each institution varies in its requirements to participate, but most require you to undergo a complete personal and professional background check. They also require an office profile (instrumentation, number of exam rooms, etc.), patient profile (demographics, number of exams performed, etc.), educational opportunities (student support), IT services and photographs of your office, along with copies of exam forms and office materials.

Once the application has been reviewed, a site visit usually follows. It's not an arduous process, just a thorough one to ensure that the integrity of the program and the students' best interest are respected.

pital," you think "the best." The same goes for private practice. Patient perception of our practice is elevated when they learn that it is a teaching site. Similarly, when I see my patients after my extern has completed their care, the patients feel like they are getting a second opinion and seem to be more engaged in their care.

I query my patients about the care they received from my externs, and they are always forthright in their assessment. As a matter of fact, their comments are usually offered unsolicited. Fortunately, most comments are positive about the clinical care and the extern's chairside manner.

Allay Your Fears

I've often heard practitioners say they elect not to have externs because they're concerned about losing patients, or that their standing with their patients will be downgraded. It is really just the opposite, in my experience. It's up to the doctor to create the environment that sets a teaching practice apart.

If you think that you might be a good candidate to teach externs and make your practice a teaching site, contact the optometry school of your choice to discuss their clinical programs and opportunities. (I'm a clinical preceptor for the Pennsylvania College of Optometry and the Illinois College of Optometry.) I feel that my participation provides much value to me, my staff and my practice as a whole.

You can benefit from the experience as well. If you're looking to help shape our profession, what better way than to train our future colleagues? ■

Dr. Corbin is in private group practice in Wyomissing, Pa.

Under the Specular Microscope

Take a closer look at the clinical and financial benefits that non-contact specular microscopy could offer your practice.

By Abbey Johnson Bonnell, O.D., and Mike Cymbor, O.D.

Many dystrophies can affect the endothelial cell layer of the cornea. Given the limited magnification available on a slit lamp, it is difficult to visualize changes in the endothelium with much precision, especially during the early stages of dystrophic endothelial diseases. Many times, these types of dystrophies go undiagnosed until the damage has progressed.

To that end, non-contact specular microscopy allows optometrists to visualize and diagnose dystrophies earlier and more accurately.[1] Having the ability to perform endothelial cell counts noninvasively allows eye care practitioners to provide quicker, more accurate diagnosis and treatment.

Specular microscopy also offers valuable insight when making decisions that range from contact lens selection to surgical referral. While the hefty price tag on this technology may deter some practices, others will find it a worthwhile investment that pays off not just in better patient care but also in reimbursement dollars.

Average Cell Densities by Age[3]

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

How It Works

At Nittany Eye Associates, we use a CellChek XL clinical specular microscope system (Konan Medical), which allows us to visualize the corneal endothelium at the cellular level in vivo. The endothelium, the most posterior layer of the cornea, is a monolayer of approximately 350,000 to 500,000 highly specialized cells.[2]

As these cells do not have the ability to replicate, humans are born with the maximum number of endothelial cells they will ever have. Histologically, these cells initially have a hexagonal shape. As endothelial cells die, neighboring cells enlarge to cover the empty space once occupied by the cell. This, in turn, causes the remaining cells to lose their hexagonal shape.

The specular microscope projects light onto the cornea, and then captures the image that is reflected from the optical interface between the corneal endothelium and the aqueous humor.

CellChek XL quantitatively analyzes this information and generates four numeric indices: cell density (CD), coefficient of variation (CV), percentage of hexagonal cells (HEX) and number of cells used to calculate the results (NUM). The first three indices are useful for measuring endothelial cell death.

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• ***CD is a measurement of cell density in mm² and decreases with age.*** (*See "Average Cell Densities by Age," page 58.*) A low CD value for a particular age may indicate that the endothelium is depleting faster than normal.

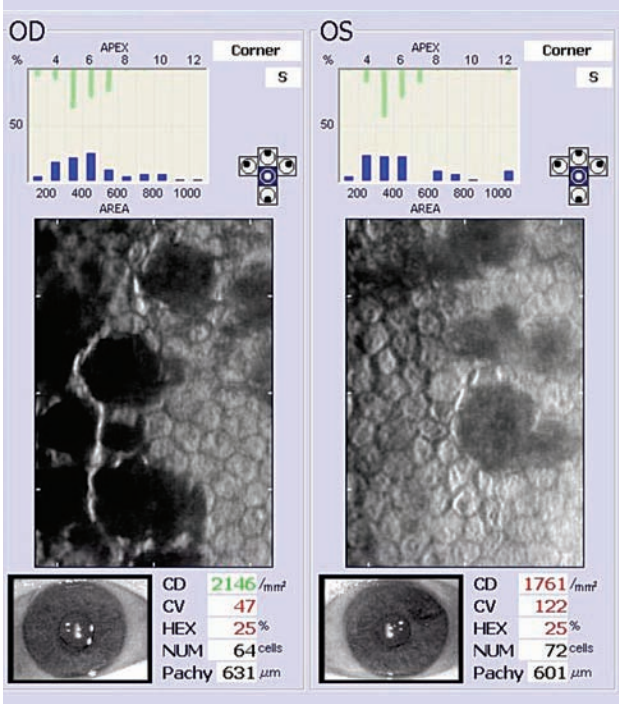
• ***CV represents the coefficient, or degree, of variation in the sizes of the endothelial cells (polymegethism).*** By measuring the variation in size between endothelial cells, the system can measure how much cell loss is occurring. A CV less than 40 is normal.

• ***HEX indicates the variability in hexagonal cell shape over time.*** Hexagonality above 50% is suggested to be normal.[3]

Clinical Benefits in Practice

The ability to properly diagnose and treat corneal endothelial dystrophies allows optometrists to provide a high level of care. Knowing the integrity of the endothelium is another useful tool in a cornea and contact lens practice. In addition, it can explain seemingly idiopathic decreases in vision by revealing subclinical corneal issues.

Taking an endothelial cell count can be helpful before referring patients for cataract or refractive surgery so that any potential endothelial dysfunction or thinning is recognized before they have the surgical intervention. Pre-existing corneal conditions, such as Fuchs' dystrophy, dry eye, epithelial basement membrane dystrophy and Salzmann's nodular degeneration, can decrease the potential for positive surgical outcomes.[4] This information provides valuable insight to both the referring optometrist and the surgeon.



1. Fuchs' endothelial dystrophy—note the significantly increased CV and decreased HEX in the left eye.

At Nittany Eye Associates, the cataract surgeon may lower the phacoemulsification energy in an attempt to preserve endothelial function in patients with a mildly compromised endothelium as determined by specular microscopy. One frequently-touted benefit of the new "femto-phaco" cataract surgery is its potential to be less traumatic to the endothelium by reducing the phaco energy used during the procedure; prescreening with specular microscopy could identify patients better suited to this approach than conventional cataract surgery.

An endothelial cell count also can play an important role in fitting a patient with the right type of contact lens. For example, overnight wear may work successfully in someone with a properly functioning endothelium, but it may cause a variety of issues in a patient with inadequate endothelial function.

With the evolution of lens materials and wearing schedules, there are many options in the optometrist's arsenal. Having this information available before fitting patients in contact lenses can allow the eye care practitioner to make an informed decision when choosing the appropriate contact lens.

In our practice, specular microscopy has served as a helpful aid in diagnosis of endothelial dysfunction and other underlying corneal issues, as illustrated in the following case examples.

Case Examples

Fuchs' Endothelial Dystrophy

A 54-year-old white female presented with a chief complaint of reduced distance and near vision in her left eye. BCVA was 20/20 O.D. and 20/40 O.S.

External testing was unremarkable. Slit lamp examination was remarkable for a filtering bleb and laser peripheral iridotomy in the left eye. There was temporal corneal scarring in the left eye as well as striae in the corneal stroma. General corneal haze was noted in both eyes, but was more significant in the left. Her dilated fundus examination was unremarkable.

Specular microscopy of the left eye shows a significantly increased CV as well as a significantly decreased HEX (*figure 1*). This information reveals cell loss and subsequent morphing of surrounding cells to fill in the compromised endothelium. Her

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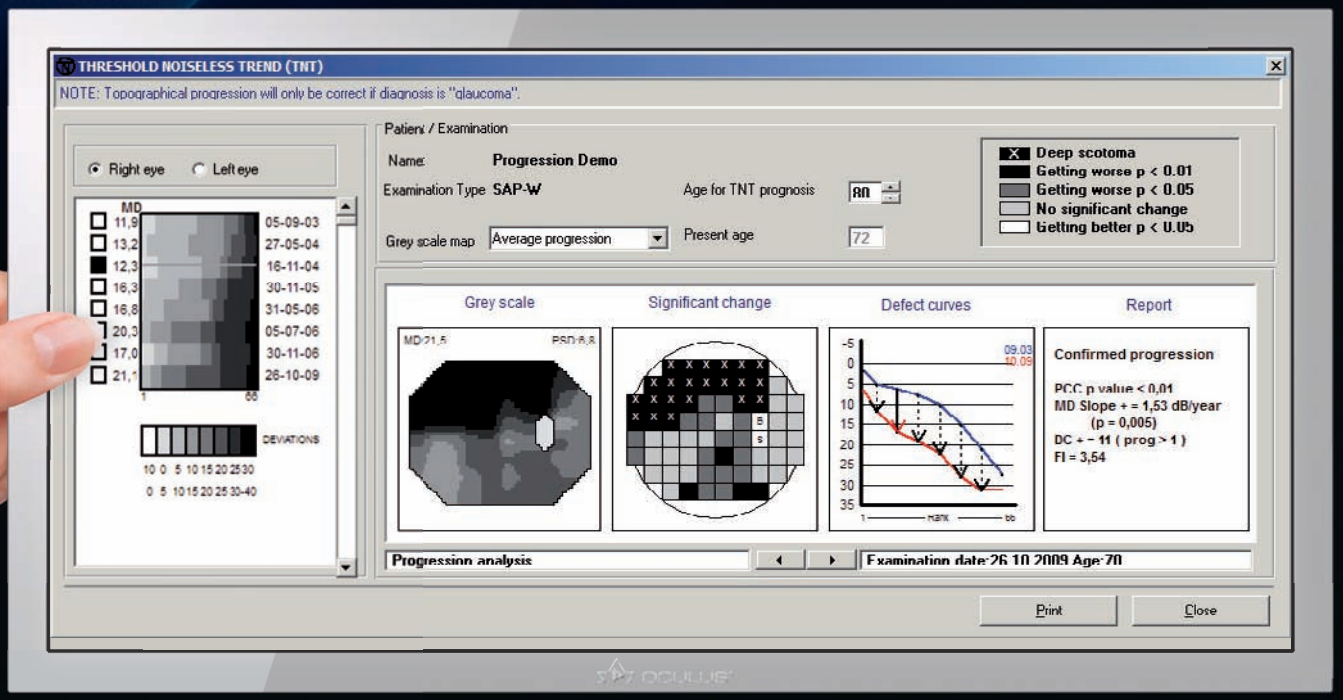

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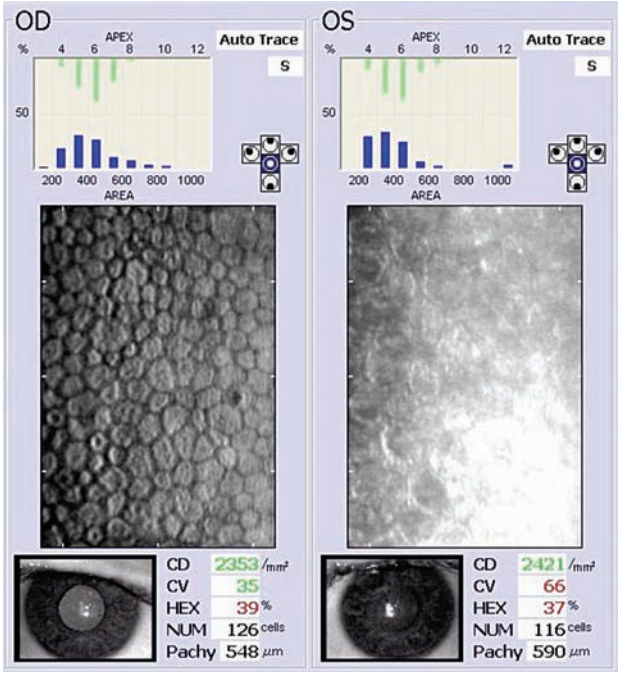
pachymetry shows thickened corneas, measured at 630µm O.D. and 601µm O.S., further confirming the endothelial dysfunction. In addition to grade three guttata, coalescing guttatae are noted in the cell count image. Due to these signs—particularly the guttata and abnormal CV values—we diagnosed the patient with Fuchs' endothelial dystrophy.

Fuchs' is a dysfunction of the cornea's endothelial layer that typically presents between the fifth and sixth decade of life, and affects women more than men.[5] Be sure to question patients about their family history, as Fuchs' is inherited in an autosomal dominant fashion.[6] Treatment of Fuchs' typically involves hyperosmotic agents to pull excess fluid out of the cornea to reduce edema. IOP-lowering medications, such as topical prostaglandins or beta-blockers, are also used in patients with increased IOP.

Limit use of medications preserved with benzalkonium chloride, as chronic exposure may be toxic to endothelial cells.[7] In advanced cases where topical agents cannot combat corneal edema, corneal grafts are inserted. Currently, the surgical treatment of choice is Descemet's membrane stripping automated endothelial keratoplasty (DSAEK).[8]

Chandler's Syndrome

A 54-year-old white male presented with a chief complaint of blur at near in the left eye. His BCVA was 20/20 O.D. and 20/40 O.S. External testing was unremarkable. Slit lamp examination revealed a peripheral iridotomy and a filtering bleb in the patient's left eye.



2. Endothelial cell count indicative of Chandler's syndrome, O.S.

The left cornea also showed corneal scarring temporally and a diffuse haze noted throughout the cornea, including centrally. The dilated fundus examination was unremarkable.

Specular microscopy data from the left eye shows normal cell counts, but abnormal CV and HEX indicates pleomorphism is occurring (*figure 2*). The left endothelial cell layer clearly shows blurred cell margins. This patient has a common presentation of Chandler's syndrome, a disorder characterized by abnormal proliferation of the cornea's endothelial layer.

The excess endothelial cells adhere to the iris and lodge themselves in the trabecular meshwork. This results in distortion of the iris, increased IOP and corneal edema. Chandler's syndrome frequently presents in young adulthood or middle age and often is found unilaterally with the uninvolved eye manifesting only subclinical signs.[9]

This holds true in the patient's scan. The right eye (the uninvolved eye) clearly shows a more uniform and functioning endothelium. In addition, the pachymetry readings show the left cornea is thicker than the right eye, indicating greater edema O.S. Specular microscopy allowed us to confirm this diagnosis, and is a valuable tool in identifying the clinical signs of Chandler's syndrome.

Treatment of Chandler's syndrome often centers on controlling the corneal edema and managing increased IOP. Corneal edema is treated with hyperosmotic solutions and ointments in the early stages. In advanced cases, a corneal transplant is warranted. Increased IOP is managed with glaucoma medications.

Agents that decrease aqueous production, such as beta-blockers, yield better results than those that work by increasing aqueous outflow. This is because the endothelial pumps are not working at sufficient capacity to successfully manage the existing aqueous.[8]

Contact Lens Intolerance

A 31-year-old white male had a history of contact lens intolerance. He reported decreasing vision throughout the day. His BCVA was 20/20 O.D. and 20/20 O.S. His contact lens history was lengthy and included many different materials as well as various replacement schedules—all with no relief of symptoms.

His endothelial cell count in a 26 Dk daily lens was normal, with a decreased CV and HEX in the right

eye (*figure 3, page 64*). While CD is normal, the decreased CV and HEX indicate cell loss.

The left eye demonstrates decreased CD, CV and HEX, which suggests a dysfunctional endothelial cell layer and explains the patient's contact lens intolerance and decrease in vision throughout the day. The patient was instructed to discontinue contact lens wear for two to three weeks. After this time, the patient was to resume contact lens wear with a 55 Dk daily lens to increase oxygen transmission to the ocular surface.

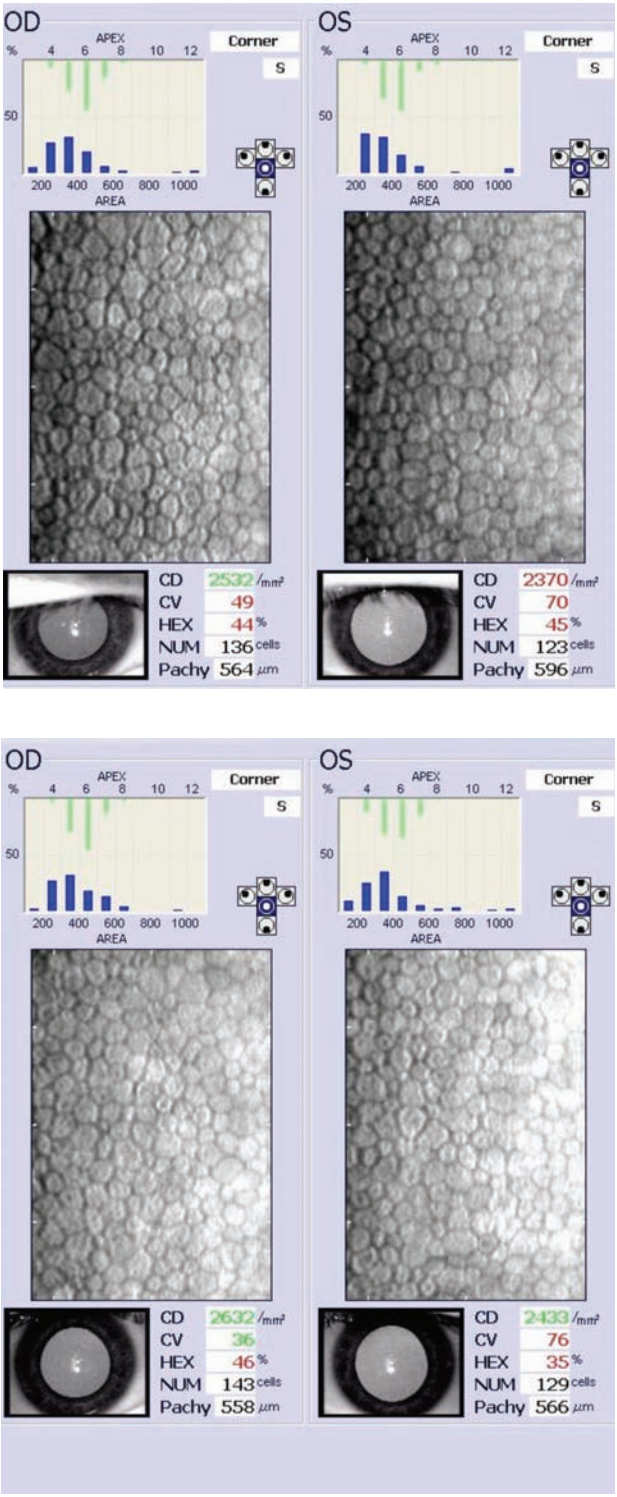
Reimbursable Diagnosis Codes for Specular Microscopy[11]

Condition	Diagnosis Code
Endothelial dystrophy	371.57
Corneal edema	371.20-371.24
Posterior polymorphous dystrophy	371.58
Iridocorneal endothelium syndrome	364.51, 371.57
Extended wear contacts after intraocular surgery	379.31, V43.1, V45.69
History of previous intraocular surgery and require cataract surgery	V45.69
Aphakic and undergoing secondary IOL implantation	379.31

The endothelial cell count was repeated after approximately three months (*figure 4, page 64*). The right eye showed an improved CV, and there was also an increase in CD of the left eye. While endothelial cells do not regenerate throughout a person's lifetime, the increase in CD may be a reflection of a healthier endothelium and a more reliable reading.

The pachymetry readings were also decreased compared to the previous cell count, which indicates resolution of prior corneal edema. This cell count suggested to us that increasing the oxygen permeability of the patient's contact lenses would allow the endothelium to function at a higher level, thus permitting longer wear time and improved comfort.

With advances in high-Dk lens materials and FDA approval of overnight contact lens wear, assessing corneal integrity is more important than ever. Being able to analyze important endothelial characteristics provides eye care practitioners with another tool to evaluate corneal health. It also allows practitioners to advise against overnight wear or lower Dk lenses in those



3, 4. Endothelial cell count of patient with contact lens intolerance in a 26 Dk daily lens (top). Endothelial cell count of same patient with contact lens intolerance in a 55 Dk daily lens.

patients who have dysfunctional endothelial cells.

The ability to assess the endothelial cell layer has allowed clinicians in our practice to look at additional facets of the patient's corneal health that can help to explain why a seemingly healthy person is unable to comfortably and successfully wear contact lenses.

In our practice, using specular microscopy has helped us to better predict which type of lens (i.e., high Dk, daily replacement) will work for the patient and reduce frustration.

Does It Pay Off?

Although the initial investment in a specular microscope may cost your practice anywhere from $25,000 to $30,000 per unit, manufacturers offer monthly payment plans and leasing options to make the cost more feasible. Reimbursement also can help you get a reasonable return on your investment.

Insurance companies will reimburse for specular microscopy when medically necessary, which can provide a significant addition to practice revenue. The current CPT code for specular microscopy is 92286 (special anterior segment photography).

As it is a bilateral code, the reimbursement is the same whether specular microscopy is performed on one or both eyes.[11] Medicare currently reimburses the procedure for a wide variety of diagnoses. (*See "Reimbursable Diagnosis Codes for Specular Microscopy," page 63.*)

Endothelial cell photography is a covered procedure under Medicare when reasonable and necessary for patients who meet one or more of the following medical criteria:[12]

- slit lamp evidence of endothelial dystrophy (corneal guttata).
- slit lamp evidence of corneal edema (unilateral or bilateral).
- about to undergo a secondary intraocular lens implantation.
- had previous intraocular surgery and require cataract surgery.
- about to undergo a surgical procedure associated with a higher risk to the corneal endothelium (i.e., phacoemulsification or refractive surgery, with evidence of posterior polymorphous dystrophy of the cornea or irido-corneal-endothelium syndrome).
- about to be fitted with extended wear contact lenses after intraocular surgery.

It is important to know Medicare's coverage policy for specular microscopy before cataract surgery.[11] Simply stated, if performing cataract surgery in an

individual for the first time whose only visual issue is cataracts, specular microscopy is covered as part of the presurgical work-up. The patient's record should contain: an order for the test, an endothelial image, test reliability, findings, diagnosis and doctor's signature.[11]

Medicare currently reimburses about $85, while other third-party payers reimburse $120.[12] These rates are national averages and vary throughout the country. The break-even point is between 250 and 300 scans.

In one year, we performed more than 100 scans at Nittany Eye Associates. We expect to attain our break-even point in about three years. In the time that we've owned our specular microscope, we have had no issues obtaining reimbursement through Medicare or local insurance companies.

Not all optometry practices can justify purchasing an endothelial microscope. But for those practices that see a considerable amount of corneal pathology, cataracts and/or contact lens patients, this technology affords an opportunity to provide a higher level of care. ■

Dr. Bonnell interned at Nittany Eye Associates, State College, Pa., and is now a staff optometrist at The Eye Center of Central Pa., an M.D./O.D. practice with 12 locations throughout central Pennsylvania. Dr. Cymbor is a partner and director of externship programs at Nittany Eye Associates.

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Monthly Multifocal Pearl

Setting Expectations for Multifocal Patients

By Steven J. Lowinger, OD

Our profession requires us to ensure that patients are happy and comfortable with their vision as they age, and multifocal contact lenses are a major resource in our arsenal to help in this quest. There are many different multifocal lenses available and just as many approaches to fitting these lenses, but the best practices seem to share the same mindset on screening, fitting and preparing their patients for multifocal wear. What's more, the evolving technology of these lenses, including the AIR OPTIX® AQUA Multifocal contact lens with Precision Profile Design, has made fitting these lenses and addressing patient expectations easier. The main goal is to be aware of the options *before* screening and selecting patients for multifocal wear.

FIRST THINGS FIRST

Screening patients for multifocal wear does not mean prejudging them. It is important to be open to fitting all patients, regardless of stereotypes/expectations so as to avoid a missed opportunity. A more sophisticated and targeted approach to gauging a patient's visual and occupational needs is required to align the strengths of the multifocal contact lens with the patient's need. A –5.00 patient who is a jeweler and takes his glasses off when working is an extreme example of a bad patient for multifocals, but if that same jeweler is looking for a modality to use when away from the office to see a menu, then you have an opportunity to make that patient happy with a multifocal contact lens. Once the patient is in your chair and you both have decided that multifocals are the best option, it is important to set expectations.

GET PATIENT EXPECTATIONS IN CHECK

The most important thing in setting expectations is to keep it simple. Mile Brujic, OD, and Jason Miller, OD, suggest that when, for example, you recommend multifocal contact lenses for your patients, describe it as a process where you will be personalizing the fit.[1] Using information about the patient that you have gathered in your history allows you to frame their expectations so that they will start out on the right foot toward successful wear. Follow that up by informing the patient of expected visual outcomes and reassuring them that adjustments can be made during the usual and normal follow-up visits. Give patients tangible goals that they can expect without over-promising. Failure to set patient expectations appropriately can result in unhappy patients, increased chair time and lost revenue.

Patients must understand that this visual system, like all visual systems (e.g., spectacles), is not 100% perfect over all ranges, but they should expect to be somewhere in the 90% for most ranges. We also inform patients that just with any other visual device, these lenses will have an adjustment period and may need to be modified at follow-up appointments. Moreover, we simplify what we want the patient to concern themselves with in assessing the lenses: near, intermediate, and distance vision, and comfort.

Prior to fitting the patient with a multifocal, we also review the other presbyopic options available (i.e., reading glasses over contact lenses and monovision.) Each of these modalities has its benefits and must be considered in discussions with your patients. Wearing glasses over contact lenses can be beneficial for a patient with multiple visual needs, but most contact lens patients seek freedom from eyeglasses, not a different pair. Monovision was the gold standard for presbyopic lens fitting for a long time, but studies show that when patients are presented with a monovision vs multifocal option, they tend to prefer multifocal over monovision at a rate of about 3:1.[2,3]

In our office, we use the AIR OPTIX® AQUA Multifocal contact lens as the first lens of choice because of the near, intermediate, and distance acuity it provides. Maintaining good distance visual acuity when presenting patients with a lens that aids in reading is a huge step toward acceptance of this visual system. One of the many benefits of the unique Precision Profile Design of AIR OPTIX® AQUA Multifocal contact lenses is the adaptive minus power profile. This allows us to "push plus" at distance for improved near vision, without impairing distance vision. The lens also allows for smooth transitions between near, intermediate, and distance, providing presbyopes with a more natural visual progression. In fact, the center-near design of the lens works with the eyes' natural binocular function. For these reasons, and more, AIR OPTIX® AQUA Multifocal contact lenses are a great choice for your presbyopic patients and for your practice.

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Informing and educating patients about recent technological improvements in multifocals and the available options creates value in our practices. That value is further enhanced as we solidify our fitting expertise by setting the right expectations and explaining them to patients as well as helping them attain a successful multifocal lens system for their visual needs. Practitioners who embrace this model will be seen as an expert and will enjoy a larger patient base and practice.

Dr. Lowinger is in private and retail practice in South Florida. He is a speaker on multiple topics and is the former chair of the leased tenant committee for the Florida Optometric Association.

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PEER • REVIEWED • RRA • ARTICLE

The Many Faces of Ocular Toxoplasmosis

Ocular toxoplasmosis frequently exhibits variable clinical presentations, which can present an added diagnostic challenge. **By Sherrol A. Reynolds, O.D., Laura A. Falco, O.D., Diana L. Shechtman, O.D., and Joseph J. Pizzimenti, O.D.**

Ocular toxoplasmosis occurs as a consequence of *Toxoplasma gondii* infection. *T. gondii*, an obligate intracellular parasite, is estimated to infect at least one billion people worldwide.

At least 25% of individuals who have *T. Gondii* present with associated ocular manifestations.[1] Ocular inflammation secondary to *T. gondii* infection is the most frequent cause of posterior uveitis.

Classic findings include a white fundus lesion with overlying, intense vitreous cells that frequently is described as "headlights in a fog." The presentation of ocular toxoplasmosis can include a wide range of clinical signs, which poses a diagnostic challenge.

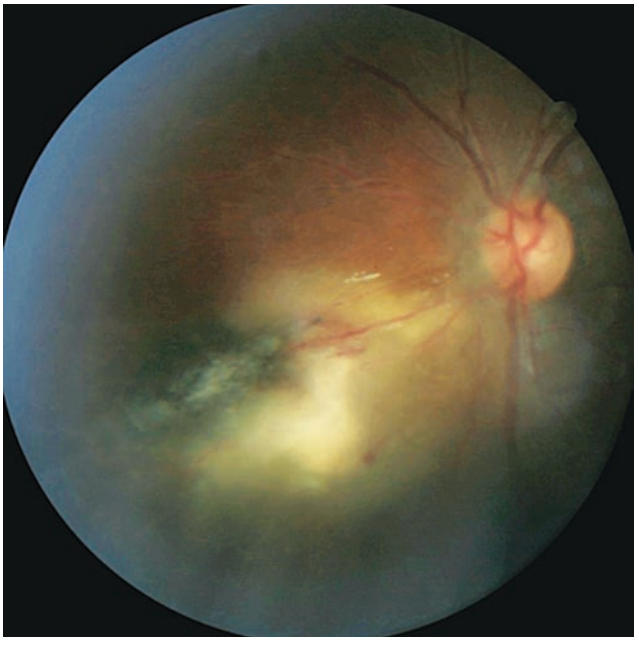
Here, we present three serologically confirmed cases of ocular toxoplasmosis and discuss the variable—and sometimes complex—clinical picture, including diagnostic and management strategies.

Patient 1

A 41-year-old black male presented with a chief complaint of floaters in his left eye that had persisted for three weeks. His medical history was remarkable for type 2 diabetes, which was controlled with oral medications.

His best-corrected visual acuity was 20/20 O.U. Intraocular pressure measured 12mm Hg O.D. and 15mm Hg O.S.

We identified several cells in the left vitreous. A dilated fundus examination revealed healthy optic nerves. Additionally, his right retina was unremarkable. In the left eye, however, we noted the presence of a retinitis that was located adjacent to a large chorioretinal scar (*figure 1*). We also detected a dilated retinal vein with associated superficial hemorrhages O.S.

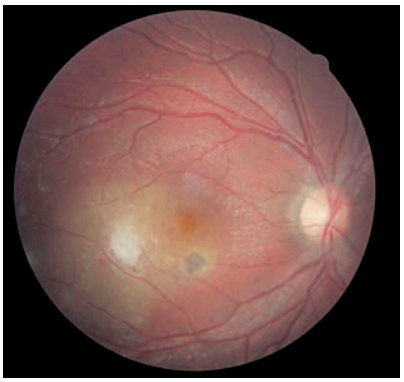


1. Fundus photograph of Patent 1's left eye. Note the presence of a retinitis located adjacent to a large chorioretinal scar.

Patient 2

An 18-year-old white male presented with decreased vision in his right eye. His medical history was unremarkable, and his ocular history revealed possible evidence of an old infection O.D.

Best-corrected visual acuity was 20/50 O.D. and 20/20 O.S. His



2. Fundus photograph of Patient 2's right eye. What do you notice?

intraocular pressure measured 21mm Hg O.D. and 15mm Hg O.S. We documented panuveitis in his right eye.

Both optic nerve heads appeared healthy. However, there was an active retinochoroiditis with associated vitreous cells located temporal to the right fovea. Chorioretinal anastomosis was noted below the lesion. In addition, we identified a small chorioretinal scar that was associated with a neurosensory detachment located inferior to the right fovea (*figure 2*).

Patient 3

A 23-year-old white female presented with a painful left eye. She informed us that the pain began four days earlier, and was accompanied by photophobia. Her ocular and medical histories were unremarkable.

The anterior segment examination revealed several pigmented keratic precipitates, as well as cells and flare O.S. Intraocular pressure measured 18mm Hg O.D. and 24mm Hg O.S.

A dilated fundus examination of the right eye did not reveal any anomalous findings. However, her left eye exhibited a moderate amount of vitreous cells. On optical coherence tomography (OCT), we were able to further evaluate the vitreous cells as well as uncover the presence of an accompanying posterior vitreous detachment O.S. (*figure 3*). Additionally, we documented an inferonasal midperipheral hemorrhagic vasculitis with an associated chorioretinal scar in her left eye (*figure 4*).

Discussion

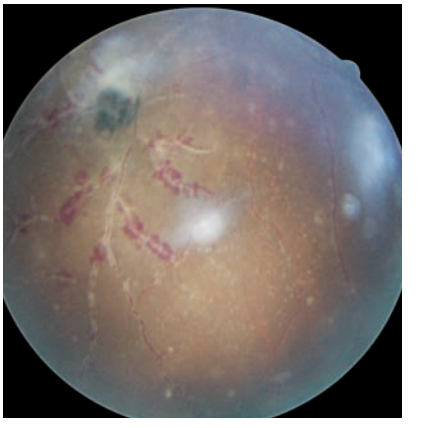
• ***Overview of* T. Gondii**. The life cycle of *T. gondii* has multiple stages. Once the parasite is ingested by a cat (the primary host), it multiplies and is released into the feline feces. Once the intermediate host is exposed to the infected feces, the *T. gondii* oocysts rupture to release tachyzoites, which ultimately travel to target tissues and become bradyzoites.[2]

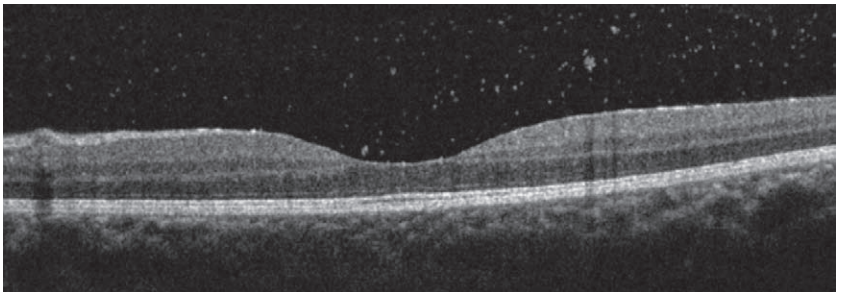
T. gondii infection can be acquired or congenital. Humans may acquire it by direct contact with infected feces (e.g., ingesting contaminated raw/undercooked meat and vegetables, consuming unpasteurized milk products or drinking contaminated water).

The risk of infection is significantly greater if the person has a compromised immune system (e.g., secondary to AIDS or chronic granulomatous disease), is of advanced age or is undergoing immunosuppressive therapy. Once infected, such high-risk individuals often experience a subsequent reactivation of a *T. gondii* infection. Also, it is important to note that there are rare case reports of patients becoming infected via blood transfusion or organ transplantation.[3]

In the congenital form, *T. gondii* is directly transferred through the placenta. Depending on when the fetus becomes infected, the disease may manifest with mild to severe signs and symptoms. Infection early in pregnancy typically leads to a more severe clinical presentation.

• ***Ocular Toxoplasmosis***. Individuals with ocular toxoplasmosis may present with myriad signs and symptoms. These include decreased vision, floaters, pain or ocular redness.[1-3] For example, Patient 1 complained of new floaters, while Patient 2 presented with a concern





3, 4. On optical coherence tomography (below), Patient 3's left eye exhibited a vitreous detachment. The fundus exam of his left eye (right) revealed the presence of a hemorrhagic retinal vasculitis as well as a chorioretinal scar.

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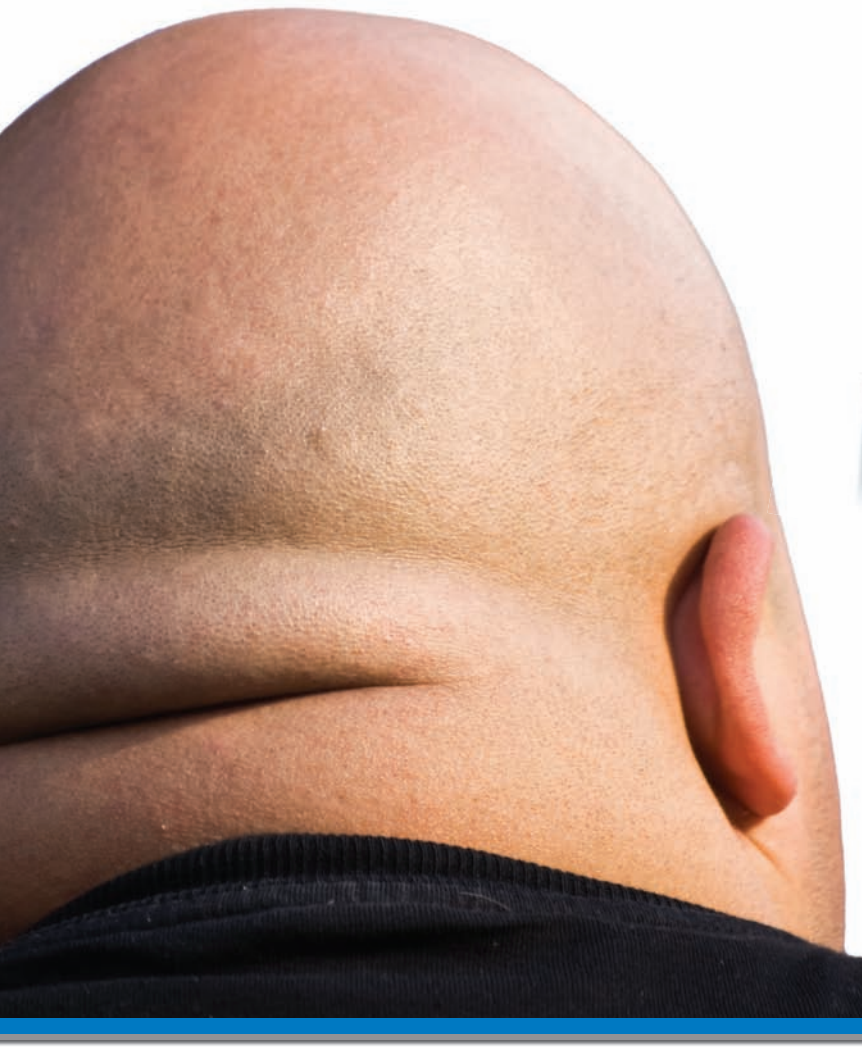

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about decreased vision. On the other hand, Patient 3 presented with a painful, red eye.

The hallmark clinical finding of ocular toxoplasmosis is a retinochoroiditis. Characteristically, it appears as a fluffy, white or yellowish fundus lesion with overlying vitreous cells (Patient 2). The lesion initiates within the inner layers of the retina, while the choroid and sclera may become sites of contiguous inflammation and subsequent necrosis.[4] The inflammation may lead to a posterior vitreous detachment (Patient 3) and/or the formation of vitreous "snowball" precipitates.

In addition, patients may have a granulomatous or non-granulomatous anterior uveitis (Patients 2 and 3). Severe inflammation may lead to posterior synechiae. Further, it is common to note increased intraocular pressure in patients who exhibit an accompanying anterior uveitis (Patients 2 and 3).

Inactive ocular toxoplasmosis fundus lesions appear as "punched-out" chorioretinal scarring. Upon reactivation of the initial infection, satellite areas of newly affected retinal tissue typically present adjacent to existing lesions.

Other posterior segment findings have been associated with ocular toxoplasmosis. Retinal vascular changes may include periphlebitis/arteritis (Patient 3), perivascular sheathing, branch retinal artery or vein occlusion (Patient 1), and choroidal anastomosis (Patient 2).[5,6]

Punctate outer retinal toxoplasmosis—characterized by multifocal punctate outer retinal lesions—is a less commonly reported finding that may be associated with a low-grade vitritis.[7] Its appearance is similar to that of the white dot syndromes and histoplasmosis. (However, histoplasmosis does not present with an associated vitritis.[7])

Complications from ocular toxoplasmosis may pose further diagnostic challenges as well as threaten the eye's visual function. One such challenge is neovascularization secondary to retinal ischemia. Severe retinal vasculitis and/or inflammation, for example, may predispose a patient to neovascularization.[8] The inflammatory response may extend to the optic nerve head, manifesting as an optic neuritis or papillitis.

Also, patients with ocular toxoplasmosis are at increased risk for retinochoroiditis juxtapapillaris (Jensen disease). Jensen disease has been described as an active retinal lesion with concomittant optic nerve involvement.[9] Neuroretinitis in toxoplasmosis is associated with optic nerve head edema and macular exudate that presents in a stellate pattern (similar to the presentation exhibited by patients with cat scratch disease).

• ***Diagnosis and Treatment.*** A timely, accurate diagnosis of ocular toxoplasmosis is imperative to ensure that proper treatment is initiated immediately. In addition to a thorough clinical examinatinon, other diagnostic tests include OCT, ultrasonography, fluorescein angiography (FA) and indocyanine green angiography (ICG).

OCT is useful in determining the level of activity with respect to tissue depth, morphological changes and assocaited retinal edema, as well as aids in the evaluation of the vitreous and vitreoretinal layer. Also, OCT has shown that inflammation caused by *T. gondii* primarily involves the inner retina.

Other findings on OCT include epiretinal membrane, cystoid macular edema, vitreomacular traction, serous macular detachment and secondary choroidal neovascularization.[10]

FA can assist in the diagnosis of active toxoplasmosis lesions by depicting leakage and/or associated hypoperfusion. Further, ICG imaging may be used specifically to monitor for choroidal vascular activity.

The primary serologic test used to confirm the diagnosis is the Sabin-Feldman dye test. Additional laboratory evaluations include indirect fluorescent antibody testing as well as the enzyme-linked immunosorbent assay, IgG and IgM titers.

The clinical presentation of ocular toxoplasmosis may be similar to that associated with several other infectious and inflammatory conditions, including tuberculosis (TB), syphilis and sarcoidosis. In Patient 3, our differential diagnoses included TB, sarcoidosis and cytomegalovirus retinitis. Thus, a systemic evaluation (including serology) is necessary prior to the initiation of medical treatment. This is particularly imperative in cases with atypical presentations, because treatment with steroids may have a profound adverse effect.

Location, size and severity of the retinochoroiditis, as well as the systemic health of the patient, contribute to the overall management plan. In normal patients, peripheral lesions that present with minimal signs and symptoms simply may be monitored. However, if such signs and symptoms are evident in individuals with compromised immune systems, prompt treatment is warranted.

Further, lesions that are located in the posterior pole (Patients 1 and 2) are considered vision threatening. Thus, treatment is indicated for these lesions. Lesions

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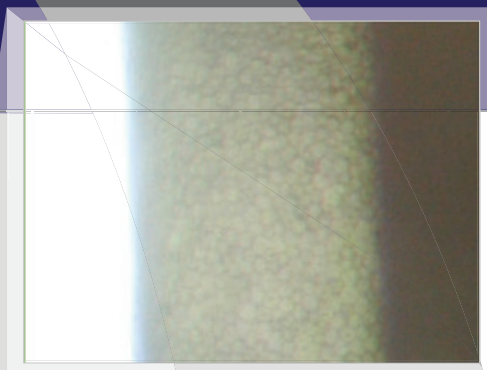

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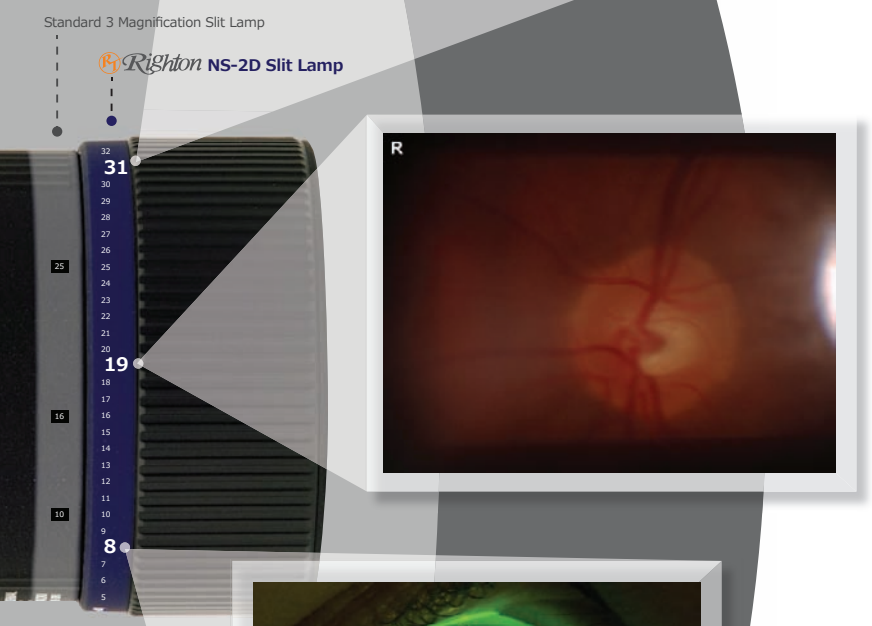

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that affect the macula or optic nerve, or those associated with two lines or more of vision loss, should receive treatment. Further, lesions that are associated with severe vitritis (Patient 3) also should be treated.

Conventional treatment is described as "triple" or "quadruple" therapy. The classic triple therapy consists of pyrimethamine, sulfadiazine and prednisone; quadruple therapy adds clindamycin as well. In patients with sulfa allergy, clindamycin is often substituted.

The typical treatment regimen persists for four to six weeks, with a high loading dosage during the first few days. Steroids are added two to three days after initiating the antibiotic therapy, and then are tapered during the next several weeks.

It is important to note that pyrimethamine use has been associated with adverse reactions and side effects, including thrombocytopenia and leukopenia.[11] Thus, alternative antibiotics, such as azithromycin may be substituted. In most instances, however, folic acid supplementation simply is added to the drug regimen to avoid hematologic complications.

Contemporary treatment includes the use of Bactrim (sulfamethoxazole and trimethoprim, Mutual Pharmaceutical Company, Inc.), with adjunctive prednisone for a four- to six-week duration. This treatment option appears to be a safe and effective substitute for the conventional triple or quadruple therapeutic approaches.

A study showed that toxoplasmosis patients who use Bactrim are also 6.6% to 23.8% less likely to experience recurrent retinochoroiditis.[11]

Additional Discussion

During the last five years, researchers have identified several additional risk factors for *T. gondii* infection. Although it was once believed that most presentations of ocular toxoplasmosis were congenital in nature, the acquired form may be more common than previously considered. Higher dietary intake levels of locally cured, dried or smoked meat and unpasteurized goat's milk, yogurt and cheese has been linked to increased parasite exposure.[12] Eating raw oysters, clams or mussels also may increase an individual's chances for ocular toxoplasmosis.[12]

The more time that passes without a reactivation following initial infection, the lower the patient's overall probability of experiencing disease resurgence. However, epidemiologic data show that patients of advanced age have an increased risk of disease reactivation.[13]

Various studies regarding the epidemiology, prevalence and treatment of ocular toxoplasmosis are ongoing. For instance, one clinical trial is evaluating the treatment effect of intravitreal clindamycin plus dexamethasone.[14] Preliminary data suggest that this drug combination may be a safer, more convenient treatment option than conventional triple or quadruple therapies.[14]

All three of our patients were managed successfully with either classic triple/quadruple therapy or the use of Bactrim and steroids for four weeks. Both treatment approaches yielded satisfactory disease resolution. More importantly, however, these cases illustrate the wide spectrum of ocular toxoplasmosis.

Atypical presentations can pose diagnostic dilemmas. Proper diagnosis can be established by carefully evaluating patient history, demographics and the overall clinical picture. Disease confirmation can be obtained serologically. It is critical to make a prompt, accurate diagnosis to ensure timely treatment and minimize or prevent secondary visual complications. ■

Dr. Reynolds is an associate professor at Nova Southeastern University School of Optometry in Ft. Lauderdale, Fla. Dr. Falco is an assistant professor at Nova Southeastern. Dr. Shechtman is an associate professor at Nova Southeastern and co-author of our "Research Review" column. Dr. Pizzimenti is an associate professor at Nova Southeastern and co-author of our "Review of Systems" column.

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14th Annual Diabetes Report

New Report: Diabetic Retinopathy Rates Spike

Although diabetic eye disease has increased by 89% in a decade, O.D.s counter that the risk of vision loss as a result has greatly diminished. **By Jane Cole, Contributing Editor**

Whether you practice in Portland, Maine, Portland, Ore., or some location in between, a new report indicates you'll have more patients sitting in your chair with diabetic retinopathy than ever before.

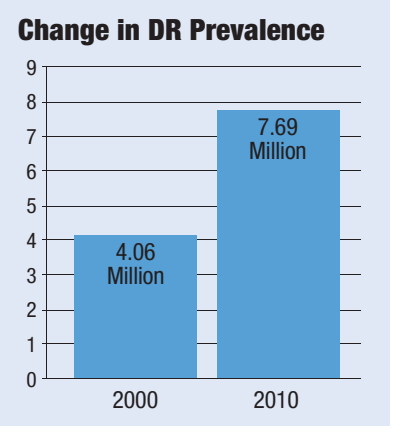
The report, *Vision Problems in the U.S.*, by Prevent Blindness America and the National Eye Institute (www.visionproblemsus.org), cites an 89% spike in diabetic eye disease from 2000 to 2010, based on U.S. Census data and 12 epidemiological studies. The report, prepared by researchers at John Hopkins University, indicates that 7.69 million people in the U.S. age 40 and older suffer from diabetic retinopathy, up from 4.06 million in 2000. Additionally, those in the 40-plus age bracket with vision impairment and blindness from any etiology has increased by 23% since 2000.

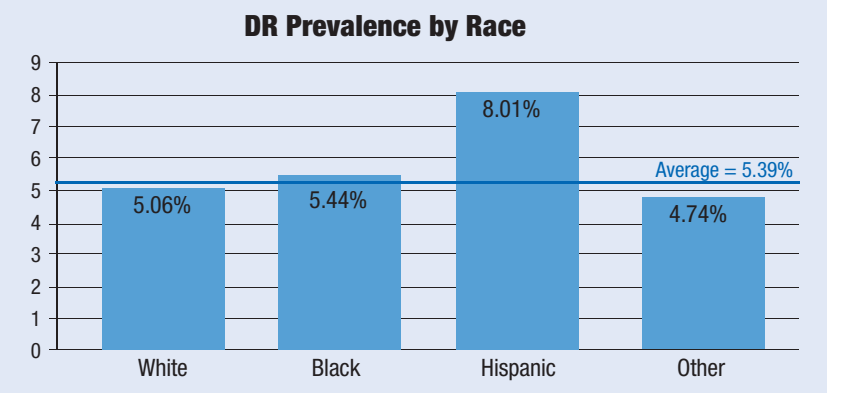
"Rates of diabetes are growing alarmingly, so it's not surprising that cases of diabetic retinopathy have gone up," says Washington State optometrist and diabetes specialist Paul Chous, O.D., who has had type 1 diabetes for 44 years.

Troubling Statistics

The findings may not be a surprise, but the escalating diabetic retinopathy rates are still troubling. "We are so fortunate to have access to information like this from Prevent Blindness America and NEI; however, I am disheartened by the data," says optometrist and certified diabetes educator Tina MacDonald, O.D., of Los Angeles. "Diabetic retinopathy has increased by more than 89%, while those in the United States population 40 and older grew by 19.5% between 2000 and 2010—it's really a shocking number."

Prevent Blindness America recently unveiled the *Vision Problems in the U.S.* report during the "Focus on Eye Health" summit in Washington, D.C., where the Centers for Disease Control and Prevention's Division of Diabetic





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MOXEZA® Solution is a topical fluoroquinolone anti-infective indicated for the treatment of bacterial conjunctivitis caused by susceptible strains of the following organisms: *Aerococcus viridans*, Corynebacterium macginleyi*, Enterococcus faecalis*, Micrococcus luteus*, Staphylococcus arlettae*, S. aureus, S. capitis, S. epidermidis, S. haemolyticus, S. hominis, S. saprophyticus*, S. warneri*, Streptococcus mitis*, S. pneumoniae, S. parasanguinis*, Escherichia coli*, Haemophilus influenzae, Klebsiella pneumoniae*, Propionibacterium acnes, Chlamydia trachomatis** (*efficacy for this organism was studied in fewer than 10 infections).

Dosage and Administration:
Instill 1 drop in the affected eye(s) 2 times daily for 7 days.

IMPORTANT SAFETY INFORMATION
Warnings and Precautions:

- Topical ophthalmic use only.
- Hypersensitivity and anaphylaxis have been reported with systemic use of moxifloxacin.
- Prolonged use may result in overgrowth of non-susceptible organisms, including fungi.
- Patients should not wear contact lenses if they have signs or symptoms of bacterial conjunctivitis.

Adverse Reactions:
The most common adverse reactions reported in 1-2% of patients were eye irritation, pyrexia, and conjunctivitis.

For additional information please refer to the accompanying brief summary of prescribing information on adjacent page.

References:
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MOXEZA® solution is indicated for the treatment of bacterial conjunctivitis caused by susceptible strains of the following organisms:
Aerococcus viridans, Corynebacterium macginleyi*, Enterococcus faecalis*, Micrococcus luteus*, Staphylococcus arlettae*, Staphylococcus aureus, Staphylococcus capitis, Staphylococcus epidermidis, Staphylococcus haemolyticus, Staphylococcus hominis, Staphylococcus saprophyticus*, Staphylococcus warneri*, Streptococcus mitis*, Streptococcus pneumonia, Streptococcus parasanguinis*, Escherichia coli*, Haemophilus influenzae, Klebsiella pneumoniae*, Propionibacterium acnes, Chlamydia trachomatis**

*Efficacy for this organism was studied in fewer than 10 infections.

DOSAGE AND ADMINISTRATION

Instill 1 drop in the affected eye(s) 2 times daily for 7 days.

DOSAGE FORMS AND STRENGTHS

4 mL bottle filled with 3 mL of sterile ophthalmic solution of moxifloxacin hydrochloride, 0.5% as base.

CONTRAINDICATIONS

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WARNINGS AND PRECAUTIONS

Topical Ophthalmic Use Only
NOT FOR INJECTION. MOXEZA® solution is for topical ophthalmic use only and should not be injected subconjunctivally or introduced directly into the anterior chamber of the eye.

Hypersensitivity Reactions
In patients receiving systemically administered quinolones, including moxifloxacin, serious and occasionally fatal hypersensitivity (anaphylactic) reactions have been reported, some following the first dose. Some reactions were accompanied by cardiovascular collapse, loss of consciousness, angioedema (including laryngeal, pharyngeal or facial edema), airway obstruction, dyspnea, urticaria, and itching. If an allergic reaction to moxifloxacin occurs, discontinue use of the drug. Serious acute hypersensitivity reactions may require immediate emergency treatment. Oxygen and airway management should be administered as clinically indicated.

Prolonged Use
As with other anti-infectives, prolonged use may result in overgrowth of non-susceptible organisms, including fungi. If superinfection occurs, discontinue use and institute alternative therapy. Whenever clinical judgment dictates, the patient should be examined with the aid of magnification, such as slit-lamp biomicroscopy, and, where appropriate, fluorescein staining.

Contact Lens Wear
Patients should be advised not to wear contact lenses if they have signs or symptoms of bacterial conjunctivitis.

ADVERSE REACTIONS

Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared to the rates in the clinical trials of another drug and may not reflect the rates observed in practice. The data described below reflect exposure to MOXEZA® solution in 1263 patients, between 4 months and 92 years of age, with signs and symptoms of bacterial conjunctivitis. The most frequently reported adverse reactions were eye irritation, pyrexia and conjunctivitis, reported in 1-2% of patients.

USE IN SPECIFIC POPULATIONS

Pregnancy
Pregnancy Category C. Moxifloxacin was not teratogenic when administered to pregnant rats during organogenesis at oral doses as high as 500 mg/kg/day (approximately 25,000 times the highest recommended total daily human ophthalmic dose); however, decreased fetal body weights and slightly delayed fetal skeletal development were observed. There was no evidence of teratogenicity when pregnant Cynomolgus monkeys were given oral doses as high as 100 mg/kg/day (approximately 5,000 times the highest recommended total daily human ophthalmic dose). An increased incidence of smaller fetuses was observed at 100 mg/kg/day. Since there are no adequate and well-controlled studies in pregnant women, MOXEZA® solution should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers

Moxifloxacin has not been measured in human milk, although it can be presumed to be excreted in human milk. Caution should be exercised when MOXEZA® solution is administered to a nursing mother.

Pediatric Use
The safety and effectiveness of MOXEZA® solution in infants below 4 months of age have not been established. There is no evidence that the ophthalmic administration of moxifloxacin has any effect on weight bearing joints, even though oral administration of some quinolones has been shown to cause arthropathy in immature animals.

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

Microbiology
The antibacterial action of moxifloxacin results from inhibition of the topoisomerase II (DNA gyrase) and topoisomerase IV. DNA gyrase is an essential enzyme that is involved in the replication, transcription and repair of bacterial DNA. Topoisomerase IV is an enzyme known to play a key role in the partitioning of the chromosomal DNA during bacterial cell division. The mechanism of action for quinolones, including moxifloxacin, is different from that of macrolides, aminoglycosides, or tetracyclines. Therefore, moxifloxacin may be active against pathogens that are resistant to these antibiotics and these antibiotics may be active against pathogens that are resistant to moxifloxacin. There is no cross-resistance between moxifloxacin and the aforementioned classes of antibiotics. Cross-resistance has been observed between systemic moxifloxacin and some other quinolones. *In vitro* resistance to moxifloxacin develops via multiplestep mutations. Resistance to moxifloxacin occurs *in vitro* at a general frequency of between 1.8×10^{-9} to $< 1 \times 10^{-11}$ for Gram-positive bacteria.

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment of Fertility
Long-term studies in animals to determine the carcinogenic potential of moxifloxacin have not been performed. Moxifloxacin was not mutagenic in four bacterial strains used in the Ames *Salmonella* reversion assay. As with other quinolones, the positive response observed with moxifloxacin in strain TA 102 using the same assay may be due to the inhibition of DNA gyrase. Moxifloxacin was not mutagenic in the CHO/HGPRT mammalian cell gene mutation assay. An equivocal result was obtained in the same assay when v79 cells were used. Moxifloxacin was clastogenic in the v79 chromosome aberration assay, but it did not induce unscheduled DNA synthesis in cultured rat hepatocytes. There was no evidence of genotoxicity *in vivo* in a micronucleus test or a dominant lethal test in mice. Moxifloxacin had no effect on fertility in male and female rats at oral doses as high as 500 mg/kg/day, approximately 25,000 times the highest recommended total daily human ophthalmic dose. At 500 mg/kg orally there were slight effects on sperm morphology (head-tail separation) in male rats and on the estrous cycle in female rats.

PATIENT COUNSELING INFORMATION

Patients should be advised not to touch the dropper tip to any surface to avoid contaminating the contents. Patients should be advised not to wear contact lenses if they have signs and symptoms of bacterial conjunctivitis. Systemically administered quinolones, including moxifloxacin, have been associated with hypersensitivity reactions, even following a single dose. Patients should be told to discontinue use immediately and contact their physician at the first sign of a rash or allergic reaction.

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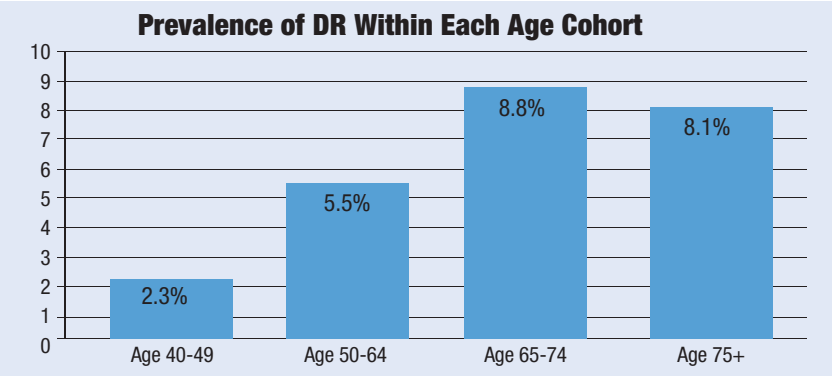
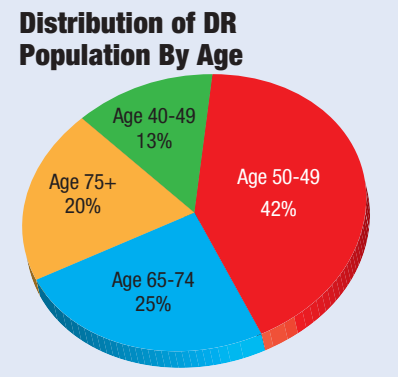
Translation presented some alarming statistics:

- 26 million Americans suffer from diabetes and 79 million more have pre-diabetes.
- One in three U.S. adults could have diabetes by 2050 if current trends continue.
- Diabetic retinopathy is the leading cause of new cases of legal blindness among adults ages 20 to 74 in the U.S.

"Notably, there are seven million people who are undiagnosed," says Jinan Saaddine, M.D., medical epidemiologist at the CDC's Division of Diabetes Translation and leader of its Vision Health Initiative. Dr. Saaddine attributes the burgeoning diabetes rate to several factors, including an aging population, growing trends of obesity and sedentary lifestyles, and an increase in minority populations that are at high risk for developing diabetes.

Taking a closer look at the data reveals emerging trends about which patients are most affected now as well as who'll be at high risk in the future.





DR in Hispanics

Hispanics, already one of the largest ethnic groups in the U.S., accounted for more than half the growth in the total population between 2000 and 2010, according to the latest census. This group added 15.2 million people during the period studied, which constitutes about 56% of the total U.S. population growth of 27.3 million.

Results from the *Vision Problems in the U.S.* report show that Latinos/Hispanics had the highest rate of diabetic retinopathy (8%) compared to whites (5%), blacks (5%) or other groups (4%). With disease prevalence for all U.S. citizens age 40 and older at 5.4%, only Latinos exceeded the national average.

This comes as no surprise to Rohit Varma, M.D., M.P.H., principal investigator of the Los Angeles Latino Eye Study.[1] Dr. Varma's seminal study found that Latinos have higher rates of developing visual impairment, blindness, diabetic eye disease and cataracts than non-Hispanic whites.

"We know for a fact that Latinos have one of the highest prevalence rates of type 2 diabetes in the U.S.," Dr. Varma says. "And, the overall rate of Latinos who have diabetic retinopathy is on the higher end than non-Hispanic whites."

Treating Diabetic Retinopathy in New Mexico

According to the *Vision Problems in the U.S.* report, New Mexico is the state with the highest prevalence of diabetic retinopathy in the nation (6.6%). For Brent Shelley, O.D., who practices in La Mesilla, N.M., this is no surprise.

"I would say that it corresponds with the rates seen in our practice," says Dr. Shelley, president of the New Mexico Optometric Association. "Our state has a high percentage of Hispanics and Native Americans, and these ethnicities, in turn, have high incidences of diabetes. Furthermore, our state is very rural by nature and primary care, in general, is underserved. Hence, outreach programs are very much needed so that we can educate these persons to reverse this statistic."

While the rates of diabetes and diabetic retinopathy are increasing, Dr. Shelley says he has not seen an increase in sight-threatening cases. "However, I will say that early diabetic retinopathy is becoming more frequent," he says.

Because Dr. Shelley's practice is located in a medical plaza, he receives many primary care referrals. "We educate our patients thoroughly, maintain an aggressive recall strategy and correspond with the patient's physician regarding their ocular health," he says.

For Dr. Shelley, the optometrist's standard of care in a diabetic patient is to dilate and educate the patient on the pathophysiology of diabetes. "In our practice, we will not see a diabetic patient without dilating their eyes. While some practices may rely on fundus imaging for retinal exams, I would argue that this does not meet the standard of care and is not in the patient's best interests," he says.

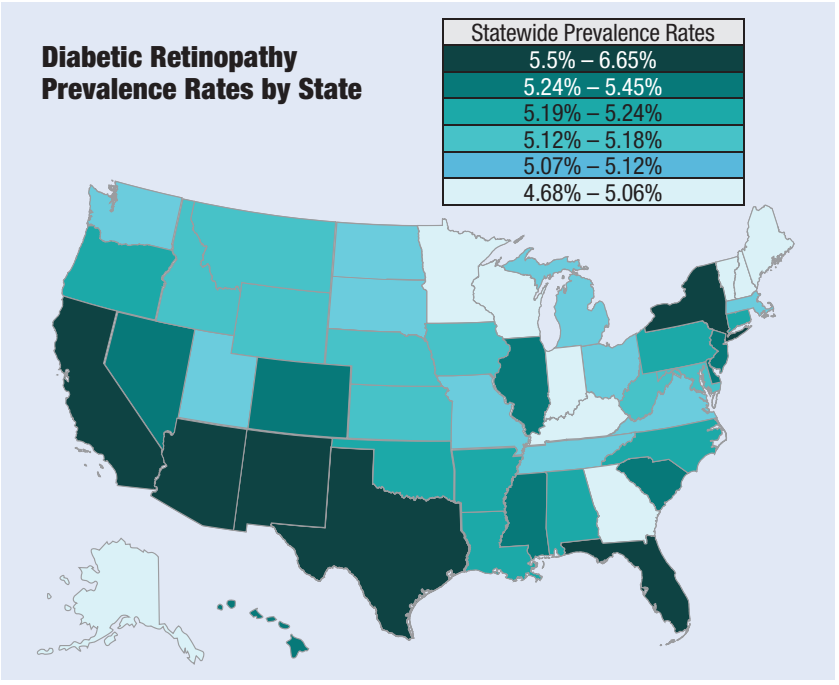
Studies are finding a high prevalence of diabetic eye disease among Hispanics because this ethnic group has a greater genetic predisposition to retinopathy than non-Hispanic whites, Dr. Varma says. "My belief is that their risk of developing retinopathy is higher at the same level of the risk factor. For example, Hispanics have a greater rate of developing eye disease than others with same blood pressure level." Dr. Varma says this is primarily due to a greater genetic predisposition in Hispanics.

In addition to a predisposition to develop diabetes, socioeconomic factors may play a part in higher prevalence rates of diabetic retinopathy in certain ethnicities, such as Latinos and blacks, Dr. Chous says. These factors include lack of access to high-quality health care and even grocery stores, he adds.

The report's finding that blacks had the same diabetic retinopathy prevalence rate as whites came as a surprise, Dr. Chous says, as previous reports indicate that blacks are about 50% more likely to develop retinopathy, compared to their European-American counterparts.[2] This statistic has been attributed to poorer health care access and higher rates of hypertension in the former community.[2]

"If this extrapolated statistic is true, this new finding may reflect the success of increased public and community education efforts developed within and aimed at the African-American community," Dr. Chous says.

However, it could also be related to higher rates of diabetic retinopathy among all Americans with diabetes in a bad economy where the financial costs of good self-care can take a back seat to other financial demands, he adds. "This is not speculation. I saw a 25-year-old unemployed, type 1 Caucasian paramedic earlier this year who just lost his vision to proliferative retinopathy after taking his insulin injections every other day to save money," Dr. Chous says.



Keeping Statistics in Mind

The 89% spike in diabetic retinopathy cases as detailed by the *Vision Problems in the U.S.* report is a bit misleading, as this is not a medical study but rather a demographic statistical report, says Ojai, Calif. optometrist Roger Phelps, one of the few O.D.s who is also a certified diabetes educator. Dr. Phelps has had type 1 diabetes for more than 20 years. "The 89% figure mainly reflects the increasing number of people with diabetes in our country, not a disease process that is getting out of control," says Dr. Phelps. "The opposite is actually true: Although we have many more patients with diabetes, these individuals can be offered a plan to greatly reduce their risk of vision loss from diabetes."

All the studies Dr. Phelps has reviewed agree with the report relative to the increasing number of people in the country with diabetes, due to population increases—especially in the Baby Boomer generation—as well as an increasing percentage of the population who have diabetes.

However, many studies show those individuals with diabetes are much less likely to lose vision now than 15 years ago with proper education and care, he adds. Dr. Phelps cites the Wisconsin Epidemiology Study of Diabetic Retinopathy, which found the lower risk of proliferative diabetic retinopathy in more recently diagnosed patients possibly reflects improvement of care.[3]

Dr. Chous offers another possibility for the high prevalence of diabetic retinopathy: Because diabetic retinopathy detection has increased as a function of improved technology (e.g., greater use of retinal imaging with red-free filters, widefield imaging and OCT), rates

of actual disease (per 1,000 people diagnosed with diabetes, for example) may not have grown as much as it would appear.

While the 89% figure certainly is significant, it is also critical to distinguish between nonspecific diabetic retinopathy (mild to severe) and sight-threatening diabetic retinopathy (proliferative diabetic retinopathy and clinically significant macular edema) that causes visual disability, Dr. Chous says, as most cases of diabetic retinopathy do not lead to vision loss.

From 2005 to 2008, 4.2 million (28.5%) people with diabetes age 40 or older had diabetic retinopathy; of these, 655,000 (4.4% of those with diabetes) had advanced diabetic retinopathy that could lead to severe vision loss.[2]

"Also, every retinal specialist will tell you that rates of severe vision loss from diabetic retinopathy have gone down with improvements in diabetes care—

The Nuts and Bolts of the *Vision Problems in the U.S.* Report

- The study found states with the highest prevalence of diabetic retinopathy, in order, are New Mexico (6.6%), Florida (6.0%), Texas (5.9%), California (5.8%), Arizona (5.8%), the District of Columbia (5.5%) and New York (5.5%). Why these particular states? Dr. Varma attributes it to a higher Hispanic and aging population in the majority of these states. Interestingly, however, the CDC reports that counties and states in the southeast region of the country have the highest rates of diabetes, according to Dr. Saaddine.
- The age group with the highest prevalence of diabetic retinopathy by percent (among both men and women) is 65 to 74. The age group with the highest number of cases is 50 to 64 (3.2 million).
- Data from this report was derived from pooling information from 12 major epidemiological studies and accounting for the number of individuals with diabetic retinopathy, the number at risk, age, race and gender. U.S. Census 2010 data was then applied to generate the final prevalence rates in the various categories.
- The numbers reported in this study are estimates, not exact measurements. All data from the report can be obtained through a new searchable database on the Prevent Blindness America website: www.preventblindness.org/visionproblems. This tool enables users to research a wide range of information, including eye disease and condition numbers broken down by state, age, gender and race, and provides comparisons across disease conditions.

especially blood glucose—improved detection and earlier diagnosis, and better treatments to prevent vision loss," says Dr. Chous.

However, even mild diabetic retinopathy has been linked to increased risk of other diabetes complications, including kidney and cardiovascular disease, so from a public health perspective, increased rates or detection of diabetic retinopathy are important and underscore the need to prevent the onset of diabetes, Dr. Chous adds.

The Optometrist's Role in DR

As studies such as the Vision Problems in the U.S. report find that more patients are developing diabetes—and diabetic retinopathy as a result—the optometrist's role in treating diabetes patients becomes more critical than ever.

"We can encourage our patients to get to know their disease, and as they do, they can fully participate in their care and do much to stop the loss of vision that was so prevalent in the past," Dr. Phelps says. He recommends optometrists implement the following in caring patients with diabetes:

• ***Know your patient's A1C level, and encourage patients to know it as well.*** (See the *"The ABCs of A1Cs"*, at right.) If a patient's A1Cs are staying significantly over 7%, let them know that they are at a higher risk of retinopathy even though there may not be any diabetic changes in their eyes today. Encourage them to discuss and agree upon the appropriate A1C level for their individual medical status with their primary care physician. When a patient who returns for an annual dilated exam shows increasing retinopathy, it is time to move the slit lamp aside and have another discussion about glycemic control. Realize that, even with an improved A1C, ocular changes can take place up to three years later from prior poor control.[4] If a patient's A1C is still above the goal set with their physician and increasing retinopathy is present, it may be best to get a retinal consult even before pre-proliferative changes are apparent.

• ***Perform a dilated exam annually.*** "We may find many patients with non-threatening mild non-proliferative retinopathy, but let the patient know of the importance of regular eye exams, as we can also spot asymptomatic sight-threatening changes," Dr. Phelps says.

• ***Always send a report of your retinal findings to the patient's primary care physician after the exam, even if there are no signs of diabetic retinopathy.*** This report can simply state: "No diabetic retinopathy."

• ***Consider an OCT if a patient is developing even moderate levels of diabetic retinopathy.*** "I recommend an OCT sooner rather

The ABCs of A1Cs

As the diabetic population in the U.S. continues to grow, Dr. Phelps recommends that every optometrist should at least know and document their diabetic patients' A1C levels.

A1C is a blood test that gives the average amount of glucose in the blood over the past three to four months. An A1C of 5.6% or below is normal. In pre-diabetes, A1C levels range between 5.7% and 6.4%. If the A1C is 6.5% or above, the patient has diabetes.

Also be aware that if your patient's A1C level increases, there generally is a three-year time delay until it will impact their eyes. Refractive shifts can show up quickly with a rapid A1C change as well, Dr. Phelps says.

"If a person comes in, and their A1C had been maintained at around 7% but now it's up to 12%, it may be two to three years until it affects their eyes, but when it does, there is very little you can do to get it back," Dr. Phelps says. "If the A1C levels are truly going up, even if there is no diabetic retinopathy, I am still very concerned."

Another critical factor to keep in mind: In general, every percentage point drop in A1C blood test results (e.g., from 8% to 7%) can reduce the risk of microvascular complications (eye, kidney and nerve diseases) by 40%.[1]

"Optometry needs to learn the importance of monitoring their patients' A1Cs. If a patient doesn't know what their level is, you need to ask them: 'May I call your doctor?'" Dr. Phelps says.

Optometrists should encourage their patients with diabetes to see their primary care physician regularly so their doctor can monitor them regularly and set their A1C goals. They should also get to know the physicians in their area and personally call their patients' primary care doctors, to find out A1C levels, Dr. Phelps advises.

The A1C level may affect the glasses you prescribe now, as well as their risk of retinopathy in the future. You can let the M.D. know that you just saw your common patient, and although the patient's eyes show no diabetic retinopathy (or level of retinopathy), you want to know the patient's current A1C or recent history of A1C. If the patient's A1C is variable, recheck the refraction in a few weeks. If the A1C has been stable over the past three to six months, you can prescribe glasses right away, Dr. Phelps says.

Developing a dialog with the patient's primary care physician can help the optometrist become a "team member" in the care of their mutual patient, Dr. Phelps adds.

1. Centers for Disease Control and Prevention. National diabetes fact sheet: national estimates and general information on diabetes and prediabetes in the United States, 2011. Atlanta. U.S. Department of Health and Human Services, Centers for Disease.

than later, because sometimes it might show changes you might not normally see, and it will provide a baseline," Dr. Phelps says. "When I see a few minor changes in the retina such as microaneurysms and small blot hemorrhages, it is mild. When more of these are present in more than three quadrants combined with some cotton wool spots, it gets into the moderate range," Dr. Phelps explains.

• ***Work closely with retinal specialists in your area and make timely referrals.***

"Although we have a confirmed epidemic increase in our national patient population of those with diabetes, we have made major progress in individual prevention of vision loss," Dr. Phelps says. Most vision loss from diabetes has now been shown to be preventable by teaching patients the importance of controlling their diabetes (as best reflected in the A1C testing two to four times a year), and for them to return each year for a dilated eye exam to discern the need for timely laser treatment or anti-VEGF injections for DME, he adds.

Diagnostic Codes for DR

- Mild non-proliferative diabetic retinopathy: 362.04
- Moderate non-proliferative diabetic retinopathy: 362.05
- Severe non-proliferative diabetic retinopathy: 362.06

Patients who have suffered vision loss as a result of diabetic retinopathy also need low vision rehabilitation, in addition to managing their diabetes, Dr. MacDonald adds.

"Optometrists are really good at educating patients, but we are going to have to do even better," Dr. MacDonald says. "Diabetic retinopathy is the leading cause of blindness among those of working age, and it doesn't have to be that way." With appropriate treatment, she says, progression can be stopped. "There are health disparities among racial and ethnic groups that we need to be aware of. We need to get people in the habit of, and emphasize the need for, complete dilated exams even if there are no symptoms. Finally, we need to have good communication not only with the patient, but also the rest of the health care team." ■

1. Varma R, Ying-Lai M, Klein R, Azen SP. Prevalence and risk indicators of visual impairment and blindness in Latinos: The Los Angeles Latino Eye Study. Ophthalmology. 2004 Jun;111(5):1132-40.

2. Zhang X, Saaddine JB, Chou CF, et al. Prevalence of diabetic retinopathy in the United States, 2005-2008. JAMA. 2010 Aug 11;304(6):649-56.

3. Klein R, Knudtson MD, Lee KE, et al. The Wisconsin Epidemiologic Study of Diabetic Retinopathy: XXII the twenty-five-year progression of retinopathy in persons with type 1 diabetes. Ophthalmology. 2008 Nov;115(11):1859-68.

4. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. The Diabetes Control and Complications Trial Research Group. N Engl J Med. 1993 Sep 30;329(14):977-86.

14th Annual Diabetes Report

Can Myopia Delay Diabetic Retinopathy?

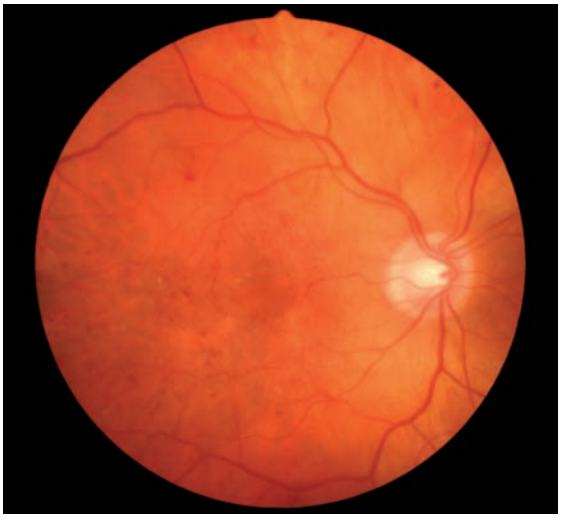
These two common conditions can interact as well as simply coexist. But, that may be a good thing. **By Madhavendra Bhandari, M.Phil. Opt.**

In recent decades, diabetic retinopathy (DR) and myopia have been increasing in prevalence; both contribute greatly to visual impairment.[1-4] Diabetes has been linked to changes in refractive error under hyperglycemic conditions. It is essential to understand this relationship when determining the proper refractive correction; hyperopic rather than myopic shift is the more common effect, although the literature has been somewhat contradictory on that in the past.[5-19] Interestingly, refractive error—especially high myopia—may actually decrease the progression of DR, even though it is associated with serious ocular complications, such as an increased risk of retinal detachment.[20-24]

This paper will summarize the relationship between refractive status and fluctuations in glucose levels, and attempt to explain the different theories that various authors have proposed over the years. This review also will help us learn more details about the relationship between refractive error and DR pathogenesis, to help us uncover the mechanisms of a possible protective effect in highly myopic eyes.

Hyperglycemia and Refractive Error

The poor metabolic control of blood glucose that diabetes patients experience, particularly hyperglycemia, leads to changes in the eye's refractive status. Although the effect on refraction has been investigated by many studies—some dating back at least to 1925—there is no agreement in the literature on the mechanism or the typical disease course. Indeed, both hyperopic and myopic shifts in subjects with diabetes have been documented by several studies.



Diabetic retinopathy in a moderately myopic patient (-5.00D). Research has shown an inverse relationship between myopia level and risk of proliferative DR.

Notable additions to the literature on myopic shift under hyperglycemic conditions have come from Duke-Elder (1925), Birnbaum & Leu (1975), Gwinup & Villarreal (1976), Fledelius (1990) and Furushima (1999). Scholars documenting hyperopic shift during intensive treatment of acute metabolic dysregulation include Planten (1975 & 1978), Kluxen & Scholz (1987), Saito (1993), Okamoto (2000), Giusti (2003) Sonmez (2005) and Tai (2006).[5-17] Verma (1980) and Herse (2005) induced hyperglycemia in rabbits and found hyperopic shift upon refraction.[18,19]

Duke-Elder suggested that

hyperglycemia causes myopic shift, while a decrease in blood glucose levels leads to hyperopic shift due to the osmotic force between the crystalline lens and the aqueous humor that results from changes in molecular concentration.[5] This same theory was suggested by Gwinup (1976), who studied the phenomenon in 10 subjects with diabetes, including four aphakic eyes, and concluded that higher and lower glucose levels produce myopia and hyperopia, respectively.[7] Although a few early studies had suggested that hyperglycemia leads to myopic shift, noting that the eye's refractive error shifted towards hyperopia as the glucose level reduced, later studies observed precisely the opposite—during hyperglycemic states, the refractive shift is more likely hyperopic. Fledelius (1990) studied 15 subjects with high glucose levels and documented refractive fluctuation of 1.00D to 6.50D toward hyperopia.[8]

Some studies in which hyperglycemia was induced have shown a change in the thickness and/or curvature of the lens, altering its refractive index. Sito and associates (1993) stated that thickening of the lens correlates with the refractive shift towards hyperopia. This team also found a decrease in anterior chamber depth.[13] Huggert (1953-54) studied ways in which diabetes affects the crystalline lens and suggested that changes occur anterior to the nucleus.[22,23] Both in early-onset and late-onset diabetes, there is change in lens thickness when compared with healthy, subjects without diabetes.[24,25] Kluxen and Scholz (1987) reported a case demonstrating enlargement of the axial diameter of the cortex and nucleus, as revealed by Scheimpflug photography during an episode of transient hyperopia.[12]

After inducing acute hyperglycemia in seven healthy subjects, Furushima (1999) found an increase in lens thickness of 1mm and a myopic shift of 2.00D.[9] Kato (2000) reported a significant increase in lens thickness (0.3mm) after rapid control of hyperglycemia.[26] In a small study, Wiemer (2009) reported that one of his five subjects experienced hyperopic shift during induced acute hyperglycemia, but there was no change of refractive status in the other four cases.[27] The authors reiterated the theory that the hyperopic shift in their one case could have been due to changes in the shape of lens. Lending credence, their report also showed no change to either the anterior and posterior corneal curvature.

In a study investigating the optical properties of LeGrand's "full theoretical eye," Planten (1975) demonstrated that a decrease in the refractive index of

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the lens from 1.42 to 1.40 would produce a hyperopic change of 3.20D.[11] The same author found changes in lens thickness, as measured by A-scan ultrasound, and suggested the hyperopic shift was caused by changes in the refractive indices of the different layers of the lens.[10]

Herse (2005) found a similar hyperopic shift in rabbit eyes and suggested it could be due to changes in the refractive index of the cortical fibers of the lens.[19] It is important to note that blurred vision during hyperglycemia also can be caused by factors other than refractive changes.[26] For instance, changes in metabolism can also lead to alterations in media transparency and autofluorescence. In addition, tear film instability secondary to increased tear osmolarity can reduce visual acuity in patients with poorly controlled diabetes.[28]

Both corneal and lens autofluorescence are higher in the diabetic population than in healthy control groups because of fluorophore accumulation in the cornea and lens of diabetic patients, which is increased in proportion to the level of glycemic control. The concentration of glucose in aqueous humor increased along with glucose levels when there was a breakdown of the blood-aqueous barrier, giving rise to autofluorescence.[29]

Although many of the studies cited above noted refractive shifts in hyperglycemic subjects, bear in mind that only a few found significant changes in refractive error, and the effect tends to be transient. The physiology of the anterior segment and chamber—and their relationship to refractive error during hyperglycemia—still need to be studied in greater detail, but the prevailing theory seems to be that hyperopia might result from intraocular alternations of the lens that change its thickness and hence its refractive index.

Refractive Error and the Myopic Retina

High myopia is associated with progressive elongation of the globe, resulting in a variety of fundus changes that lead to visual impairment, including lacquer cracks in Bruch's membrane, choroidal neovascularization and chorioretinal atrophy. Increased axial elongation in myopes may also lead to mechanical stretching and thinning of the choroid and retinal pigment epithelium, resulting in posterior staphyloma.[30,31] Histopathologic studies in postmortem eyes indicate that occlusion and disappearance of large choroidal vessels and capillaries, and consequent replacement of the normal choroidal structure with fibrous tissue, are common.[32-34]

Myopic refractive error and longer axial length are associated with narrower retinal arterioles and venules, less tortuous arterioles and increased branching coefficients in both arterioles and venules.[35] This reduces retinal blood flow and, as a result, can potentially decrease the risk of DR development. Complete posterior vitreous detachment (CPVD) was more frequently noted in myopic eyes and this finding was associated with reduced progression of neovascularization.

DR usually develops in a symmetric pattern over a long period of time; development of asymmetric DR is exceptionally rare. However, high myopia is one finding that can be responsible for asymmetric DR retinopathy, along with other ocular factors such as optic atrophy and PVD, and systemic factors such as glycosylated hemoglobin level and high blood pressure. The Early Treatment of Diabetic Retinopathy Study defined asymmetric DR as proliferative diabetic retinopathy (PDR) in one eye and non-proliferative diabetic retinopathy (NPDR) or no retinopathy in the fellow eye persisting for more than two years.[36] Asymmetric DR occurs in 5% to 10% of diabetic patients with PDR.[37]

Some studies of anisometropic cases (defined as a difference of more than 1.00D between eyes) have shown that the eye with the higher level of myopia had a lesser degree of retinopathy, while the fellow eye typically demonstrated a higher degree of retinopathy.[38,39] This showed that anisometropia plays a role in asymmetric DR presentations, and suggests that the degree of refractive error is inversely related to the severity of DR.

Dogru (1998) showed that high myopia along with optic atrophy leads to a decreased risk of PDR.[40] He suggested that high myopia associated with elongation of the eyeball, and atrophy leads to a decrease in blood flow in the retinal blood vessels.

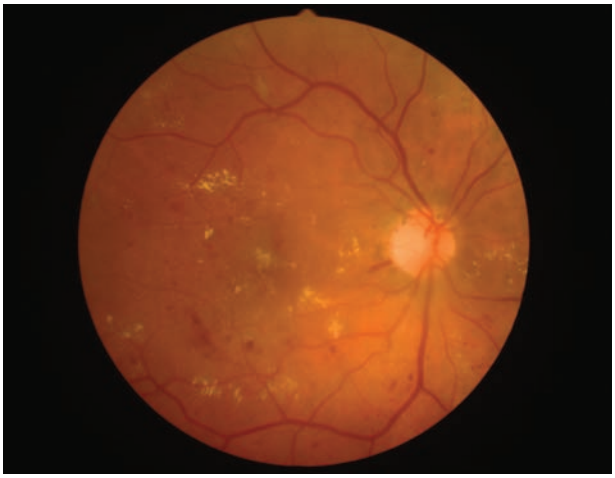
High Myopia as a Protective Factor

There are some clinic- and population-based studies showing that myopia—especially higher myopia—has a protective effect against DR.[41,42] Both of the population-based studies, the Wisconsin Epidemiologic Study of Diabetic Retinopathy (WESDR) and Singapore Malay Eye Study (SiMES), suggested that high myopia reduces the progression of DR.

WESDR defined myopia as spherical equivalent of -2.00D or more and found that myopia was associated with a lower risk of development of PDR in younger-onset diabetes. The authors

suggested that myopia confers a protective factor through axial globe elongation, deformation of the posterior pole and alteration of the ocular blood flow.

SiMES defined low myopia as a spherical equivalent (SE) between -0.50D and -5.00D, high myopia as -5.00D or more SE, and hyperopia as >1.00D SE. The study's results suggested that highly myopic patients, with a deeper anterior chamber depth and longer axial length, have a lower risk of developing DR, and particularly sight-threatening PDR.



Hyperopic patients with diabetes may be at higher risk for sight-threatening retinopathy, as in this individual with a +1.00D prescription.

Eyes with myopia are more likely to have a longer axial length, elevating the risk of complete vitreous detachment. Shorter rather than longer axial length is a risk factor for a higher level of DR.[43] Partial posterior vitreous detachment with vitreous traction is a high risk factor for development of PDR. Incomplete PVD is usually associated with severe fibrovascular proliferation.[44,45] The vitreoretinal traction causes the progression of new blood vessels and thickening of existing fibrous proliferation.[45] Thus, incomplete PVD results in an elevated risk of developing PDR as well as vitreous hemorrhage, retinal detachment and retinal breaks. In complete vitreous detachment without vitreous traction, there is less chance of such progression.[47]

In myopia, complete posterior vitreous detachment and vitreous syneresis are more common than other refractive errors, and they protect against development of PDR by reducing the likelihood of neovascularization progression.[47-50] Reports also have suggested that vitrectomy may be a protective factor against diabetic macular edema in cases of vitreomacular traction.[51]

Conclusion

A thorough review of the literature suggests that is it more likely that hyperopia rather than myopia occurs in cases of uncontrolled diabetes. Greater understanding of the predisposing factors and mechanisms would improve our ability to properly manage these cases.

Patients may require frequent changes of spectacles in the presence of persistently high hyperglycemia. Consequently, optometrists are advised to delay the prescription of new glasses as long as possible until stabilization of refraction (and glucose levels) is noted; potential benchmarks of stabilization to consider are an HbA1C below 7.5% and a casual in-office blood glucose of less than 180mg/dl. We must also educate our patients about the causal relationship between poor blood glucose control and the risk of severe retinopathy as well as significant vision loss.

Even though high myopia may be associated with an increased risk of ocular complications, it can act as a protective factor that forestalls proliferative DR. Lastly, be mindful to consider high myopia as a risk factor for asymmetric DR.

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(continued from page 33)

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• Student Party, Saturday, Sept. 8, 7:00 p.m. – 9:00 p.m. (Location to be announced.)

14th Annual Diabetes Report

Contemporary Care Protocols for DR and DME

New treatment paradigms for diabetic eye disease are enabling our patients to enjoy improved visual function and sustained visual acuity gains.

By Carlo J. Pelino, O.D., and Joseph J. Pizzimenti, O.D.

Diabetic retinopathy (DR) is the leading cause of blindness in the working-age population of the western world. As the number of people living with type 2 diabetes mellitus (DM) continues to rise, eye care providers are seeing more cases of DR than ever before. In general, lethargy and obesity in children, adolescents and adults have widely contributed to this increased prevalence in type 2 DM.

DR is a microvascular disease. Proliferative DR is characterized by new vessel formation in the retina and optic disc as a result of hypoxia, microangiopathy and capillary occlusion. Tractional retinal detachment, diabetic macula edema (DME) and neovascular glaucoma are associated complications that may result in severe vision loss.

In 2005, we experienced a sea change in the preferred treatment of exudative age-related macular degeneration (AMD)—a shift from ablative therapy to pharmacotherapy. Now, another sweeping transition is occurring in the treatment of DR and DME, with thousands of patients achieving improved visual outcomes with fewer complications.

The Burden of Disease

People with DM are at an increased risk for a multitude of ocular complications. Common ocular symptoms in patients with DM include blurred or fluctuating vision, diplopia and ocular surface dryness. These individuals are 40% more likely to develop glaucoma and 60% more likely to develop cataracts than those without DM, according to the American Diabetes Association (ADA).[1] More specifically, cataracts develop earlier and progress faster in patients with DM, and the risk of glaucoma increases with advanced age and longer disease duration.[2,3]

Other ocular anomalies associated with DM include decreased corneal sensitivity, iris neovascularization, pupillary abnormalities secondary to autonomic neuropathy, fluctuating refractive error associated with sorbitol in the lens,

Release Date: August 2012
Expiration Date: August 1, 2015
Goal Statement: Diabetic retinopathy (DR) is the leading cause of blindness in the working-aged population of the western world. As the number of people living with type 2 diabetes mellitus continues to rise, eye care providers are seeing more cases of DR than ever before. Traditional treatment of DR still is an effective management approach. But, with newer, more effective options, such as MPLT and pharmacotherapy, the outcome profile for affected patients is changing for the better.
Faculty/Editorial Board: Carlo J. Pelino, O.D., and Joseph J. Pizzimenti, O.D.
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a "snowflake" cataract in patients with type 1 DM, optic nerve abnormalities and other cranial nerve neuropathies.[4,5]

Both the prevalence and the economic burden of DM and DR are on the rise.[6] Patients with DR represent a large and growing segment of the American population with vision impairment. The Centers for Disease Control and Prevention estimates that DR causes 12,000 to 24,000 new cases of blindness each year.[6] In fact, patients with DM are 29 times more likely to become blind than individuals of similar age and gender without diabetes.[7,8]

A loss of fine detail in central vision typically is one of the first and most common symptoms in patients with DR. Night vision problems, flashes and floaters are other, less common complaints. Severe and moderate levels of vision loss secondary to DR often are preventable with timely detection and treatment.[9-14]

Patients with DM may not understand the importance of dilated retinal examinations or recognize the benefits of early disease detection. Survey data published by the National Eye Institute indicated that just about half of all patients with DM obtained an annual dilated retinal exam.[15]

Pathophysiology of DR

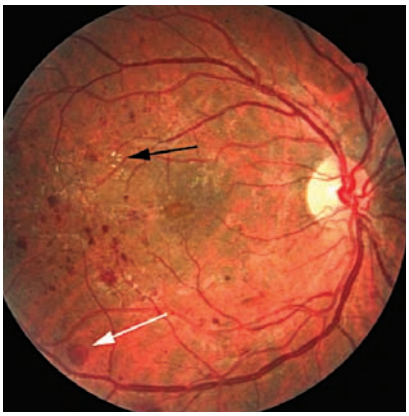
Most individuals with DM ultimately develop some degree of retinopathy.[13,16] DR results from an alteration in retinal blood flow that degrades the retina's performance. DR affects the retinal capillaries before it impacts the larger vessels.[17-19] However, the exact cause of microvascular complications in DM is unknown.[17,18]

An early finding in DR is the loss of pericytes, which may cause leakage and dysfunction of capillary endothelial cells.[17,18] Pericytes are the modified smooth muscle cells of capillaries that regulate retinal vascular flow via dilation and contraction. These mural cells provide structural support for the capillaries' endothelium and help constitute the inner blood-retinal barrier.

Excess glucose within the retinal capillary is thought to stimulate production of vascular endothelial growth factor (VEGF), protein kinase C and advanced glycation end-product.[17,20] These biochemicals alter the capillary pericyte integrity. Over time, non-perfusion weakens the capillary walls, resulting in bulging, leaking or scarring. Outpouchings of the capillaries (microaneurysms) frequently are the earliest clinically detectable sign of DR. With tissue ischemia, angiogenic growth factors (such as VEGF) are upregulated and released, causing neovascularization and increased vascular permeability—both of which lead to retinal edema.[18,21,22]

Leakage from the perifoveal vessels may cause DME, which manifests as swelling or thickening of the central retina. DME continues to be a common cause of central vision loss and decreased quality of life in working-aged Americans.[23,24] Results from the Wisconsin Epidemiologic Study of Diabetic Retinopathy indicated that, after 15 years of known diabetes, the prevalence of DME is approximately 20% in patients with type 1 DM, 25% in patients with type 2 DM who take insulin and 14% in patients with type 2 DM who do not take insulin.[25] In the United States, the incidence of DME approaches 30% in adults who have had DM for 20 years or more.

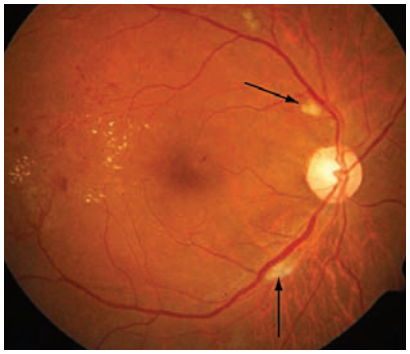
Also of note: The prevalence of DME in mild non-proliferative diabetic retinopathy (NPDR) is just 3%. However, this statistic balloons to 38% in eyes with moderate to severe NPDR, and 71% in eyes with proliferative retinopathy (PDR).[25]



1. This patient presented with non-proliferative diabetic retinopathy. Note several dot- and blot-shaped hemorrhages (white arrow) as well as hard exudates (fine yellow deposits, located adjacent to the black arrow).

DME occurs as a consequence of both vascular abnormalities and inflammatory processes. Both components interact with each other to promote disease progression. The vascular abnormalities stimulate the inflammatory processes that eventually lead to further vascular compromise and leakage. Hypoxia, altered blood flow, ischemia, toxicity and inflammation are processes that cause macular edema formation. Compromise to the inner blood-retinal barrier causes increased vascular permeability as well as extravasation of lipoproteins and other macromolecules. Over time, accumulation of intraretinal fluid in combination with macular thickening causes decreased visual acuity.[19]

Much more is now understood about the origins of diabetic inflammation. Leukocytes are recruited to the retinal vasculature after the retinal tissue has been stressed. Intracellular adhesion molecules eventually are expressed on the luminal surface of the



2. Note multiple areas of "cotton-wool" infarct (black arrows) in a patient with non-proliferative diabetic retinopathy.

vascular endothelial cells, which allows the adhesion of leukocytes and possible blockage of the capillary (known as leukostasis). In DR, the white blood cells no longer flow freely within the retinal vessels. Eventually, they damage and kill the cells that line the blood vessel wall. After adhesion occurs, several chemotactic molecules, such as monocyte chemo-attractive protein-1 (MCP-1), are secreted by the vascular lumen. MCP-1 influences the migration of leukocytes into the retinal tissues.

Once the leukocyte is inside the retina, a variety of inflammatory cytokine mediators, such as interleukin-1 beta, interleukin-6, tumor necrosis factor-alpha (TNF-alpha), insulin-like growth factor 1, stromal-cell derived factor-1 and VEGF-A, are secreted and propagate the vascular permeability process.[19]

Also, the insulin-sensitizing agents Avandia (rosiglitazone, GlaxoSmithKline) and Actos (pioglitazone, Takeda Pharmaceuticals) may increase the risk of DME in patients with type 2 DM who experience peripheral edema and weight gain.[26] So, if your patients are using either agent, you may wish to switch them to an entirely different category of diabetes medications.

Clinical Features and Classification of DR

The classic clinical features of NPDR include dot- and blot-shaped hemorrhages, microaneurysms, intraretinal microvascular abnormalities (IRMA), venous beading, hard exudates (lipid), cotton-wool spots and retinal edema (*figure 1*).

Cotton-wool spots represent focal infarcts of the retinal nerve fiber layer (*figure 2*). IRMA are dilated and tortuous capillaries, and are good indicators of progressive DR. Venous beading is a focal irregularity in the caliber of retinal veins that serves as a strong predictor for the development of neovascularization.[27]

Ruptured microaneurysms, leaking capillaries and IRMA may result in intraretinal hemorrhages. The ophthalmoscopic appearance of these hemorrhages is consistent with the retinal level in which they occur.

Hemorrhages in the retinal nerve fiber layer have a flame-shaped appearance that is consistent with the layer's structure. (Also, note that flame-shaped hemorrhages typically occur in patients with hypertension.[27]) Hemorrhages located deeper in the retina assume a pinpoint, blot or dot shape and are more characteristic of DR.

While NPDR is characterized by a microangiopathy that involves intraluminal, intramural and extravascular damage, PDR is an entirely different disease entity. The hallmark of PDR is the formation of new blood vessels at the vitreoretinal interface and in the vitreous itself. Fibrovascular tissue proliferates on the surface of the retina, optic nerve and/or iris. After 15 to 20 years of DM, this proliferative form affects about 50% of patients with type 1 DM; 5% to 10% of patients with non-insulin-dependent type 2 DM; and 30% of patients with insulin-dependent type 2 DM.[28]

For an eye to be classified as having PDR, it must exhibit one or more of the following characteristics: neovascularization of the optic nerve head or disc (NVD); neovascularization elsewhere (NVE); or vitreous or pre-retinal hemorrhage associated with NVE (*figure 3*).[29]

Leakage from perifoveal vessels causes DME, which could result in permanent central vision loss if left untreated. This edema can occur at any stage of retinopathy, whether proliferative or non-proliferative. For macular edema to be classified as clinically significant (CSME), an eye must exhibit one or more of the following characteristics: thickening of the retina within 500µm of the center of the macula; hard exudates within 500µm of the center of the macula with associated thickening of the adjacent retina; or at least one zone of retinal thickening that is greater than one optic disc diameter in size.[27,29]

Is "tomographically significant" macular edema—visible on OCT but not on fundoscopy—changing the definition of what constitutes CSME? Clinical evidence certainly suggests so, because OCT yields both qualitative and quantitative information about retinal thickness and edema.

Thus, OCT can be helpful, in a non-invasive manner, to support a diagnosis of CSME or to rule it out. On OCT, CSME is represented by increased retinal thickening due to intraretinal fluid leakage, which appears as hyporeflective (dark) areas on the cross-sectional image (*figure 4*). Additionally, OCT is valuable in monitoring the eye's response to treatment.[30]

Further, there is now evidence that OCT plays a defining role in determining DME treatment

criteria. Researchers from the Diabetic Retinopathy Clinical Research Network (DRCR.net) have recommended OCT-measured central retinal thickness values greater than or equal to 250µm as an eligibility criterion for at least 11 study protocols. In the RISE and RIDE studies, which investigated the efficacy of intravitreal ranibizumab in treating DME, an OCT-measured central subfield thickness greater than or equal to 275µm was used as one of the two eligibility criteria for treatment.[30] Subsequently, the Safety and Efficacy of Ranibizumab in Diabetic Macular Edema With Center Involvement (RESOLVE) trial used an OCT-measured central retinal thickness of greater than or equal to 300µm, as well as with visual acuity parameters, as eligibility criteria for the treatment of DME with ranibizumab.[30]

As these studies indicate, OCT imaging could enable closer monitoring, more intensive systemic diabetes management and more timely treatment of DME.[30]

Management of Concomitant Diseases

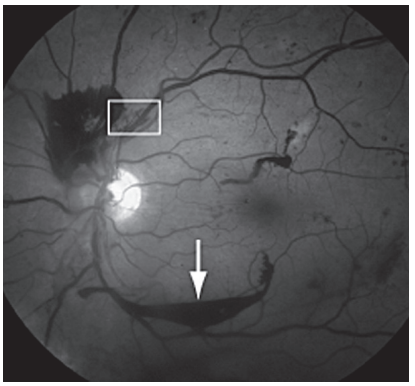
Proper management of associated systemic disorders ultimately influences the onset, progression and visual outcome of DR.

• ***Hypertension.*** Systemic hypertension is an established risk factor for the development and progression of retinopathy. In the third U.S. National Health and Nutritional Examination Survey, hypertension was documented in up to 75% of adults with diabetes.[31] Research has shown that individuals with DM and hypertension are more likely to develop DR, and likely experience more rapid disease progression compared to DM patients without hypertension.[9] Additionally, DM patients with concomitant hypertension are up to three times more likely to develop DME.

Fortunately, reduced blood pressure levels have been shown to decrease the risk of DR progression.[9] Several antihypertensive agents have been evaluated for their therapeutic effect on DR. Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers have been shown to reduce DR progression in normotensive patients with type 1 DM and mild DR.[32] In multiple trials, aggressive blood pressure treatment was accompanied by improved outcomes in both retinopathy and nephropathy. As a result, the Joint British Diabetes Societies have recommended that both type 1 and type 2 DM patients should be aggressively treated to blood pressure levels of less than 130mm Hg systolic and 80mm Hg diastolic.[31] If any degree of proteinuria is present, the diastolic blood pressure should be lower than 75mm Hg. This research also indicated that any individual with DM should have a diastolic blood pressure lower than 75mm Hg if there is any evidence of retinopathy.[31]

• ***Kidney disease.*** Because DR is also associated with renal disease (proteinuria/albuminuria), diabetes patients with renal dysfunction should be monitored closely for progressive retinopathy. Also, any patient with rapidly progressing DR should be evaluated for possible nephropathy.[9] Further, most patients with type 1 DM should receive ACE inhibitors to reduce the risk of progression from microalbuminuria to macroalbuminuria.

• ***Elevated cholesterol.*** Dyslipidemia may also play a role in the progression of DR and DME—although there is conflicting evidence. Some studies have linked elevated HDL and total cholesterol levels with a higher incidence of DR.[9] A cross-sectional analysis from the Wisconsin Epidemiology Study of Diabetic Retinopathy trial, however, showed no association between elevated cholesterol levels and the severity of DME in either type 1 or type 2 diabetes patients.[9,33]



3. Proliferative diabetic retinopathy using red-free filter. Note the boat-shaped, pre-retinal hemorrhage (arrow) as well as a fine, web-like pattern of neovascularization at the optic nerve (area within rectangle).

Nonetheless, lipid-lowering medications should be considered for any diabetes patients with high cholesterol.[9] Studies of fenofibrate monotherapy have indicated that the drug may slow DME progression.[34]

• ***Anemia.*** Anemia is a common finding in patients with DM due to the high systemic burden of chronic kidney disease. Reduced hemoglobin levels independently help to identify DM patients who are at an increased risk for microvascular complications (including retinopathy), cardiovascular disease and mortality.[35]

Anemia often accompanies diabetic kidney disease. When glomerular filtration rates are less than 60mL/minute, the most common cause of the anemia is a relative erythropoietin deficiency that reduces hemoglobin levels to less than 11g/dL.[31] An ETDRS analysis found that low hematocrit

Diabetes Affects All Retinal Cell Types[64]

Cell Type	Characteristics
Vascular	Altered tight junctions; endothelial cell and pericyte death.
Glial	Altered contacts with vessels; release inflammatory mediators; impaired glutamate metabolism.
Microglial	Increased number; release inflammatory mediators.
Neuronal	Death of ganglion cells; inner nuclear layer; axonal atrophy.

was a risk factor for the development of high-risk DR. A separate cross-sectional study uncovered an increased risk of retinopathy in patients with hemoglobin levels lower than 12g/dl.[9]

• ***Sleep apnea.*** Obstructive sleep apnea (OSA) is a common disorder that often coexists with DM.[36] The consequences of OSA include cardiovascular morbidity (coronary artery disease/myocardial ischemia), cerebrovascular accident and overall mortality.[36]

Obesity, a well-known risk factor for type 2 DM, is also a risk factor for OSA. Patients with OSA often have a body mass index greater than 25kg/m^2 and a neck circumference larger than 17 inches in men and 16 inches in women.[36] Several studies also have found a link between OSA and hypertension.[36-39] OSA may aggravate DR secondary to nocturnal hypertension and hypoxemia.

• ***Tobacco use.*** The role of smoking in DR has not been clearly established. Some studies have shown a definitive association, while others have shown no relationship when controlling for additional risk factors, such as age of onset and duration of diabetes and/or associated hypertension.[31] Smoking increases circulating leukocytes and platelet activation. Nicotine in tobacco smoke causes severe retinal vasoconstriction. Smokers often exhibit higher levels of LDL and lower levels of HDL. Because the link between cigarette smoking and cardiovascular disease is well established—especially among patients with DM—it is essential that doctors encourage their patients to stop smoking.[31]

Treatment of DR (Without CSME)

• ***Treatment of NPDR (without CSME).*** NPDR is significant because of its potential to progress to PDR. The current stage of NPDR at the initial diagnosis dictates the patient's follow-up schedule.

Mild NPDR carries a 5% chance of progression to PDR in one year, and a 15% chance of progression to high-risk PDR within five years.[27,40,41] Moderate NPDR has a 12% to 27% chance of progressing to PDR in one year, and a 33% chance of progressing to high-risk PDR within five years.[27,40,41] Finally, severe NPDR has a 52% chance of progressing to PDR in one year, and a 60% chance of progressing to high-risk PDR within five years.[27,40,41]

Both the Diabetes Control and Complications Trial (DCCT) and the United Kingdom Prospective Diabetes Study showed the therapeutic benefit of intensive glycemic control in patients with type 1 and type 2 DM. Improved glucose control significantly reduced the likelihood of vitreous hemorrhage, retinopathy that required laser photocoagulation and renal failure.[10,42]

Management of NPDR centers on stabilizing the condition and arresting the progression to PDR. The DCCT showed that intensive glycemic control involving multiple daily blood sugar measurements, nutritional counseling, and both medical and glycosylated hemoglobin evaluations every three months decreased the risk of the development and progression of retinopathy.[10]

The ADA recommends an interdisciplinary approach to management of DM and its complications, with close monitoring of blood pressure, blood glucose and cholesterol, as well as smoking avoidance/cessation, exercise and weight control.[1,43] Further, proper instruction from a certified diabetes educator regarding self-management techniques is a mainstay of any interdisciplinary treatment approach.

• ***Treatment of PDR (without CSME).*** In cases of PDR, retinal imaging with fluorescein angiography is needed to determine if/when the patient has reached any treatment landmarks, as well as to document any leakage patterns.[17,29] Treatment of PDR usually involves laser surgery to seal leaking vessels (indirectly) and prevent further development of neovascularization. New, weaker blood vessels can rupture, scar and cause retinal tissue necrosis.

The Diabetic Retinopathy Study (DRS) and the Early Treatment of Diabetic Retinopathy Study (ETDRS) provided evidence that laser photocoagulation significantly reduced the risk of severe vision loss in patients with DR. Results of the DRS indicated that panretinal photocoagulation reduced the risk of severe vision loss in most (60%) patients with PDR.[44] Earlier and more adequate treatment is effective in more than 90% of cases.[11,45-47]

The inherent retinal tissue scarring associated with thermal laser photocoagulation may cause reduced contrast sensitivity, poor

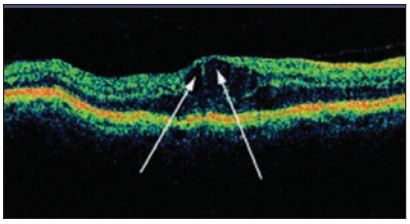
dark adaptation and visual field loss. Intravitreal injections of anti-VEGF agents have been shown to be effective as individual and/or combination treatments for PDR. Several studies are still being conducted to assess the safety of repeated intravitreal injections, which frequently are needed to achieve optimum benefit.[48]

Because thickened posterior vitreous cortex is one of the main factors in the development of disease proliferation in patients with PDR, a consequent shrinkage of the posterior vitreous cortex often leads to hemorrhages and tractional retinal detachments. In this instance, the new vessels use the posterior vitreous face as a scaffold. Therefore, some clinicians believe that PDR should be called "proliferative diabetic vitreoretinopathy."[49]

In some cases of PDR, vitrectomy surgery can be beneficial. Indications for vitrectomy include vitreous hemorrhage that blocks the view of the retina, dense premacular hemorrhage, complicated retinal detachment and severe neovascular proliferation that does not respond to laser treatment. The Diabetic Retinopathy Vitrectomy Study results showed that early vitrectomy was beneficial in restoring and preserving vision in patients with PDR with or without associated vitreous hemorrhage.[49]

So-called pharmacologic vitreolysis has been suggested as another important consideration for future management. Diabetes induces significant biochemical and structural changes within the vitreous. Because a diabetic vitreous is different from a normal vitreous, pharmacologic vitreolysis of a normal vitreous may fail to uncover an agent that is effective for pathologic conditions.

This may explain why Vitrase (hyaluronidase, Bausch + Lomb) failed in phase III FDA clinical trials for treatment of vitreous hemorrhage in patients with DR. Hyaluronidase is a vitreous liquefactant, not a vitreous interfactant. Thus, the agent will liquefy the gel vitreous, but will not induce vitreoretinal dehiscence. In PDR, this results in persistent traction on the neovascularization, which may lead to possible recurrent vitreous hemorrhage and vision loss.



4. Optical coherence tomography scan of a patient with diabetic macular edema. Notice the dark, thickened areas that are indicative of intraretinal fluid accumulation (arrows).

It is worth noting that Ocriplasmin (microplasmin, ThromboGenics) likely will be the first drug approved for clinical pharmacologic vitreolysis in cases of symptomatic vitreomacular adhesion. Biologically, Ocriplasmin serves as both a liquefactant and an interfactant.[50]

Treatment of DR (With CSME)

Although PDR is more likely to cause severe vision loss (20/200 or worse) than NPDR, the most common cause of functional visual loss (worse than 20/40) in patients with DR is DME (specifically CSME). With more than 25 million people estimated to have DM in the United States, the morbidity of CSME has a significant impact on public health.[33] So, CSME needs to be treated early—before chronic disease leads to irreversible functional vision loss.

• ***Conventional laser treatment.*** Laser photocoagulation is the standard of care in the treatment of CSME. Focal or grid laser photocoagulation reduces macular edema by inducing coagulation necrosis. Focal laser treatment is intended to close leaky microaneurysms, while grid laser is used to treat more diffuse edema.[46]

The goal of laser treatment for CSME is not to improve vision, but to slow or prevent central visual loss as a result of chronic edema and secondary tissue damage. The ETDRS indicated that focal or grid laser photocoagulation reduced the risk of moderate visual loss due to CSME by 50%.[51]

As previously mentioned, the anatomical and visual benefits of laser photocoagulation have been shown to be effective over the long term; however, the treatment of DME is associated with risks and side effects caused by iatrogenic damage of retinal tissue.

• ***Micropulse laser treatment.*** Micropulse laser technology (MPLT) has been shown to be as effective as conventional argon laser for DME.[52] Micropulse technology with 810nm and 577nm lasers is used to produce a therapeutic effect without inducing collateral retinal damage during or after treatment.[53] With MPLT, the induced temperature increase in the targeted tissue remains sublethal, and no visible lesion is produced (subvisible-threshold). Micropulse power as low as 25% of the visible threshold intensity has been shown to be have a therapeutic effect, while sparing neurosensory retinal tissue.[53]

MPLT is less destructive to tissue, yet achieves the desired therapeutic effect. For instance, one study showed that MPLT appears to be as effective as laser photocoagulation for the treatment of DME, but causes far less damage to the retinal pigment epithelium.[54]

• ***Pharmaceutical options.***

The Optometrist's Role in DM and DR Management

- **Prevention**—Eduate patients about proper nutrition and healthy lifestyle.
- **Evaluation**—Perform a comprehensive ophthalmic workup and annual dilated fundus evaluation.
- **Early detection**—Conduct regular monitoring of reported ocular complications.
- **Comanagement**—Provide timely consultation and appropriate referral.
- **Rehabilitation**—Offer or arrange low vision care for individuals who experience significant vision loss.

Although laser therapy may slow visual loss, it is not often accompanied by visual gain. This led to evaluation of other CSME management options, including potential pharmacologic therapies that may be used alone or in combination with laser therapy.

The anti-inflammatory effect of intravitreal corticosteroids contributes to a reduction of edema.[55] More specifically, intravitreal triamcinolone has been shown to reduce macular edema and improve visual acuity.[56,57] However, the short-term effect of steroids necessitates multiple treatments, which may increase the risk of adverse effects, including ocular hypertension, glaucoma, cataract, retinal detachment, epimacular membrane and endophthalmitis.

Sustained-release devices containing the corticosteroids dexamethasone and fluocinolone may provide long-term therapeutic benefits, and are undergoing clinical trials. However, such devices also may increase the patient's risk for the aforementioned adverse effects.

A large, multicenter study that compared intravitreal triamcinolone with laser photocoagulation was designed to help elucidate the therapeutic role of steroids in CSME management.[58] The researchers determined that, over a two-year period, focal/grid photocoagulation was more effective and had fewer side effects than 1mg or 4mg doses of preservative-free intravitreal triamcinolone. These results also suggest that focal/grid photocoagulation should remain the benchmark against which other CSME treatments are compared in future clinical studies.[58]

Recently, several studies have evaluated the therapeutic effect of anti-VEGF agents on CSME.[59,60] It appears that pan-VEGF-A inhibitors (which block uptake of all VEGF-A isoforms) exhibit better bioactivity than selective VEGF-A inhibitors in patients with CSME.[48]

Eylea (aflibercept, Regeneron Pharmaceuticals) is a recombinant fusion protein that inhibits the function of both VEGF-1 and VEGF-2 receptors. The DA VINCI study showed that aflibercept, compared to macular laser photocoagulation, produced a statistically significant and clinically relevant visual acuity improvement in patients with DME.[60]

DRCR.net conducted a randomized controlled trial to assess whether an intravitreal injection of Lucentis (ranibizumab, Genentech/Roche) combined with either prompt or deferred laser or intravitreal triamcinolone acetonide combined with prompt laser could improve visual acuity outcomes for patients with DME when compared to focal/grid photocoagulation.[61] This Phase III study clearly showed that intravitreal Lucentis, with either prompt or deferred laser, provided superior anatomic and functional outcomes in individuals with DME through two years of follow-up compared to laser therapy alone.[61]

- ***Surgical intervention.*** Vitrectomy also may aid in the resolution of DME. The initial rationale for use of vitrectomy was justified by evidence from early epidemiologic studies. Researchers observed a lower incidence of complete posterior vitreous detachment in patients with DME than in those without evidence of edema.[62] This finding suggested that a partially attached vitreous is a risk factor for DME, and that vitrectomy will remove the tractional forces at the retinal surface, reduce oxygen consumption of the vitreous and reduce hypoxia at the retina.[62]

Treatment Safety

On the basis of currently available data, we don't know yet if prolonged ocular treatment with anti-VEGF agents will yield increased systemic side effects, such as hypertension and cardiovascular or thromboembolic events. The probable need for repeated injections also elevates the risk of visually devastating ocular side effects, such as endophthalmitis.[63]

Combination therapy comprised of laser and pharmacologic agents potentially can yield additional benefits, including improved visual outcome and less frequent re-treatments—which, in turn, can reduce the risk of adverse events.

The longer a patient has been living with diabetes, the more likely it is that he or she will develop DR. Diabetic retinopathy is a significant public health problem, especially among blacks and Hispanics. Fortunately, however, DR is a treatable condition.

Traditional treatment of DR still is an effective management approach. But, with newer, more effective options, such as MPLT and pharmacotherapy, the out-

come profile for affected patients is changing for the better. ■

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1. During the last several decades, the prevalence and incidence of type 2 diabetes mellitus (DM) in America:
a. Has decreased in response to the development of new medications.
b. Has decreased due to improved disease awareness.
c. Has stabilized due to improved diagnostic testing.
d. Has increased because of higher overall levels of obesity.

2. Patients with diabetic retinopathy (DR) are at an increased risk for developing:
a. Tractional retinal detachment.
b. Diabetic macular edema (DME).
c. Neovascular glaucoma.
d. All of the above.

3. What is NOT a common ocular symptom associated with DM?
a. Blurred vision.
b. Excess tear production.
c. Diplopia.
d. Ocular surface dryness.

4. According to the American Diabetes Association, how much more likely are DM patients to develop cataracts than normal patients?
a. 40%.
b. 50%.
c. 60%.
d. 70%.

5. The Centers for Disease Control and Prevention estimates that DR causes up to how many new cases of blindness each year?
a. 7,000.
b. 10,000.
c. 16,000.
d. 24,000.

6. Typically, what is the earliest clinically detectable sign of DR?
a. Cataract formation.
b. Neovascularization of the optic disc.
c. Intraocular pressure increase.
d. Microaneurysms.

7. What is the estimated incidence of DME in adults who have had DM for at least 20 years?
a. 10%.
b. 20%.
c. 30%.
d. 50%.

8. What is the approximate incidence of DME in patients with proliferative diabetic retinopathy (PDR)?
a. 23%.
b. 38%.
c. 66%.
d. 71%.

9. Which pharmacologic treatment for DM recently has been linked to an increased risk of DME?
a. Metformin.
b. Rosiglitazone.
c. Exenatide.
d. Glyburide.

10. What is the hallmark clinical feature of PDR?
a. Cotton-wool spots.
b. Neovascularization.
c. Flame-shaped hemorrhages.
d. None of the above.

11. Which clinical finding is NOT indicative of PDR?
a. Evidence of venous dilation.
b. Neovascularization of the optic nerve head.
c. Neovascularization elsewhere.
d. Vitreous hemorrhage.

12. Which diagnostic technology is increasingly becoming essential in confirming a diagnosis of clinically significant macular edema (CSME)?
a. Fluorescein angiography.
b. Optical coherence tomography.
c. Preferential hyperacuity perimetry.
d. B-scan ultrasonography.

13. DM patients with concomitant hypertension are _____ more likely to develop DME than those without hypertension.
a. Two times.
b. Three times.
c. Five times.
d. Eight times.

14. According to the Joint British Diabetes Societies, patients with either type 1 or type 2 DM should maintain blood pressure levels that are lower than:
a. 150mm Hg/85mm Hg.
b. 140mm Hg/80mm Hg.
c. 130mm Hg/80mm Hg.
d. 120mm Hg/70mm Hg.

15. What is a common risk factor for obstructive sleep apnea?
a. Female gender.
b. Younger age.
c. Obesity.
d. Hispanic ethnicity.

16. What is the most effective management strategy for patients with non-proliferative diabetic retinopathy?
a. Anti-VEGF injection.
b. Intravitreal corticosteroid injection.
c. Nutritional counseling and tight glucose control.
d. Vitrectomy.

17. What is the primary limitation of conventional laser photocoagulation?
a. Increased risk of retinal detachment.
b. Retinal tissue damage.
c. Permanent intraocular pressure increase.
d. All of the above.

18. Historically, how was macular edema typically managed?
a. Focal or grid laser.
b. Vitrectomy.

OSC QUIZ

c. Intravitreal Lucentis (ranibizumab, Genentech/Roche).
d. Intravitreal Kenalog (triamcinolone, Bristol-Myers Squibb).

19. What is the principal treatment goal of laser photocoagulation for CSME?
a. Restore lost visual acuity.
b. Improve contrast sensitivity and dark adaptation.
c. Eliminate the risk of retinal detachment.
d. Prevent further central vision loss.

20. Patients who receive intravitreal corticosteroid injections are at an increased risk for:
a. Ocular hypertension.
b. Cataract development.
c. Retinal detachment.
d. All of the above.

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4.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
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7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
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17.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
18.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
19.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
20.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

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RO-OSC-0812

Which Way to Go for Wet AMD?

Lucentis, Avastin or Eylea—what's the difference? And why does every retina specialist seem to take a different approach? **Edited by Paul C. Ajamian, O.D.**

Q I frequently send patients for treatment of wet AMD. Some get Lucentis, some Avastin, some Eyelea—what's the difference, and why does every retina specialist seem to have a different protocol?

A "Most distinctions in practice patterns involving these agents have to do with patient affordability, practice overhead and patient demographics, as well as the indicated conditions," says Mohammad Rafieetary, O.D., of the Charles Retina Institute in Memphis.

Lucentis (ranibizumab, Genentech/Roche), Avastin (bevacizumab, Genentech/Roche) and Eylea (aflibercept, Regeneron) are all anti-vascular endothelial growth factor (VEGF) agents that have shown efficacy in the treatment of CNV associated with wet AMD, he says.

So how are they different? Pharmacologically, Lucentis has a greater affinity for VEGF (in vitro) than Avastin, but Avastin improves visual acuity as well as Lucentis in clinical studies. Eylea is longer lasting than both Lucentis and Avastin, and is possibly more efficacious because it binds to both VEGF-A and placental growth factor (PGIF). "These factors, plus the physiologic differences between diseases, play a significant role in the selection of a specific agent," he says.

Retina specialty practices that treat these conditions must have Avastin available for off-label use, because the other agents have limited FDA approval and therefore restricted third-party coverage, he says. "Practically speaking, availability of the drastically cheaper Avastin (as low as $50) allows a segment of patients to be treated who otherwise might not have access. This consideration, combined with the apparent equivalence of Avastin and Lucentis in clinical efficacy, have led some vitreoretinal practitioners to use only Avastin due to the significant overhead cost incurred by Lucentis (about $2,000 per injection)," Dr. Rafieetary explains. "However, most have Lucentis and Eylea available for approved diseases, and use Avastin for all others."

The recent Comparison of Age-Related Macular Degeneration Treatment Trials (CATT) provided insight on the similarities and differences between Avastin and Lucentis.[1] After one year of treatment, vision scores were equivalent for the monthly injection groups between Lucentis and Avastin, but the Avastin group revealed more patients with subretinal fluid still present on time-domain OCT, as well as more patients with leakage on angiography. The two-year results demonstrated similar findings, but used spectral-domain OCT, and noted a higher incidence of geographic atrophy in patients undergoing Lucentis therapy who did not have this finding at study initiation.

"These results suggest that while both Lucentis and Avastin are efficacious, Lucentis is associated with improved anatomical findings as revealed by OCT and FA," Dr. Rafieetary says. "Only long-term experience will demonstrate if there is any true difference in efficacy between these two medications, although the cautious observer would likely argue that both are reasonable options for AMD, and both offer significant improvements over previous treatments."

In November 2011, the FDA approved Eylea for wet AMD. Its chief advantage is its reduced number of doses: It is dosed monthly for the first three months, then—unlike Lucentis or Avastin—every other month thereafter. Each dose of Eylea costs about $1,850.

"Anecdotally, our practice, which uses a morphology-based approach to treat AMD, has found that many patients are able to extend the time between injections with Eylea to six weeks," Dr. Rafieetary says. "Another observation is that pigment epithelial detachments often flatten quicker with Eylea, giving the false appearance of increased subretinal fluid after the first administration. Only time will tell if Eylea is truly a superior or equivalent drug to Lucentis and Avastin, and if it is as clinically effective for decreasing the treatment burden of intravitreal injections."

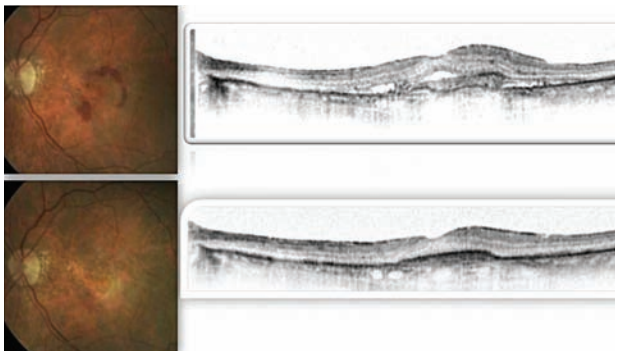
Overall, Lucentis, Avastin and Eylea are all good choices for AMD, he says. There is no clear difference in visual acuity outcomes to date between these three medications.

Q What is my role in comanaging these patients?

"The optometrist plays a critical role in the management of AMD patients," Dr. Rafieetary says. In patients with atrophic disease, counseling, education and periodic examination are essential, he explains. Patients with more severe cases require early detection of conversion from dry to wet AMD, along with prompt referrals to retina specialists.

"As with any referral, be sure to at least suggest a diagnosis prior to referral. Make the appointment for the patient, and document that you did so clearly in the chart," he says.

In the early stage of treatment, when most patients require monthly injections by the retina specialist, there may be a gap in patients' visits to their referring optometrist. "In remote and rural areas, patients may have difficulty returning to the specialist for every follow-up exam," Dr. Rafieetary says. "So, AMD patients are often comanaged in alternating visits between the specialist and the local optometrist. In this scenario, OCT examinations can detect subtle areas of increased fluid, which is currently the most common re-treatment criterion. When applicable, images can be electronically shared between the providers, which can speed communication."



Fundus photo and OCT scan of AMD with CNV and subretinal hemorrhage (top), which is significantly improved (bottom) following a single intravitreal anti-VEGF injection.

The doctor of optometry is also an essential resource in cases where the patient might have co-existing conditions and/or unusual or unexpected symptoms that might require immediate care. Finally, he adds, refractive exams—and particularly low vision aids—remain an integral aspect of care for many AMD patients. ■

1. Comparison of Age-related Macular Degeneration Treatments Trials (CATT) Research Group. Martin DF, Maguire MG, Fine SL, et al. Ranibizumab and bevacizumab for treatment of neovascular age-related macular degeneration: Two-year results. Ophthalmology. 2012 Jul;119(7):1388-98.

Rosacea in Full Bloom

Due to ocular rosacea's chronic nature, it's a lifelong battle for most patients to maintain corneal health. **Edited by Joseph P. Shovlin, O.D.**

Q **Recently, I saw a patient with severe rosacea-related corneal opacity that is essentially 360° and, at certain areas, is approaching the visual axis. Some marginal thinning is evident, with a marked blepharoconjunctivitis and corneal findings that are apparently refractory (she has seen several doctors). She has tried oral doxycycline and topical steroids, but still has problems. Can you recommend any treatment options?**

A Rosacea is a chronic condition with periods of remission and exacerbations that requires long-term therapy to maintain control. "It is important when dealing with rosacea patients to remember that you can treat the symptoms effectively, but you cannot cure the patient," says Lloyd Pate, O.D., a clinical associate professor at the University of Houston College of Optometry.

First, examine the patient's medical records and ask her if any of the prior therapies were successful in controlling her condition while she was using them. Perhaps she only experienced a flare-up after cessation of the therapy.

"It's not a uniform disease—everybody has a different tolerance to the actual insult, and everybody has a different response to the drugs," says J. James Thimons, O.D., center director for Ophthalmic Consultants of Connecticut. Each patient will require an individualized treatment plan, which may integrate multiple treatments. Current treatment options include:

• ***Low-dose oral doxycycline.*** Oracea (Galderma) q.d. is available as a once-daily 40mg capsule, or you can use Periostat (CollaGenex) b.i.d. in a 20mg tablet, also available generically. "Keep the dosage below the therapeutic threshold because the therapy relies on doxycycline's anti-inflammatory properties, not its antibiotic activity," Dr. Pate says.

The drug typically is used in a pulsed fashion—with one month on treatment and one month off.

• ***Azithromycin ointment.*** Many O.D.s also pulse-dose Azasite (Merck), with one month on and 30 to 45 days off. "The on and off pulsing doesn't seem to create bacterial resistance," Dr. Thimons says. "It also gets to the root of the problem—poor meibomium gland function."

• ***Topical cyclosporine.*** A clinical trial of 37 patients with rosacea-associated eyelid and corneal changes found that those who used Restasis (Allergan) for three months showed an increase in Schirmer scores, improved mean tear film break-up time and a greater mean reduction in corneal staining scores.[1] Another study showed topical cyclosporine is effective in treating ocular rosacea that is unresponsive to standard therapy.[2] The majority of responsive patients (71%) were able to discontinue oral medications, but most required long-term maintenance with topical cyclosporine.[2]

• ***Topical metronidazole.*** Studies have suggested that metronidazole 1% gel or 0.75% cream q.d. or b.i.d. also is effective in treating rosacea blepharitis.[3]

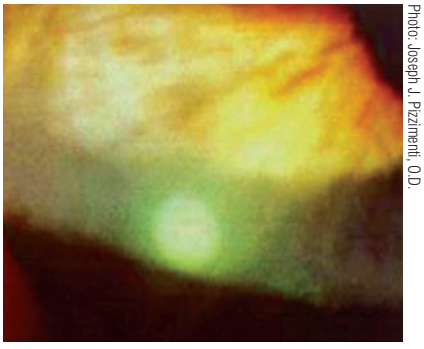


Photo: Joseph J. Pizzimenti, O.D.

This marginal infiltrate is an example of another clinical entity you may encounter with rosacea patients.

• ***Tea tree oil.*** "In-office tea tree oil treatment followed by twice-daily lid scrubs with tea tree oil shampoo is a good therapy to consider," Dr. Pate says.

• ***Intense pulsed light (IPL) therapy.*** IPL is a newer therapy that has been showing promise in ocular rosacea. Currently, Dr. Thimons is working on a clinical trial in which patients receive IPL treatments once a month for six months, with the goal of improving meibomium gland function. "So far the data looks really good," he says. "I'd be happy if I had a treatment like this in addition to topical therapy." ■

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New Diet Drug Also 'Slims' Eyes

A new weight control drug will soon hit the market. But, will patients with ocular side effects soon hit your office? **By James L. Fanelli, O.D.**

In February 2012, a 49-year-old white female presented with complaints of blurred vision in both eyes for the past six days and a dull, achy feeling "behind" both eyes for the previous 48 hours. Her primary care physician referred her to my office after having seen her one day earlier.

History

The patient's current medications included Abilify (aripiprazole, Bristol-Myers Squibb/Otsuka America) q.d., Topamax (topiramate, Janssen Pharmaceuticals) q.d. and Geodon (ziprasidone HCl, Pfizer) b.i.d.

She had recently discontinued Depakote (divalproex sodium, Abbott) and Seroquel XR (quetiapine fumarate, AstraZeneca) approximately six weeks earlier. She explained that her family physician was in the process of adjusting her medications to find the combination that would give her the best therapeutic relief of her depression and bipolar disorder with the fewest side effects. She subjectively reported that she seemed to be less manic, but was bothered by somnolence and "not thinking clearly."

Her vision decrease had been constant for the past six days, she said. But the pressure sensation had worsened, prompting her visit to the primary care doctor.

Diagnostic Data

Entering corrected visual acuity was 20/100 O.D. and 20/80 O.S. Pinhole improved acuity to 20/25- O.D. and 20/25 O.S. Best-corrected visual acuity was 20/20- O.D. and 20/20- O.S. through myopic astigmatic correction. Her manifest refraction was approximately -1.50D more myopic than her entering Rx, which she reported was only one year old.

Pupils were round and reactive to light, with physiological anisocoria—the right pupil measured 6mm and the left pupil measured 5mm in ambient light. The anisocoria did not change in bright or dim illumination. Confrontation visual fields were full as were extraocular motilities, in all positions of gaze.

Slit lamp examination was unremarkable, with bilateral clear corneas, deep and quiet anterior chambers, and grade 3 open angles as estimated by Van Herick method. Iris anatomy was normal. Intraocular pressure measured 19mm Hg O.D. and 18mm Hg O.S. at 10:35 a.m.

Upon dilation, both crystalline lenses appeared clear. Stereoscopic examination of the optic nerves revealed moderately tilted discs with temporal peripapillary atrophy. Cup-to-disc ratios were 0.40 x 0.65 O.D. and 0.30 x 0.60 O.S. Close examination of the neuroretinal rims demonstrated healthy optic nerves with no areas of rim tissue suspicious for damage. Retinal vasculature was unremarkable. Both maculae exhibited good foveal reflexes and appeared healthy. Peripheral retinal exams were remarkable for microcystoid degeneration 360° O.U. as well as scattered areas of lattice degeneration without hole formation.

Diagnosis and Management

Given the absence of overt disease, I suspected that the blurred vision and achy sensation around the eyes were associated with her myopic shift. Prior to the patient's arrival at our office, and as part of the referral from the primary care medical provider, I received a copy of that doctor's EMR notes from her last three visits, confirming the changes to her medications as outlined in her history. They also included recent lab work from approximately six weeks earlier that reported her fasting glucose level and HbA1c, both of which were entirely normal.

Given normal glycemic indices, my working diagnosis at this point: Her myopic shift was associated with having started Topamax. My level of suspicion for other possible causes—those associated with neurological field loss or occult orbital disease—was relatively low. After discussing my suspicions with her primary care doctor, we discontinued the Topamax and asked her to return to the office for threshold field studies, exophthalmometry and follow-up examination.

When she returned for follow-up two weeks later, she reported almost complete resolution of the blurred vision and periocular ache after about four to five days of stopping the Topamax. At this visit, refraction of -5.75-1.00x175 yielded 20/20 O.D., and -6.50

-1.50x008 yielded 20/20 O.S. This refraction was within -0.50D of her habitual spectacle Rx. Pupils were unchanged from the initial visit.

Threshold visual fields were normal in both eyes with no evidence of neurological field loss. IOP measured 16mm Hg O.U. at 2:55 p.m. Slit lamp exam was unremarkable, with grade 4 wide-open angles. Gonioscopy confirmed 360° views of the ciliary body, no plateau iris configuration, a flat insertion of the iris and normal trabecular pigmentation in both eyes. Her posterior pole evaluations were unchanged from baseline exam.

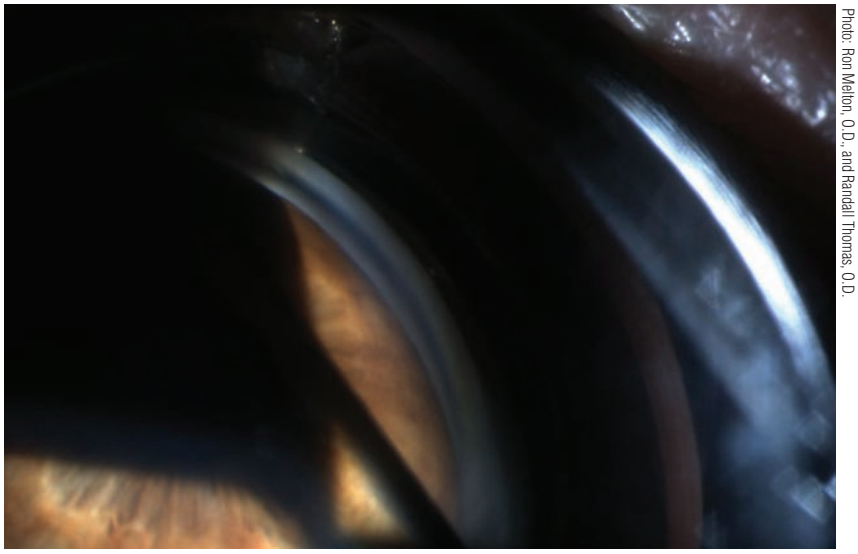

Photo: Ron Melton, O.D., and Randall Thomas, O.D.

Get ready to see more of this. A newly-approved drug for obesity includes topiramate, which puts patients at risk for acute-angle closure (shown here) and myopic shift.

Discussion

Topamax is typically prescribed to treat epileptic seizures and convulsions, but is also used to treat migraines, bipolar disorder and other psychiatric conditions, such as depression, mania and obsessive-compulsive disorder.

As with any medication, you must be aware of side effects—and Topamax has two that are particular to the eye that manifest clinically: a sudden increase in myopia and a shallowing of the anterior chamber, sometimes to the point of inducing and acute angle-closure crisis. This is often bilateral in cases of topiramate use. These ophthalmic problems are well documented, and even prompted the FDA to require the manufacturer to issue a "Dear Doctor" letter about them.[1]

Shallowing of the anterior chamber is believed to occur through anterior rotation of the ciliary body (and subsequent anterior movement of the lens), resulting in an increase in myopia.[2] The believed mechanism of angle closure is not related to the development of pupillary block, and may also include the development of shallow anterior choroidal detachments.[3] These changes typically occur within the first few weeks of initiating topiramate, and are usually reversible upon ceasing it. However, should the anterior chamber angle close, prompt treatment is required.

One of the other side effects of topiramate is a pronounced decrease in appetite, and the medication has been used quite regularly off label to facilitate weight loss. Its exact mechanism in weight loss is not known, but may involve the hypothalamic pituitary and endocrine pathways related to insulin sensitivity.[4]

In July, the FDA approved a new medication for the treatment of obesity called Qsymia (Vivus) that combines topiramate with phentermine, an adrenergic agent long used for the short-term treatment of obesity. Its side effects include increased heart rate, dry mouth and dry eyes.[5] Recent data indicate that the two medications do not have interactions with each other, but that their side effect profile merely is the sum of the adverse effects caused by the individual components.[6]

Given the epidemic of obesity in the United States, we will all be seeing patients who are medicated with this new drug. So, it's important for us to be aware that both of the ingredients in this drug have ocular side effects—especially topiramate. While the ocular effects of phentermine may be annoying (dry eyes), the potential effects of topiramate—myopic shift and angle shallowing/closure—are more serious, and require immediate attention in the form of medication cessation. ■

1. FDA website. MedWatch: The FDA Safety Information and Adverse Event Reporting Program. Topamax (topiramate). Sep 2001. Available at: www.fda.gov/Safety/MedWatch/SafetyInformation/SafetyAlertsforHumanMedicalProducts/ucm172669.htm.
2. Craig JE, Ong TJ, Louis DL, et al. Mechanism of topiramate-induced acute-onset myopia and angle closure glaucoma. Am J Ophthalmol. 2004 Jan;137(1):193-5
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6. Bays HE, Gadde KM. Phentermine/topiramate for weight reduction and treatment of adverse metabolic consequences in obesity. Drugs Today (Barc). 2011 Dec;47(12):903-14.

Just a Simple Refraction?

Our patient presented for an updated Rx, but we uncovered a more serious problem.

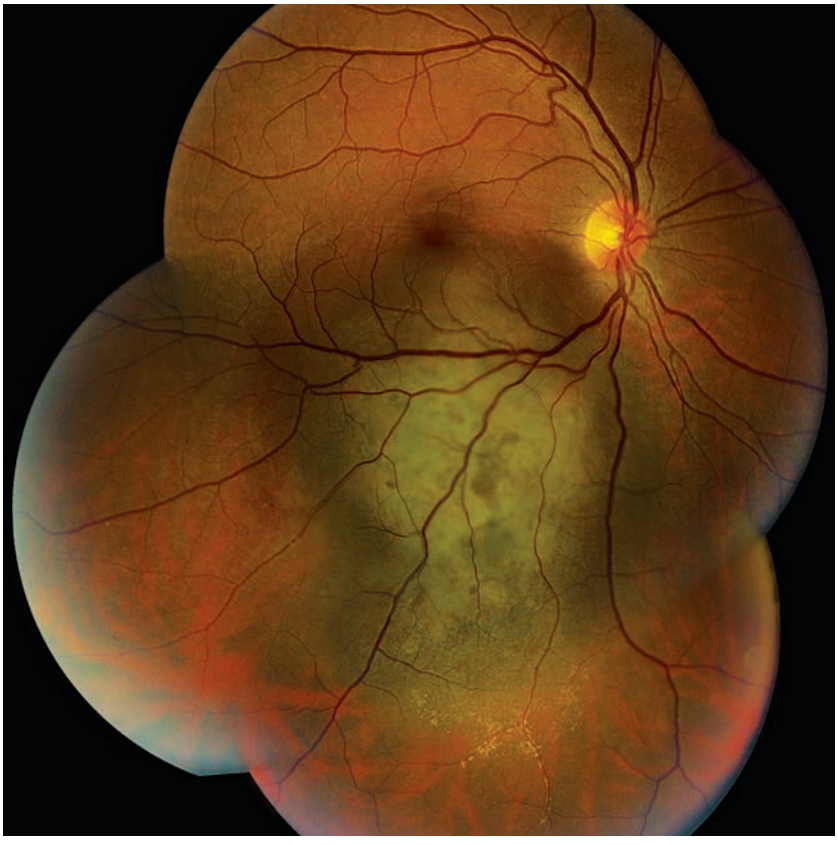
By Mark T. Dunbar, O.D.

A 39-year-old Middle Eastern male presented to the eye clinic for an updated refraction. He reported decreased vision while wearing his glasses or contact lenses (O.D. > O.S.), which he attributed to a change in refractive error.

He was in excellent health and took no medications. His ocular history was significant only for myopia, which—until now—was successfully corrected with glasses and contact lenses.

On examination, his best-corrected visual acuity measured 20/30 O.D. and 20/20 O.S. Extraocular motility testing was normal. Confrontation visual fields were full to careful finger counting O.U. His pupils were equally round and strongly reactive, with no afferent defect. The anterior segment examination was unremarkable.

Dilated fundus exam of the right eye showed small cups with good rim coloration and perfusion. However, we noted clinically significant changes (*figure 1*). Fundoscopic examination of the left eye was completely normal. Additionally, we performed a spectral-domain optical coherence tomography (SD-OCT) scan (*figure 2*) and a standardized echographic ultrasound (*figures 3 and 4*).



1. A fundus photograph of our patient's right eye. Note the lesion located inferior to the macula, as well as the changes observed directly in the macula.

Take the Retina Quiz

1. What does the SD-OCT image of the right macula show?
   a. Cystoid macular edema (CME).
   b. Neurosensory retinal detachment.
   c. Retinal pigment epithelium (RPE) detachment.
   d. Choroidal mass pushing up the macula.

2. Based on the ultrasound, what is the lesion's reflectivity?
   a. Low.
   b. Low to medium.
   c. Medium.
   d. High.

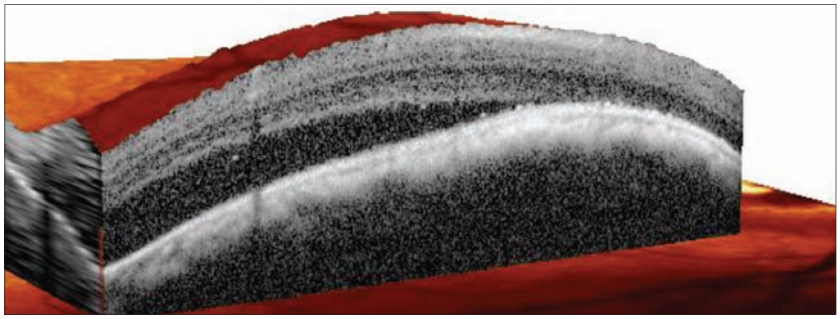
3. What is the most likely diagnosis for this patient?
   a. Choroidal nevus.
   b. Choroidal melanoma.
   c. Choroidal hemangioma.
   d. Choroidal metastasis.

4. How should this patient be managed?
   a. Observation.
   b. Enucleation.

c. Plaque radiotherapy.
d. Avastin (bevacizumab, Genentech/Roche) injection.

5. What is the overall prognosis for this patient?
a. Very poor.
b. Moderate.
c. Excellent.
d. Unknown.

For answers, go to page 130.



2. The SD-OCT scan of the right macula revealed unique changes.

Discussion

Clearly, our patient's problem was more significant than an underpowered refractive correction. Upon examination, we detected an elevated pigment mass located along the inferotemporal arcade that extended peripherally. Additionally, the SD-OCT scan indicated the presence of a neurosensory retinal detachment that involved his macula. Judging by the clinical evidence, the lesion appeared to be a choroidal melanoma. On ultrasound testing, the lesion measured 16mm x 12.5mm x 4.1mm. It exhibited low to medium internal reflectivity, consistent with a choroidal melanoma.

With a definitive diagnosis in mind, a bigger question arose: How should we manage our patient? Do we recommend enucleation or plaque radiotherapy, and would either influence his chance of survival?

Enucleation seemed to be the obvious choice, with the idea that, "If it's cancer, why wouldn't you want it removed?" For this very reason, enucleation has been the preferred uveal melanoma treatment for more than 100 years. But what about the potential use of other "globe-preserving" modalities, such as plaque radiotherapy?

A little more than a decade ago, researchers from the National Eye Institute compared the therapeutic effects of enucleation and radioactive plaque therapy in the Collaborative Ocular Melanoma Study (COMS).

In one arm of COMS, 1,317 patients with medium-sized choroidal melanomas were randomized to receive either enucleation (660 patients) or iodine-125 plaque therapy (657 patients). At five-year follow-up, the survival rate of patients in both groups was equal, at approximately 90%—which is far better than what previous studies of ocular melanoma patients had shown.[1] Given these impressive results, it seems that most patients would opt for iodine-125 plaque therapy vs. outright globe removal. Needless to say, our patient elected iodine-125 plaque therapy within the same week of his examination.

Our patient's procedure was successful. So, he was in the clear, right? Unfortunately, a subsequent report from COMS indicated that just 45% of all uveal melanoma patients were alive and cancer-free after 12 years of treatment.[2] As bleak as that statistic sounds, remember that just because a patient has died, it doesn't necessarily mean that he or she passed away because of complications from metastatic disease. Indeed, many patients in COMS were elderly individuals who likely died of unrelated causes during the 12-year follow-up period.

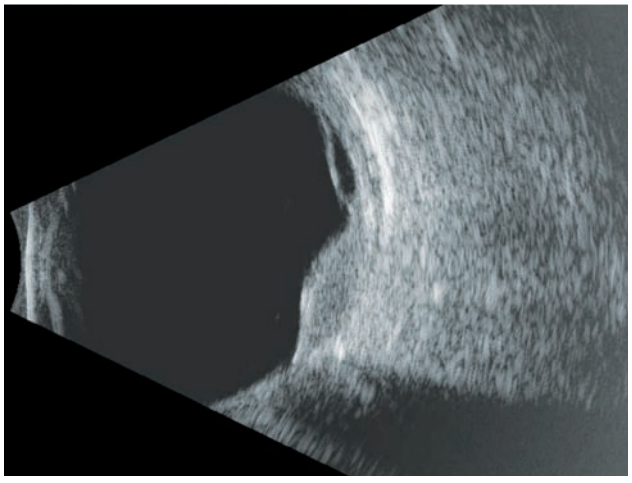
At 39 years of age, our patient probably has a long life ahead. Like all cancer patients, he always will be at risk for metastasis. But, is there a way to better predict if he will continue to live cancer-free? Thanks to several recent advances in molecular genetics: Yes.

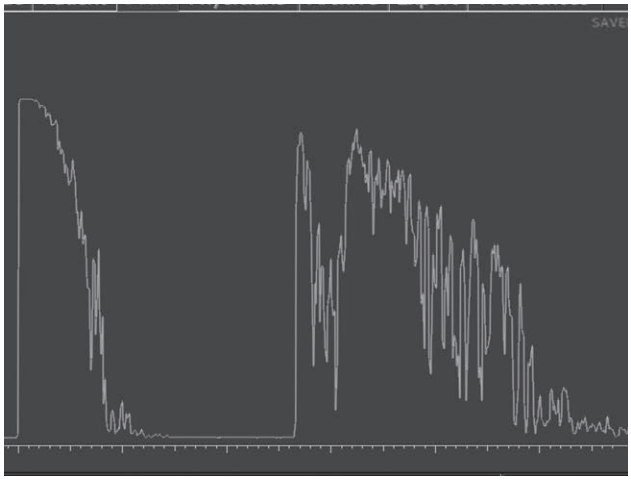
Researchers have uncovered two distinct classes of uveal melanoma based on the tumor's molecular genetics.[3,4] Tumors in Class 1 carry a low risk of metastasis—less than 10%. Class 2 tumors, on the other hand, have a 90% likelihood of spreading to the liver.[3,4]

Molecular genetic testing has shown that aberrations on chromosomes 3, 6 and 8 are common in patients with uveal melanoma.[4] In fact, gene expression profiling is now believed to be a more reliable predictive metric for uveal melanoma metastasis than any other clinical or histologic testing method.

Typically, molecular genetic testing is conducted using a fine-needle aspiration biopsy at the time of initial treatment. In this diagnostic procedure, a surgeon performs the biopsy with a 25-gauge needle that is passed directly into the tumor via a trans-scleral route.

We performed molecular genetic testing on our patient at the time of his iodine-125 plaque therapy.





3, 4. Here's a look at our patient's ultrasound (B-scan left, A-scan right). What do you notice?

Fortunately, the results suggested that he had a Class 1A molecular genetic profile—which exhibits just a 2% tumor-related mortality rate within five years of diagnosis.[4] An ocular oncologist and a cancer specialist will continue to follow the patient closely for any signs of metastasis. ■

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REVIEW OF OPTOMETRY

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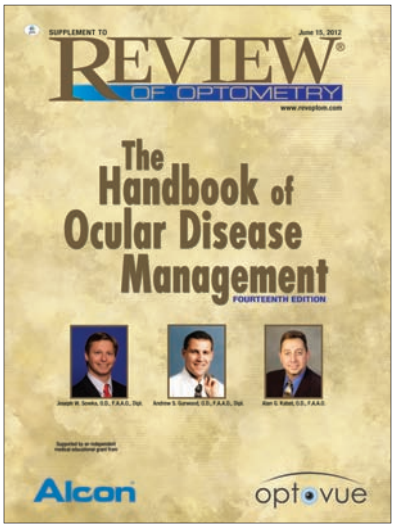

This fourteenth edition, brought to you by Alcon and Optovue, contains updated disease conditions featured in the previous editions of the *Handbook,* as well as numerous new entries.

Subjects Include:

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Fuchs' Dystrophy: A New Hope

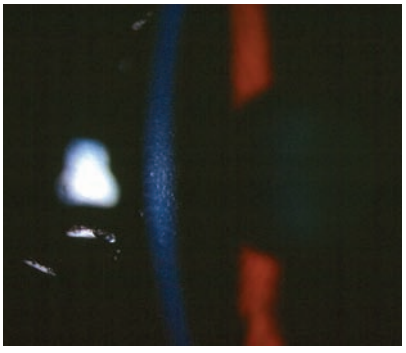
Treatment options for this troubling condition have improved dramatically during the last two decades. **By Alan G. Kabat, O.D., and Joseph W. Sowka, O.D.**

A 75-year-old white male presented to our clinic for a second opinion regarding his visual status. Several weeks prior, he was evaluated by a local optometrist and received a new spectacle prescription. However, he was unhappy with the results. At the time, the patient was told that his vision could not be improved further because of cataracts and "some type of cornea problem."

In our office, his best-corrected visual acuity measured 20/50 O.D. and 20/70 O.S., with a refraction that was nearly identical to the spectacle Rx prescribed by the local optometrist. An ocular health evaluation revealed early nuclear sclerosis and cortical spoking O.U.; however, these changes were mild and inconsistent with his reduced vision. Closer inspection of the corneas revealed a posterior stromal haze and dense, dot-like irregularities at the level of the endothelium. We determined that this was a classic case of Fuchs' corneal dystrophy.

An Overview of Fuchs'

Fuchs' dystrophy—named for Austrian physician Ernst Fuchs, who first described the condition in the early 1900s—is a relatively common disorder in adults that tends to present bilaterally yet asymmetrically. Rarely symptomatic before 50 years of age, patients typically report symptoms of diminished vision, foreign body sensation, and pain or discomfort (particularly upon awakening).[1]



Corneal guttae, as seen in this patient with Fuchs' dystrophy, can be subtle and easily overlooked without careful slit lamp inspection.

The key clinical finding is central corneal guttae, which represent focal thickenings at the level of Descemet's membrane. When viewed in direct illumination, guttae appear as gold-colored, hyperreflective bodies on the posterior corneal surface; under retroillumination they resemble small bubbles or holes in the endothelium. Fine endothelial pigment dusting also commonly is seen in association with guttae. In later disease stages, the clinician may observe stromal edema with folds in Descemet's membrane. And in the most severe presentations, patients may exhibit corneal pannus or bullous keratopathy.

Fuchs' dystrophy is caused by a primary malfunction of the endothelium, likely inherited via an autosomal-dominant mechanism with incomplete penetrance.[1] This leads to widespread loss of endothelial cells and subsequent disruption of the endothelial pump mechanisms that are responsible for maintaining normal stromal hydration.[2] The consequence is an excessive influx of aqueous, which results in corneal stromal edema as well as physiologically and optically compromised tissue.

Back In the Day...

Fuchs' dystrophy was once perceived as a troublesome condition with no genuinely effective treatment—save radical corneal transplantation. It was a troubling diagnosis to make, especially knowing that little could be done to help the patient cope with his or her symptoms. Most individuals sought relief by frequently instilling hypertonic saline drops or ointments throughout the day, and even regularly blasting a hair dryer toward the ocular surface in an attempt to deturgesce the cornea.[3]

Fortunately, the last 20 years have witnessed not only galactic leaps forward in pharmacologic advancements, but also the refinement of surgical interventions. Today, corneal surgeons are employing remarkable procedures to restore functional vision to patients with Fuchs' dystrophy.

Current Treatment Strategies

Conservative therapy for early Fuchs' dystrophy still involves the use of 5% sodium chloride

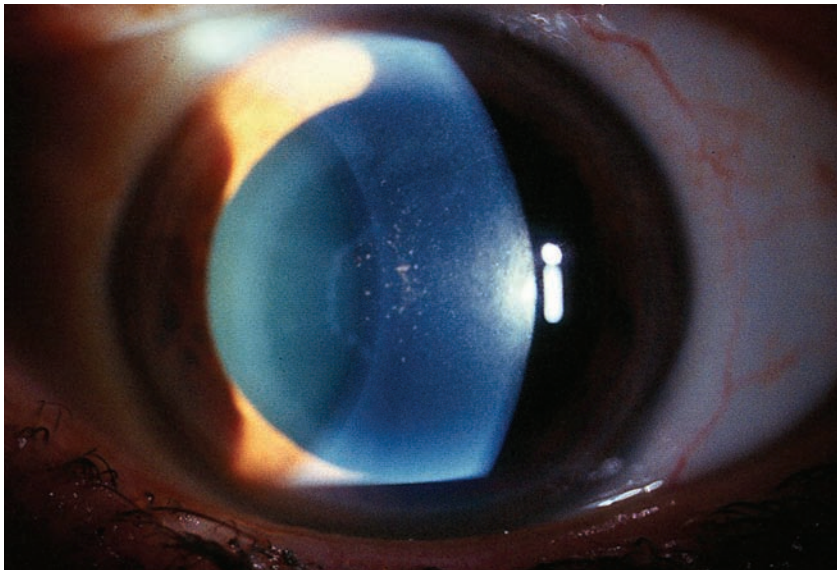
solution throughout the day (e.g., Muro 128 [Bausch + Lomb] every two to six hours) and 5% sodium chloride ointment at bedtime. For more symptomatic cases, NSAIDs such as ketorolac, bromfenac or nepafenac may be helpful in managing patients with painful bullae. It is important to note, however, that NSAIDs merely provide analgesia. Additionally, corneal melts have been associated with excessive and prolonged use of certain NSAIDs, so they should be dosed judiciously.[4]

Bandage soft contact lenses also may serve to alleviate patient discomfort in cases of advanced Fuchs' dystrophy. A flatly fit, high water content lens helps to mask the irregular astigmatism and diminish pain associated with epithelial bullae.[2,5] SiHi lenses also have been used in this capacity with some success.[6]

Prior to 2000, penetrating keratoplasty remained the last recourse for most patients with advanced Fuchs' dystrophy. However, with the advent of deep lamellar keratoplasty, patients now have a surgical option that is less invasive and painful, necessitates a shorter recovery time, and results in fewer instances of rejection.[7]

In deep lamellar keratoplasty, only the posterior aspect of the cornea is removed, which is replaced with donor tissue in an effort to restore a functional endothelial layer. The preoperative corneal surface is preserved, and transplantation of donor tissue is achieved via a scleral tunnel. The donor "button" is then inserted into the anterior chamber and positioned with the aid of an air bubble.

The currently favored surgical technique is known as DSEK, which



This individual with Fuchs' dystrophy exhibited marked endothelial pigment deposition.

stands for Descemet's stripping endothelial keratoplasty. DSEK was first reported in 2005.[8] Unlike prior techniques that dissected the recipient cornea at mid-stroma, this procedure peels away approximately 150µm—or about 25%—of the posterior stroma, including Descemet's membrane and the endothelium (much in the same fashion that a capsulorhexis is performed on the anterior lens capsule during cataract surgery). The donor button of posterior stroma, Descemet's membrane and endothelium are then implanted.

DSEK has the advantage of a smaller, potentially self-sealing incision, as well as a smoother recipient interface for the donor tissue. Additionally, DSEK offers a more rapid rate of visual recovery than penetrating keratoplasty; most individuals achieve a functional level (e.g., 20/40 or better) within one to six months postoperatively.[9]

Although still relatively new and outside the "comfort zone" of many practicing corneal surgeons, DSEK is gaining acceptance both in the U.S. and abroad. A recent publication from the U.K. showed a 36% increase in DSEK procedures performed between 2007 and 2010.[10]

Also, improvements in microsurgical technique and instrumentation have yielded even more dramatic results with regard to patient outcomes. A three-year retrospective study indicated that a best spectacle-corrected visual acuity (BSCVA) of 20/25 was achieved in more than 70% of individuals who underwent DSEK.[11] More impressively, a BSCVA of 20/20 was attained in nearly 50% of DSEK patients.[11]

Going forward, it is expected that lamellar keratoplasty will become even more precise, as DSEK gives way to the newest procedure—DMEK, or Descemet's membrane endothelial keratoplasty. Like DSEK, DMEK involves an in vivo stripping of Descemet's membrane through a scleral incision. However, DMEK peels away only about 80µm—or approximately 15%—of the

Photo courtesy Mozambique Eyecare Project

WORLD SIGHT DAY CHALLENGE

World Sight Day is October 11.

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posterior stroma. DMEK combines the anatomical benefits of DSEK with enhanced visual rehabilitation—typically to 20/40 or better in 90% of cases and 20/25 or better in 60% of cases within the first three months following surgery.[9]

While there is still no magic bullet for Fuchs' dystrophy, the prognosis for surgical intervention and visual recovery is now much brighter than ever before. Patients no longer must fear the arduous and uncertain postoperative period that inevitably follows penetrating keratoplasty. Likewise, optometrists can comfortably and confidently refer patients with Fuchs' dystrophy to corneal surgeons who are actively performing DSEK and DMEK, knowing that they will experience minimal downtime and will be able to resume normal activities in a short period of time.

As for our patient, he underwent uncomplicated DSEK. His visual acuity now measures 20/25 O.U., and he is highly satisfied with the postoperative outcome. ■

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A Tap List of MGD Treatments

Considering MGD is one of the hottest topics in eye care today, we must familiarize ourselves with all available treatments. **By Paul M. Karpecki, O.D., and Diana L. Shechtman, O.D.**

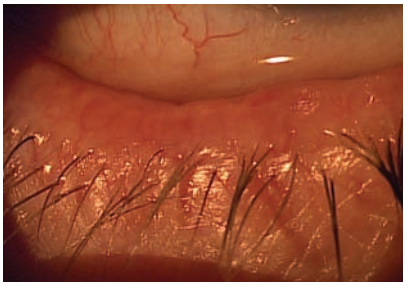
Because ongoing research is only now providing a clear understanding of meibomian gland dysfunction's pathogenesis, many eye care providers remain widely uninformed about the newest and most effective treatment options. Here's an overview of what you need to know.

Liposome Spray

One report from the International Workshop on Meibomian Gland Dysfunction shed light on a rather novel therapy known as liposomal spray.[1] This product delivers phospholipids to the tear film via the surface of closed eyelids. Upon application, the phospholipids penetrate the eyelids and reach the ocular surface to help eliminate symptoms of MGD and evaporative dry eye.

In one study, patients with MGD were randomized to receive saline spray or Tears Again Advanced (Ocusoft) liposome spray.[2] In the Tears Again Advanced group, 46% of patients reported increased comfort and exhibited improved lipid layer grades and tear film break-up times (TFBUTs), compared to 18% in the saline group.[2] Another study indicated that patients who received liposome spray showed better than a two-fold improvement in TFBUT compared to those who received artificial tears.[2,3]

So, is there any downside to the use of liposome spray? In another study of 382 patients, the most commonly reported complaint was a burning sensation upon application.[4] Still, all patients in the study preferred the therapeutic effect of liposome spray vs. saline spray.[4]



What's the most effective treatment option for this patient with MGD?

Warm Compresses

Some impressive research published by Donald R. Korb, O.D., and associates in 2007 examined the impact of heat on the meibomian glands.[5] The researchers suggested that, in order to optimize the therapeutic efficacy of warm compresses, patients should heat them to approximately 45°C, optimize contact between the compresses and the outer eyelid surface, and maintain contact for at least four minutes to achieve an inner lower eyelid temperature greater than or equal to 40°C.

Another landmark study looked at the effect of warm compress therapy on tear film lipid layer thickness (TFLLT) after five, 15 and 30 minutes of continuous contact with the eyelid surface.[6] Previous studies have shown that TFLLT correlates well with patient symptoms, and that individuals with thinner lipid layers (<60nm) tend to have more severe dry eye symptoms.[6]

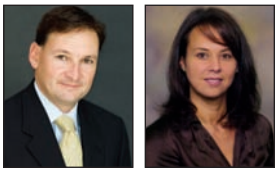
The mean TFLLT at baseline or prior to treatment was 57.8nm +/-12.9nm. After five minutes of treatment with warm compresses, TFLLT increased to 105.8nm +/-23.7nm; 117.8nm +/-26.4nm at 15 minutes; and 121.5nm +/-27.1nm at 30 minutes.[6] The researchers concluded that warm, moist compresses increase TFLLT by more than 80% after just five minutes, and an additional 20% after 15 minutes.[7]

Some effective treatment options for MGD that use heat therapy include TranquilEyes (Ocusoft), Bruder Moist Heat Compresses (Bruder Healthcare) and the LipiFlow Thermal Pulsation System (TearScience). TranquilEyes care kits include foam inserts that continuously heat and moisten the eye for more than 30 minutes. Bruder Moist Heat Compresses use moisture beads to maintain heat on the eyelid for more than 20 minutes after being placed in the microwave.

Finally, the LipiFlow device incorporates moist, warm compression with mechanical eyelid massage for 30 minutes. This relatively new technology works effectively on a variety of patients—ranging from those with significant MG dropout to obstructive MGD.[8,9] A single LipiFlow Thermal pulsation system treatment may improve meibomian gland function and reduce dry eye symptoms for up to nine months.[10]

Topical Therapeutic Options

Inflammation or meibomitis secondary to MGD may require treatment with topical antibiotics, corticosteroids and/or combination agents. But, because obstructive



MGD often presents without visible inflammation, is it wise to employ such therapeutic agents off-label?[11]

Certainly, there is evidence that inflammation is present in dry eye disease.[12-17] Also, researchers have estimated that 86% of patients with dry eye disease showed signs of MGD.[18] But are there any specific studies on the role of inflammation in evaporative dry eye disease that is caused by MGD?

In one noteworthy study, researchers compared 23 patients with known mild-to-moderate evaporative dry eye disease with nine healthy subjects.[19] In both groups, 15 cytokines and chemokines were measured by multiplex bead analysis. The results showed that 14 of 15 inflammatory molecules were reliably detected in 1µl of tears from patients with evaporative dry eye disease.[19] Many of the cytokines actually could be correlated with specific symptoms, such as heightened volumes of interleukin-6 and increased levels of pain. These data suggest that anti-inflammatory medications could be useful in managing patients with evaporative dry eye and/or MGD.

In another study, a combination of azithromycin and warm compress therapy effectively alleviated meibomian gland plugging, increased meibomian secretion and reduced eyelid redness compared to the application of warm compresses alone.[20] A separate study showed that MGD patients who used Restasis (cyclosporine 0.05%, Allergan) exhibited fewer meibomian gland inclusions than those who received a placebo.[21]

Some of the other most commonly used therapeutic options for MGD include combination agents, such as Zylet (loteprednol etabonate and tobramycin, Bausch + Lomb) and TobraDex ST (tobramycin and dexamethasone, Alcon), as well as steroidal ointments, such as TobraDex (tobramycin and dexamethasone ophthalmic ointment, Alcon) and Lotemax (loteprednol etabonate ophthalmic ointment, Bausch + Lomb). Although none of these medications has a specific indication for MGD, they demonstrate significant therapeutic success in clinical practice.

Oral Doxycycline

Oral doxycycline is often used to treat MGD. It is interesting to note, however, that doxycycline is not detectable in the tear film of patients who are undergoing treatment.[22] So, perhaps the agent works either directly on the eyelid tissue or systemically to reduce inflammation.[23]

Gland Expression and Probing

Research has shown that aqueous tear evaporation diminishes following meibomian gland expression in patients with dry eye disease and/or MGD.[24] Typically, the pressure required to properly express the meibomian glands ranges from 5psi to 40psi for the initial non-liquid material and 10psi to 40psi for the remaining contents.[25] So, expression tools, such as the Mastrota Meibomian Gland Paddle (Ocusoft), Collins Expressor Forceps (Bruder Healthcare) and the MG Expressor Kit (Gulden Ophthalmics), may play an important role in successful care. Unfortunately, one study indicated that just 7% of patients could tolerate the pain required to administer complete expression along the entire lower eyelid margin.[25]

One additional question: Is there therapeutic benefit in intraductal meibomian gland probing? One study showed that the process relieved the symptoms of obstructive MGD in 25 consecutive patients.[26] However, further research may be needed to observe the long-term benefits over time, and to make certain that the more invasive technique does not damage the glands.

The Future of MGD Treatment

Future MGD treatments include the use of pulsed-light therapy on the eyelids as well as new therapeutic agents that affect cytokines and inflammatory T-cells.

Another potentially unique treatment option includes androgen therapy.[27] One study from Schepens Eye Research Institute showed that androgen regulation of gene expression exists in human meibomian glands.[27] The researchers showed that androgens, such as dihydrotestosterone, exert a significant influence on the structure, function and pathophysiology of the meibomian glands and may play a key role in the treatment of MGD.

Today, the acronym MGD is a true "buzzword" in the eye care community. During the last several years, this overwhelming interest has fostered a widely improved understanding MGD. With further clinical study during the next decade, researchers and physicians likely will uncover a host of new treatment options to help patients with this chronic condition that affects visual acuity, personal appearance and overall quality of life. ■

Dr. Karpecki is a paid consultant and/or advisor to Allergan, Alcon, Bausch + Lomb and Ocusoft. Neither he nor Dr. Shechtman have any direct financial interests in the products mentioned.

For a complete list of references, please visit www.revoptom.com.

Product Review

Ophthalmic Ultrasound System

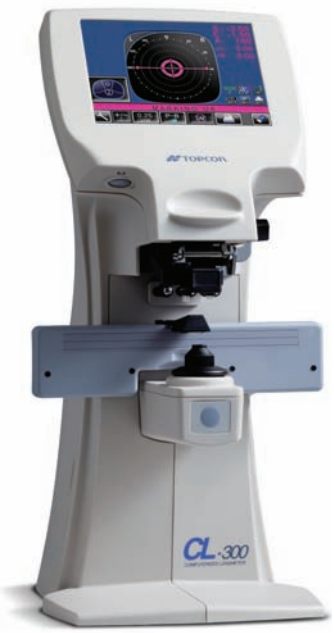
Compact Touch

Quantel Medical offers the Compact Touch, a three-in-one ultrasound system that provides a 10MHz B-scan of the globe and orbit, as well as options to perform biometry and pachymetry. Its image quality makes it possible to distinguish globe and orbit structures and to perform a detailed diagnosis of several pathologies, including cataract, opacity of the vitreous and retinal detachment, the company says.

The Compact Touch also offers automatic axial length measurements using a B-scan image, which can be helpful in cases of advanced myopia or staphyloma. The biometry option allows practitioners to measure axial length behind an opaque area (e.g., cataract). The device's pachymetry capability measures corneal thickness to an accuracy of ±5µm and includes intraocular pressure and corneal thickness correlation tables.

This compact, portable ophthalmic ultrasound system comes with a carrying case, a foot pedal for easier measurement and a mouse.

Visit www.quantel-medical.com.



Computerized Lensmeter

CL-300

Topcon recently released its newest computerized lensmeter, the CL-300, with several added features—including a new UV transmittance measurement function that the company says provides reliable results for eyeglasses and sunglasses in the range of 0% to 100%. It also provides automatic detection for single and progressive lenses and offers improved lens support.

Available in two models, the CL-300P and the CL-300PDL (allowing PD measurements), the CL-300 has a fast, easy-to-load printer and a large 5.7-inch monitor. It can easily be connected to the Topcon CV-5000S Computerized Vision Tester and to electronic medical record systems.

Visit www.topcon.com.

Lenses

Vizera Anti-Reflective Coating

Ophthonix has upgraded its entire iZon lens portfolio to feature a new anti-reflective coating, Vizera. It provides the same high level of clarity and glare reduction as the standard AR coating previously used on all iZon lenses, but with a higher Bayer value and superior abrasion-resistance, the manufacturer says.

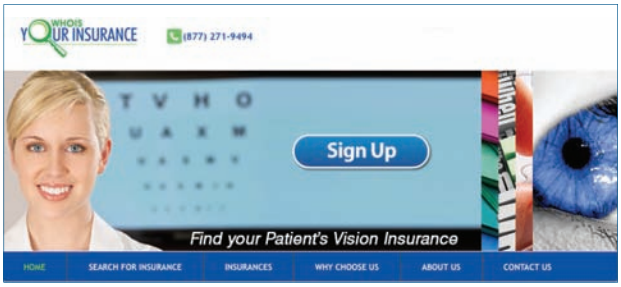
This increased protection from glare and scratches helps the lenses remain as clear as possible, increasing longevity and reducing the impact on clarity, the company says.

Visit www.izonlenses.com.

Web Resources

Who Is Your Insurance

A new website, www.whoisyourinsurance.com, can instantly provide your office manager or administrator with a patient's vision care insurance information, even if the patient is unsure of his or her vision insurance carrier. The site instantly searches all insurance carriers simultaneously from one place instead of visiting each carrier's website.



Employees do not need to know the office's insurance carrier account credentials, and the site tracks which employees are searching and what they are searching for. The website integrates easily with practice management software.

Frames

Valentino Fall/Winter Men's Eyewear Collection

The Valentino fall/winter 2013 men's eyewear collection features iconic shapes and classic styles with a contemporary edge.

• ***Rockstud (V106S):*** Two tiny metal studs anchor the temples on these aviator lenses. The double bridge is another distinctive element of this classic frame with large gradient lenses.

• ***V Logo (V105S):*** Gradient lenses are encapsulated in this large, square enameled metal frame. A fine silver line runs around the entire lens to add a touch of light, and the iconic "V" is engraved on the temple.

• ***Rockstud (V631S):*** This square acetate style has a simple masculine frame front that contrasts with the embellished temples—a metal "V" forms the hinge and four studs run along its length.

Visit www.marchon.com.


V106S


V105S


V631S

Maui Jim On the Water Sunglass Collection


Dorado


Nine Palms


Surf Rider

Maui Jim will launch seven new sunglass styles throughout 2012, adding to its On the Water sunglass collection. The complete collection will feature 27 sunglasses for men and women, ranging from $219 to $309. Three new unisex styles were recently added:

• ***Dorado (Style #259):*** Maui Evolution lenses with PolarizedPlus2 technology are designed for active water sports. Dorado comes in: matte black with Maui HT lenses; root beer with HCL bronze lenses; and translucent dark gray with neutral gray lenses. This style is available with MauiPassport prescription lenses in powers from +3.00 to -2.50.

• ***Nine Palms (Style #255):*** Nine Palms offers large, full-frame coverage with scratch- and impact-resistant Maui Evolution lenses. This style comes in: matte black with Maui HT lenses; root beer with HCL bronze lenses; and translucent dark gray with neutral gray lenses. It's available with MauiPassport prescription lenses in powers from +3.00 to -2.50.

• ***Surf Rider (Style #261):*** This midsize, full-frame nylon style is designed to go from the boat to the surfboard to the beach. These sunglasses have ultra-clear SuperThin Glass lenses. Surf Rider comes in: black with blue interior and neutral gray lenses; black stripe with neutral gray lenses; and tortoise with HCL bronze lenses for variable light conditions. It's available with MauiPassport prescription lenses in powers from +3.00 to -3.50.

Visit www.MauiJim.com. ■

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- Anterior Segment New Technology
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- Glaucoma
- Ocular Surface Disease
- Anterior Segment Disease
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Meetings + Conferences

September 2012

■ **5-8.** *International Vision Expo & Conference West 2012.* Sands Expo & Convention Center, Las Vegas. Call (800) 811-7151 or visit www.visionexpowest.com.

■ **6-9.** *72nd Annual Middle Atlantic Optometric Congress.* Doubletree Hotel and Convention Center, Pittsburgh/Monroeville, Pa. Under the auspices of: The Optometric Extension Program Foundation & the Western Pennsylvania Optometric Society. CE hours: 12. E-mail Barry Cohen, O.D., at barryc51@gmail.com.

■ **6-10.** *The Art and Science of Optometric Care: A Behavioral Perspective.* Grand Rapids, Mich. Held by: The Optometric Extension Program Foundation. CE hours: 35. E-mail Theresa Krejci at TheresaKrejciOEP@verizon.net or visit www.oepf.org.

■ **7-9.** *43rd Annual Colorado Vision Training Conference.* YMCA of the Rockies. Estes Park, Colo. Contact Jamie Anderson, O.D., F.C.O.V.D., at (303) 683-4466 or drjamieanderson@gmail.com.

■ **8.** *Neuro-Optometric Rehabilitation Continuing Education Seminar.* Western University College of Optometry, Pomona, Calif. CE hours: 9. Call (909) 706-3493 or e-mail ceoptometry@westernu.edu. Visit www.westernu.edu/optometry-continuing-education.

■ **8-9.** *Primary Eye Care Update.* Northeastern State University, Oklahoma College of Optometry, Tahlequah, Okla. CME hours: 10. Contact Ashley Beason Manes at beason01@nsuok.edu or (918) 444-4033. Visit www.optometry.nsuok.edu.

■ **8-9.** *Fall Conference 2012.* Terry Auditorium, Nova Southeastern University, Fort Lauderdale, Fla. E-mail oceaa@nova.edu or visit http://optometry.nova.edu/ce/index.html.

■ **9-10.** *Northeast Congress.* Westford Regency Inn & Conference Center, Westford, Mass. CE hours: 12. Contact Kathleen A. Prucnal, O.D., at (978) 597-5227 or drkaprucnal@msn.com.

■ **12-15.** *Envision Conference.* Hilton St. Louis at the Ballpark, St. Louis. E-mail info@envisionconference.org or call (316) 440-1530. Visit www.envisionconference.org.

■ **13-14.** *South Dakota Optometry Society Fall Conference.* Hilton Garden Inn, Sioux Falls, S.D. Call (605) 224-8199 or e-mail deb.mortenson@pie.midco.net. Visit www.sdeyes.org.

■ **14-16.** *SWCO 2012.* InterContinental Hotel, Dallas. Sponsored by: Southwest Council of Optometry. Contact Niki Bedell, M.P.H., at (713) 743-1856 or nbedell2@uh.edu. Visit www.swco.org.

■ **14-16.** *VOA Annual Meeting.* Basin Harbor Club, Vergennes, Vt. Hosted by: Vermont Optometric Association. CE hours: 17. Contact David J. DiMarco, O.D., at (802) 524-9561 or djd@nveyecare.net. Visit www.vtoptometrists.org.

■ **14-16, 18-20.** *CE in Italy: Florence and/or Castiglion Florentino, Tuscany.* To register for one or both programs, contact James L. Fanelli, O.D., at (910) 452-7225 or jamesfanelli@CEinItaly.com. Visit www.CEinItaly.com.

■ **18.** *Advanced CE for Optometric Physicians.* Crowne Plaza Hotel, Natick, Mass. Hosted by: Eye-Sight 20/20, LLC. Presenters: James Bartlett, O.D., and Michael Politzer, O.D. CE hours: 8. Call Dr. Antoinette Parvis at (508) 987-9679 or visit www.eyesightce.com.

■ **21-23.** *New Technology and Treatments in Vision Care.* Hilton La Jolla Torrey Pines, La Jolla, Calif. Hosted by: *Review of Optometry.* Meeting chair: Paul Karpecki, O.D. CE hours: 15. Contact Lois DiDomenico at ReviewMeetings@jobson.com or (866) 658-1772. Visit www.revoptom.com/conferences.

■ **23.** *CPOS CE Forum XVI.* The Hotel Hershey, Hershey, Pa. Hosted by: Central Pennsylvania Optometric Society. Featured speakers: Ron Melton, O.D., and Randall Thomas, O.D., M.P.H. CE hours: 6. E-mail Mary Good, O.D., at cposrsvp@gmail.com.

■ **27-30.** *GWCO Congress 2012.* Oregon Convention Center, Portland. Hosted by: Great Western Council of Optometry. CE hours: 59. Visit http://www.gwco.org/Congress.html.

■ **27-30.** *2012 WOA Convention and Annual Meeting.* Kalahari Resort, Wisconsin Dells, Wis. Hosted by: Wisconsin Optometric Association. CE hours: 22. Visit www.woa-eyes.org.

■ **28-30.** *Illinois Optometric Association Annual Convention.* Crowne Plaza Hotel, Springfield, Ill. Call (800) 933-7289 or visit www.ioaweb.org.

■ **30-Oct 2.** *NDOA 109th Annual Congress & Exhibition.* Ramkota Hotel, Bismarck, N.D. Hosted by: North Dakota Optometric Association. Call (877) 637-2026 or e-mail ndoaz@btinet.net. Visit www.ndeyecare.com.

October 2012

■ **4-7.** *EastWest Eye Conference.* Cleveland Convention Center, Cleveland. Hosted by: Ohio Optometric Association. Call (800) 999-4939 or e-mail info@ooa.org. Visit www.eastwesteye.org.

■ **6-7.** *PSS 2012: 2nd Annual Forum on Ocular Disease.* The Castle Hotel & Resort, Orlando, Fla. Hosted by: PSS EyeCare. CE hours: 18. Call (203) 415-3087 or e-mail education@psseyecare.com. Visit www.psseyecare.com.

■ **10-11.** *44th Annual Fall Seminar.* The Lansing Center, Lansing, Mich. Hosted by: Michigan Optometric Association. Contact Amy Possavino at amy@themoa.org or (517) 482-0616. Visit www.themoa.org.

■ **11.** *3rd North Jersey Optometric Seminar.* JCC MetroWest, West Orange, N.J. Presenters: Jai Parekh, M.D., and Mary Boname, O.D. CE hours: 4. Call William B. Potter, O.D., at (609) 947-8545 or e-mail eyedoc2180@aol.com. Visit http://optometryonwest44th.webs.com.

■ **12.** *HVOS Fall Seminar.* The Grandview, Poughkeepsie, N.Y. Hosted by: Hudson Valley Optometric Society. E-mail Robert Greenbaum, O.D., at robertgreenbaum58@gmail.com or call (845) 473-0220. Visit www.hvos.org.

■ **12-13.** *Northwoods Education Events.* Black Bear Lodge, St. Germain, Wis. Hosted by: Wisconsin Optometric Association. E-mail joleenwoaoffice@tds.net or (800) 678-5357. Visit www.woa-eyes.org.

■ **13-14.** *Fall Conference.* Lansdowne Resort, Leesburg, Va. Hosted by: Virginia Optometric Association. Call (804) 643-0309 or visit www.thevoa.org.

■ **16-20.** *COVD 42nd Annual Meeting.* Omni Fort Worth Hotel, Fort Worth, Texas. Hosted by: College of Optometrists in Vision Development. Contact info@covd.org or (330) 995-0718. Visit www.cvod.org.

■ **18.** *6th Central Jersey Optometric Seminar.* CentraState Medical Center, Freehold, N.J. Presenter: William Marcolini, O.D. CE hours: 4. Call William Potter, O.D., at (609) 947-8545 or e-mail eyedoc2180@aol.com. Visit http://optometryonwest44th.webs.com.

■ **24-27.** *Academy 2012 Phoenix.* Phoenix Convention Center. Hosted by: American Academy of Optometry. Visit www.aaopt.org/meetings/academy2012.

■ **25.** *1st SouthWest Jersey Optometric Seminar.* The Enterprise Center at Burlington County College, Mount Laurel, N.J. Presenter: William Potter, O.D. CE hours: 4. Call Dr. Potter at (609) 947-8545 or e-mail eyedoc2180@aol.com. Visit http://optometryonwest44th.webs.com.

■ **27-28.** *VOSH/International Annual Meeting.* Marriott Renaissance Phoenix Downtown Hotel. Hosted by: VOSH/International. Contact Harry I. Zeltzer, O.D., at vosh@vosh.org. Visit vosh-california.org/voshinter/annual12.html.

■ **31.** *Advanced CE for Optometric Physicians.* Crowne Plaza Hotel, Natick, Mass. Hosted by: Eye-Sight 20/20, LLC. Presenters: Jerome Sherman, O.D., and Leo Semes, O.D. CE hours: 8. Call Dr. Antoinette Parvis at (508) 987-9679 or visit www.eyesightce.com.

November 2012

■ **8-11.** *Monterey Symposium.* Monterey Marriott Hotel & Conference Center, Monterey, Calif. Hosted by: California Optometric Association. Call Will Curtis at (916) 266-5037 or e-mail wcurtis@coavision.org. Visit www.coavision.org.

■ **9-10.** *C.E. Charleston.* Doubletree Charleston Historic District, Charleston, S.C. Hosted by: Pacific University College of Optometry. CE hours: 12. Call Jeanne Oliver at (503) 352-2740 or e-mail jeanne@pacific.edu. Visit www.pacificu.edu/optometry.ce.

■ **9-11.** *FCO International 23rd Annual Educational Conference.* Abe Martin Lodge, Brown County State Park, Nashville, Ind. Hosted by: Fellowship of Christian Optometrists. Visit www.fcoint.org/services/annualConference.html.

To list your meeting, contact:
Colleen Mullarkey, Senior Editor
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IN VISION CARE

West Coast

SEPTEMBER 21-23, 2012
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New Technology & Treatments in Vision Care *West Coast*

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La Jolla, CA / September 21-23, 2012

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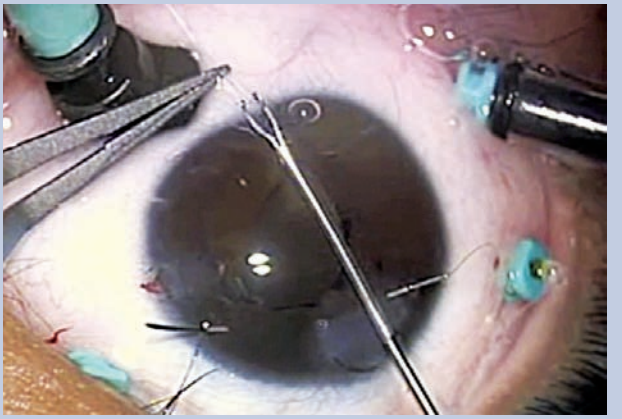
Iris-sutured IOLs

When cataract surgery goes awry, this rescue technique can help make the best of a bad situation and provide the patient with good postoperative vision.

By Derek N. Cunningham, O.D., and Walter O. Whitley, O.D., M.B.A.



Go to www.revoptom.com to see video footage of an iris-sutured intraocular lens implantation.

On The Web ›› View a narrated video of this technique in a three-year-old Marfan's patient.

Occasionally, cataract surgeons run into intraoperative complications and decide that salvaging any vision is better than none. If the capsular bag is not suitable for insertion of the preselected IOL, the surgeon can place the lens in the sulcus, suture a posterior chamber lens to the iris, implant an anterior chamber IOL or simply leave the patient aphakic for the time being.

Suturing an IOL into the iris typically is done when there is a lack of structural capsular integrity or poor zonule support behind the iris. Among the numerous possible causes are chronic uveitis, retinitis pigmentosa, previous ocular surgery (e.g., glaucoma, retina), pseudoexfoliation and congenital syndromes (e.g., Marfan's, homocystinuria). Ideally, the surgeon will be prepared for the likelihood of weak zonules or a loose lens, but this may not be discovered until the cataract surgery has begun. The IOL haptics provide an adequate fixation point for securing the lens to the peripheral iris. Because specially made lenses for iris suturing do not fold, many retina surgeons prefer to use traditional three-piece IOLs that can be folded and inserted through a small scleral incision.

In the video posted online, the lens is positioned posterior to the iris using trocars and held in place while the lens is sutured with an anterior approach. With this method, the lens can be positioned and secured without large anterior chamber incisions. The natural lens was also removed by vitrectomy prior to IOL insertion.

When comanaging these patients, understand that the visual outcome can be limited by preexisting disease, and that this technique of lens placement is far less accurate that traditional extracapsular cataract surgery. Patients may notice more visual distortion from the lens, but slight decentration of the lens typically does not cause a decrease in acuity; surgical repositioning rarely is warranted. These cases are managed very similarly to a routine cataract surgery. There may be more inflammation within the eye due to the extended surgical time, which can be managed with anti-inflammatories appropriately. The comanaging optometrist will want to ensure that the lens remains fixated to the iris and there is not an excessive amount of posterior chamber inflammation.

Visual acuity is relatively stable immediately after surgery and may only be limited by ocular inflammation. Near correction can be beneficial immediately after surgery. Corrective lenses for distance can be prescribed at approximately one week. These patients typically are safe to dilate within days of surgery if necessary, but close communication with the surgeon is required to navigate any concerns with these abnormal cases.

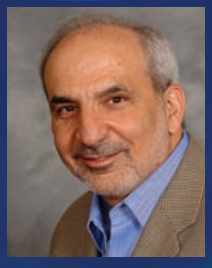
Suturing IOLs to the iris is rarely the preferred technique for surgeons, but it does represent amazing ingenuity and allows the surgeon to get the best vision out of what is available. What's truly remarkable about these cases is how well some patients can end up seeing. ■

Save the Date!

Mark your calendars for the

3rd Annual

West Coast Optometric Glaucoma Symposium



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Murray Fingeret, OD

Speakers:
Michael Chaglasian, OD
John Flanagan, MCOptom, PhD
I. Ben Gaddie, OD
Tony Realini, MD
Robert N. Weinreb, MD

Additional Faculty TBA

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I invite you to join us for this comprehensive glaucoma symposium that will take place at the Fairmont Newport Beach in Newport Beach, California. We will have a group of renowned doctors who specialize in glaucoma. The program will consist of short talks and Q&A panels. Interaction with the faculty is a large part of the program. The agenda will cover the steps taken in establishing the diagnosis of glaucoma, information on new instrumentation and changing paradigms with regard to glaucoma therapy. The program runs Friday from 9:00 am – 6:00 pm, and Saturday from 8:00 am – 1:30 pm.

I look forward to seeing you in Newport Beach!
Murray Fingeret, OD

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Diagnostic **Quiz**

His Eyes Aren't the Problem

By Andrew S. Gurwood, O.D.

History

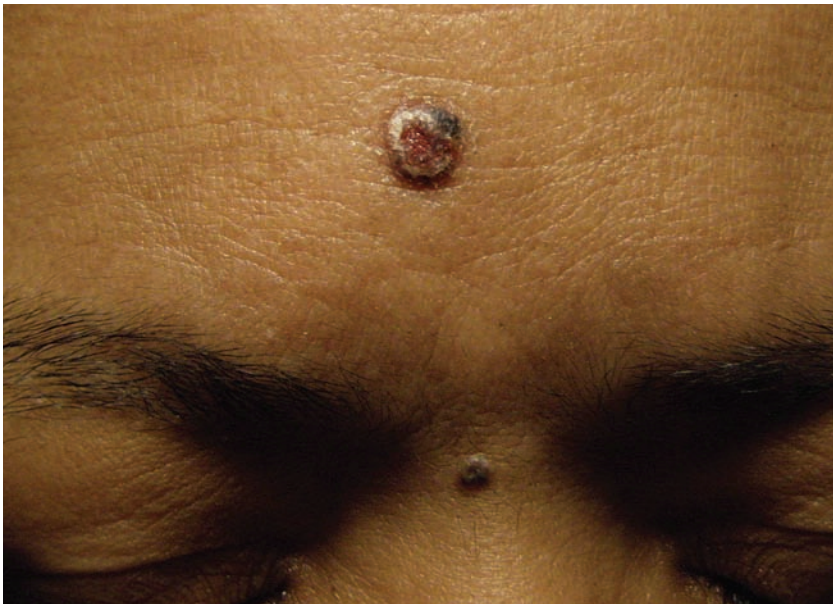
A 33-year-old black male presented for an annual eye examination. He had no complaints, and his ocular and systemic histories were unremarkable. He denied using any medications, and reported no known allergies.

Diagnostic Data

His best-uncorrected visual acuity measured 20/20 O.U. at distance and near. Refraction uncovered clinical emmetropia. His pupils were equally round, with no evidence of afferent defect.

Extraocular muscle movements were full and unrestricted in all positions of gaze. Confrontational fields were full O.U.

Slit lamp examination revealed normal and healthy anterior segment structures. His intraocular pressure measured 16mm Hg O.D. and 15mm Hg O.S. Dilated fundus examination uncovered normal posterior poles, with healthy and intact peripheries.



External examination of our patient revealed the presence of a significant lesion.

However, during gross external inspection, we observed the presence of a significant lesion.

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. ■

Retina Quiz Answers *(from page 106):* 1) b; 2) b; 3) b; 4) c; 5) c.

Next Month in the Mag

Our September issue features the 35th Annual Diagnostic Technology Report, which includes:

- *Annual Technology Survey*
- *Can Image Management Systems Improve Patient Care?*

Also in September:

- *Optometric Study Center: Differential Diagnosis and Management of Unexplained Vision Loss* (earn 2 CE credits)
- *An Update on Drugs in the Pipeline*
- *In-Office Lab*

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References: 1. Dumbleton K, Woods C, Jones L, et al. Patient and practitioner compliance with silicone hydrogel and daily disposable lens replacement in the United States. *Eye Contact Lens.* 2009;35(4):164-171. **2.** Yeung K, Forister J, Forister E, et al. Compliance with soft contact lens replacement schedules and associated contact lens-related ocular complications: The UCLA Contact Lens Study. *Optometry.* 2010; 81: 598-607. **3.** Jones L, Dumbleton K, Fonn D, et al. Comfort and compliance with frequent replacement soft contact lenses. *Optom Vis Sci.* 2002;79:259.

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